INTRODUCTION

Shock is a state of widespread tissue hypoperfusion which can be caused by a variety of illnesses or injuries. By definition, it creates an imbalance of tissue oxygen supply and demand. If left untreated, it can lead to end-organ compromise and failure. The circulatory system must be intact in order to maintain adequate tissue perfusion, therefore the fundamental treatment of shock is to treat the underlying cause, and restore perfusion. Early recognition of shock is important, as mortality increases as the stages of shock progress. It is also important to remember that hypotension and hypoperfusion do not mean the same thing. The hypoperfusion associated with shock typically occurs prior to a drop in blood pressure, and it is important to recognize the early signs and symptoms in order to provide timely treatment.

SAFETY

Consider the underlying cause of shock and take necessary precautions. If shock is due to trauma, ensure the scene is safe. If it is due to an infectious process, apply appropriate PPE.

ASSESSMENT

The circulatory system contains three main components - the heart, the blood, and the blood vessels. A disruption in any of these components can result in shock. There are 4 overall categories of shock to consider: hypovolemic, cardiogenic, distributive, and obstructive. It is important to consider that there may be multiple etiologies of shock occurring simultaneously. Consider a broad differential diagnosis.

The clinician should perform a thorough multisystem assessment in order to determine if signs of shock are present. Shock may be obvious, but often the signs and symptoms are more subtle. The clinician should consider the patient's history, vital signs, level of consciousness, mental status, respiratory status, presence and location of any pain, history or signs of volume loss (e.g. blood, emesis, diarrhea), and peripheral/extremity temperature when exploring for signs or symptoms of shock.

Vital signs provide very useful information when it comes to shock. Tachycardia and/or tachypnea are some of the first signs to appear in a patient with shock and should not be overlooked. Hypotension on the other hand is a late sign and attempts should be made to initiate treatment before hypotension occurs.

Signs of shock:

- Shortness of breath
- Tachypnea
- Hypotension
- Tachycardia with weak peripheral pulses
- Arrhythmia
- Pale/cool/clammy skin*
- Cold/ mottled extremities*
- Altered mental status (anywhere from lethargy or unresponsiveness to anxiety and combativeness)
- Decreased urination

*extremities may be warm or red in distributive shock

Hypovolemic Shock

This is a state caused by internal or external loss of fluid. When caused by a loss of blood (e.g. due to trauma or gastrointestinal bleeds) it is referred to as **hemorrhagic hypovolemic shock.** When the hypovolemia is due to other types of fluid loss (e.g. vomiting, diarrhea, osmotic diuresis with diabetic ketoacidosis, third-space shift with burns) this is called hypovolemic non-hemorrhagic shock.

Figure 1 demonstrates the changes in patient presentation associated with the various stages of hemorrhagic hypovolemic shock. As fluid loss increases, the body uses all systems in order to compensate. Finally, the body's compensatory mechanisms start failing and decompensation occurs.

Cardiogenic Shock

Cardiogenic shock is due to impaired pump function of the heart muscle such that there is insufficient perfusion of body tissues. It is most often secondary to myocardial infarction or worsening congestive heart failure, but it can also be caused by trauma, arrhythmias, valvular disease, cardiomyopathy, or papillary heart muscle rupture. A 12 lead ECG should be obtained for all shock patients to look for signs of ischemia, arrhythmia and/or toxicity. When assessing a patient with cardiogenic shock, there will often be adventitious lung sounds such as

crackles, due to left sided pump failure. They may also have a cough associated with frothy sputum and cyanosis. Right sided pump failure will result in JVD and peripheral edema. There may be a recent history of worsening chest discomfort, shortness of breath, edema, or fatigue, or the patient may simply abruptly deteriorate without warning.

Obstructive Shock

A mechanical or structural obstruction of blood flow through the great vessels, heart and/or lungs can lead to obstructive shock, such as in the case of a tension pneumothorax (pressure on the heart and vena cava), pulmonary embolus (reduces flow to left ventricle), aortic dissection, or cardiac tamponade (right ventricular collapse due to pericardial fluid). The hallmark sign of obstructive shock is distended jugular neck veins due to increased jugular venous pressure. The patient will also compensate with peripheral vasoconstriction resulting in cool clammy extremities. Again, it is important to consider multiple etiologies of shock in the polytraumatized patient. Assume you are dealing with hemorrhagic shock, but always assess for findings that might suggest tension pneumothorax (JVD, unilateral decreased breath sounds, tracheal deviation, hypoxia).

Distributive Shock

Unlike hypovolemic shock, with distributive shock the actual intravascular blood volume has not significantly changed. The peripheral blood vessels dilate in response to the underlying pathophysiology, resulting in a "relative hypovolemia". Blood is displaced away from the central circulation as a result of this vasodilation. It is important to note that unlike all other categories of shock, patients with distributive shock will classically have warm extremities, despite widespread hypoperfusion. All other categories of shock will classically result in cool, pale extremities due to compensatory vasoconstriction. Septic shock, anaphylactic shock, and neurogenic shock all fall under the category of distributive shock. There are also endocrine and toxicological etiologies of distributive shock. Assessment and management is the same as other types of distributive shock.

Septic shock occurs as a result of a severe infection causing a full body inflammatory response. Signs of increased metabolism (e.g. increased temperature, heart rate, and respiratory rate) typically occur early on, as the heart rate compensates for the vasodilation and the blood pressure is initially normal. As it progresses, septic shock manifests much like hypovolemic shock, with hypotension, altered mental status, and decreased urine output, although peripherally they will be warm to the touch despite also being systemically hypoperfused (secondary to vasodilation). Patients with septic shock will often have a recent history of infection, urinary catheterization, or other such findings that would lead the clinician to suspect infection/sepsis as the underlying cause. Refer to the Sepsis Syndrome Clinical Practice Guideline for more information on the assessment and management of sepsis, severe sepsis and septic shock.

Anaphylactic shock occurs as a result of histamine release. This mediates vasodilation, bronchoconstriction, and leaking of blood vessels involving multiple body systems. This will manifest clinically as urticaria, angioedema (often around the face), tachycardia, hypotension, nausea/vomiting, shortness of breath, and wheezing. Refer to the Allergic Reaction Clinical Practice Guideline for more information on the assessment and management of allergic reactions and anaphylaxis.

Neurogenic shock occurs as a result of an acute spinal cord injury that disrupts the sympathetic nervous system that would typically function to increase heart rate and blood pressure as needed to maintain perfusion. This loss of "sympathetic tone" results in vasodilation, relative bradycardia, and hypoperfusion. Because the vessels dilate in neurogenic shock, the patient may have warm, red, and dry skin due to the lack of sympathetic innervation, or there may be a clear sweat line correlating to the level of injury. Isolated neurogenic shock in the trauma patient will present with findings of hypoperfusion and hypotension, however the patient will be bradycardic and peripherally warm (the opposite of what you would expect with hemorrhagic shock). It is important to keep in mind that the polytraumatized patient may have multiple underlying causes of shock (e.g. hemorrhagic, neurogenic, and obstructive), and the clinical picture may therefore be mixed. With trauma patients, the clinician should always operate under the assumption that they are dealing with hemorrhagic shock until proven otherwise, while keeping other possible contributing causes in mind.

See the Neurological Trauma Clinical Practice Guideline for more information on the assessment and management of head and spine.

Toxicological distributive shock occurs as a result of the effects of exposure to drugs which affect the vasomotor status of the patient. Many times the toxidrome contributes to both cardiogenic and distributive categories of shock (e.g. beta-blocker overdoses). Common causes of toxicological shock include excessive alcohol consumption, as well as narcotic, acetaminophen, and ASA overdoses.

Endocrine distributive shock occurs as a result of the effects of disruption of the patient's endocrine system and the downstream effects on the patient's thyroid and adrenal function. A common prehospital example includes adrenal insufficiency (e.g. Addison's Disease) where patients may present in a shock state with recent corticosteroid therapy and found to have low blood glucose on paramedic assessment.

The categories of shock may also be thought of conceptually as pre-pump, pump, and post-pump pathophysiology. There may be multiple underlying causes in some cases.

Pre-Pump
[1] Hypovolemic Shock

Pump
[2] Cardiogenic Shock

Post-Pump
[3] Obstructive Shock
- RV Obstruction: PE, Severe Asthma
- LV Obstruction: Aortic Dissection, Hypertensive Crisis
- RV/LV Obstruction: tension pneumothorax, cardiac tamponade

[4] Distributive Shock
- **N**eurogenic
- **A**naphylactic
- **S**eptic
- **T**oxicological
- **E**ndocrine

MANAGEMENT

The prognosis of shock depends on the underlying cause, how soon it is treated, and the nature and extent of concurrent problems. Hypovolemic and anaphylactic shock may respond quickly to pre-hospital interventions, whereas septic and cardiogenic shock are difficult to treat in the field and have a very high mortality rate. Keep in mind multiple management strategies may be indicated if multiple causes of shock are present.

The overall goal is immediate transport to the appropriate facility for definitive care, with pre-hospital treatment focusing on supporting the ABCs and adequate perfusion in the interim. The nuances regarding management of the specific types of shock are outlined below.

General Principles

Oxygenation in Shock
Shock is a state of hypoperfusion. The clinician should therefore aim to obtain an SpO_2 of 100%, except in the setting of an MI, where an SpO_2 between 94-99% has been shown to improve patient outcome. Remember that pulse oximetry readings can be unreliable in the presence of peripheral hypoperfusion.

General Fluid Therapy in Shock
In general, intravenous fluid boluses can be given to patients in shock at a volume of 20 mL/kg. If there are adventitious lung sounds such as crackles, contact OLMC for advice. After each bolus, reassess the patient and repeat the bolus up to 3 times or until the goals of therapy have been achieved (i.e. resolution of tachycardia, tachypnea, and hypotension).

If after 3 boluses the patient remains hypotensive (SBP less than 90mmHg), proceed to vasopressors such as dopamine. Dopamine can be used to increase the peripheral vascular resistance as well as increase cardiac output by increasing the stroke volume. It is important to note however that the desired action of dopamine can also lead to increased myocardial work causing ischemia and/or arrhythmia. If a dopamine infusion is required, titrate to maintain a systolic blood pressure of 90mmHg.

There are some exceptions to the above general strategy. For instance, patients with hemorrhagic

shock should undergo less aggressive fluid resuscitation, and the use of vasopressors such as dopamine can worsen outcomes in hypovolemic shock. Patients with underlying cardiac or renal disease may not be able to tolerate aggressive fluid resuscitation, therefore smaller boluses (250 mL) followed by reassessment after each bolus is more appropriate in these patients.

Specific management strategies required to treat the various different categories of shock are detailed below.

Hypovolemic Shock

The goal of therapy is to stop the source of fluid loss if possible and restore intravascular volume enough to permit adequate physiologic function. Isotonic IV fluids can be used in an attempt to refill the system, as isotonic fluids have a similar concentration to blood. Oxygen should be administered to correct hypoxia and bleeding should be controlled with stabilization and/or direct pressure as appropriate. The clinician may also consider transporting the patient to a facility where blood product replacement (**PEP white**) can occur. If fluid loss is due to vomiting provide anti-emetics, and if it is due to fluid evaporation, such as with burns, attempt to control the loss of fluid (see Burns Clinical Practice Guideline for more information on management of burns).

When the vasculature has been disrupted, such as in the case of hemorrhagic hypovolemic shock in trauma, there is a fine balance between restoring sufficient blood pressure for "adequate" physiologic function, and driving the blood pressure up unnecessarily high (i.e. back to "normal") which increases blood loss. For this reason, when managing hemorrhagic shock the goal should be to maintain a systolic blood pressure of 100mmHg. Aggressive fluid resuscitation beyond this point may in fact cause harm to these patients. This strategy of less aggressive fluid resuscitation in hemorrhagic shock is referred to as **permissive hypotension** (**PEP 1 supportive**). One must also balance this approach against the well-established risks of hypotension in the polytraumatized head injured patient.

Patients who have hemorrhagic hypovolemic shock caused by trauma may also benefit from the administration of tranexamic acid (TXA) as long as it is given within 3 hours of injury. Refer to the General Major Trauma Clinical Practice Guideline for further information on TXA.

Cardiogenic Shock

Mortality is very high in this population, and management is very challenging. The underlying cause of cardiogenic shock must be treated, which may include reperfusion therapy in the setting of STEMI. STEMI patients in cardiogenic shock are definitively managed with PCI as opposed to TNK (**PEP white**), even when reperfusion is delayed for several hours. The Regional Hospital ED Physician should be consulted regarding management options and trip destination, and Air Medical Transport may be indicated to transfer the patient for PCI.

If an arrhythmia is causing the patient's shock state, treat the arrhythmia as per Adult Arrhythmia guidelines.

IV fluid may be used to help support hemodynamics and improve perfusion, but must be administered cautiously to avoid worsening volume overload (**PEP white**). Small boluses of 250 mL may be administered with frequent reassessment of respiratory status (oxygen saturation, lung sounds, level of respiratory distress). If the patient's respiratory status is worsening, fluid administration should be stopped or slowed. If the blood pressure is improving with IV fluids, and the respiratory status is stable, 250 mL boluses may continue until an endpoint SBP of 90mmHg is reached. If the patient is unable to tolerate IV fluids, and/or a SBP of 90mmHg is unobtainable, Dopamine may also be given to patients in cardiogenic shock.

Obstructive Shock

In all cases of obstructive shock, definitive care requires specific in-hospital management based on the underlying cause. IV fluid administration is indicated in the prehospital setting to temporarily support hemodynamics for all of these patients. A tension pneumothorax also requires immediate needle decompression as soon as it is recognized clinically.

Distributive Shock

Despite the fact that fluid loss has not actually occurred, the widespread vasodilation associated with this category of shock has resulted in the vasculature being relatively volume deplete. The goal here is to expand the intravascular volume sufficiently to permit normal physiologic function,

while also providing specific therapies to address the underlying cause. Attempting to cause vasoconstriction with pharmacologic agents such as dopamine prior to administering sufficient IV fluid is likely to be unsuccessful and may lead to avoidable complications such as dysrhythmia.

The systematic management of ***septic shock*** is termed Early Goal Directed Therapy, and is used to balance oxygen delivery with oxygen demand, and can be initiated in the prehospital setting by providing oxygen (**PEP white**), IV fluids (**PEP 1 supportive**), and vasopressors (**PEP 1 supportive**) as needed. Hypotension due to septic shock often requires large volumes of IV fluids, which is one of the hallmark signs of septic shock. A common error in management is providing insufficient fluid resuscitation. IV fluid resuscitation should be appropriately aggressive. Reassess between boluses for signs of volume overload, and consult OLMC as required. See the Sepsis Syndrome Clinical Practice Guideline for further information.

Epinephrine and antihistamines are used to manage ***anaphylactic shock***, with the inclusion of beta agonists if wheezing is present. IV fluids are appropriate to correct existing hypotension. See the Allergic Reaction Clinical Practice Guideline for further information.

Managing ***neurogenic shock*** includes administering IV fluids (**PEP 2 supportive**) to increase intravascular volume in the setting of widespread vasodilation. Treating hypotension in a patient with a head injury is one of the first priorities, as early hypotension has been found to be associated with increased mortality. Dopamine (**PEP 2 supportive**) may be used if IV fluids do not correct the hypotension. Dopamine causes peripheral vasoconstriction, as well as having a chronotropic effect to help to increase the heart rate. Both these mechanisms will help mitigate the loss of sympathetic tone. Keep in mind, there may be other causes of shock present in the polytraumatized patient, and hemorrhagic shock must be considered present until proven otherwise.

Type of Shock	Key Prehospital Management
Hypovolemic (hemorrhagic)	Fluid, TXA, control bleeding
Hypovolemic (non-hemorrhagic)	Fluid
Cardiogenic	Cautious fluid, vasopressors, PCI/TNK (if STEMI)
Obstructive	Fluid, needle decompression (if tension pneumothorax)
Distributive (Neurogenic)	Fluid, vasopressors
Distributive (Anaphylactic)	Epinephrine, fluid
Distributive (Septic)	Fluid, oxygen, vasopressors
Distributive (Toxicological)	Antidotes, fluid, vasopressors, electrical therapy
Distributive (Endocrine)	Fluid

Transport Decision

Take into consideration the status of the patients as well as the underlying cause of the shock state when choosing a receiving facility. Patients involved in trauma may require a trauma centre, those with an MI may require PCI, and blood transfusion may be required for some patients presenting with shock. It is important to know what facilities in your area are able to provide the definitive care the patient requires. Early notification to the receiving facility is also important and should include information of the type of shock which may help the facility prepare for the arrival of the patient. In the case of hemorrhagic hypovolemic shock, consider contacting the hospital to activate the massive transfusion protocol.

TRANSFER OF CARE

For patients in shock, the decision regarding appropriate destination is based on the patient's current clinical condition as well as the underlying illness or injury. Once at the receiving facility it is important to detail the mechanism of injury or illness, any pertinent physical findings and the type and volume of fluids and/or medications provided to the patient.

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CHARTING

In addition to the mandatory fields, it is important to document the following in the ePCR text fields:

- ✓ Any instance of prehospital hypotension
- ✓ Changes in vital signs
- ✓ Treatment given and the patient's response

Key Points - Shock

Goal is appropriate & timely arrival at trip destination.

Aggressive fluid resuscitation in septic shock.

Conservative fluid resuscitation in hemorrhagic and cardiogenic shock.

Recognize and treat hypoperfusion early.

Hypotension is a late sign.

KNOWLEDGE GAPS

There is still research to be done on the optimal use of fluids and pharmacological agents in the treatment of shock. The use of colloids, volume expanders, hypertonic saline and the most appropriate vasoactive medications are all areas of active research.

There are also ongoing studies addressing the concept of **permissive hypotension** (versus aggressive fluid resuscitation) in the patient with hemorrhagic shock due to trauma.

EDUCATION

Patients in shock are among the highest acuity attended to in the prehospital setting. It is essential clinicians remain current with contemporary treatment options.

QUALITY IMPROVEMENT

Important elements in burn management are: [1] targeted fluid administration, [2] oxygen administration, and [3] early transport to appropriate destination.

REFERENCES

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Sperry JL, Minei JP, et al. Early use of vasopressors after injury: caution before constriction. J Trauma. 2008; 64(1): 9-14.

	Stage I	Stage II	Stage III	Stage IV
Blood loss	Less than 15%	15-30%	30-40%	More than 40%
Heart Rate	Normal	Tachycardic (above 100)	Tachycardic (above 120)	Tachycardic (above 140)
Blood Pressure	Normal; slight rise in diastolic pressure	Orthostatic changes	Decreased systolic blood pressure (below 90)	Profoundly decreased systolic blood pressure (less than 80)
Respirations	Normal	Slight tachypnea	Moderate tachypnea	Marked tachypnea; respiratory collapse
Capillary Refill Time	Less than 2 seconds	More than 2 seconds; clammy skin	Usually more than 3 seconds; cool, pale skin	More than 3 seconds; cold, mottled skin
Mental Status	Normal or slightly anxious	Mildly anxious or agitated	Confused, agitated	Obtunded
Bowel Sounds	Present	Hypoactive	Absent	Absent
Urinary Output	More than 30 mL/hr	20-30 mL/hr	Less than 20 mL/hr	None

Figure 1: Stages of hypovolemic shock

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PEP 3x3 TABLES for SHOCK

Throughout the EHS Guidelines, you will see notations after clinical interventions (e.g.: **PEP 2 neutral**). PEP stands for: the Canadian **P**rehospital **E**vidence-based **P**rotocols Project.

The number indicates the Strength of cumulative evidence for the intervention. **1 = strong evidence exists**, usually from randomized controlled trials; **2 = fair evidence exists**, usually from non-randomized studies with a comparison group; and **3 = weak evidence** exists, usually from studies without a comparison group, or from simulation or animal studies.

The coloured word indicates the direction of the evidence for the intervention. **Green = the evidence is supportive** for the use of the intervention; **Yellow = the evidence is neutral**; and **Red = the evidence opposes** use of the intervention.

PEP Recommendations for Shock Interventions, as of 2014/02/24. See: http://emergency.medicine.dal.ca/ehsprotocols/protocols/toc.cfm for latest recommendations, and for individual appraised articles.

Hemorrhagic Shock

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF RECOMMENDATION FOR INTERVENTION	1 (strong evidence exists)	• Restricted Crystalloids	• Aggressive Crystalloids • Colloid Infusion • Hypertonic Saline	• MAST	• Blood
	2 (fair evidence exists)		• Intraosseous Infusion • Trendelenburg		
	3 (weak evidence exists)				

Neurogenic Shock

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF RECOMMENDATION FOR INTERVENTION	1 (strong evidence exists)				
	2 (fair evidence exists)	• Aggressive Crystalloids • Colloid Infusion • Pressors			
	3 (weak evidence exists)				

*PEP is the **Canadian Prehospital Evidence-based Protocols Project.** Every clinical intervention is given a recommendation based on the strength of available research evidence (1 = randomized controlled trials and systematic reviews of RCTs; 2 = studies with a comparison group; 3 studies without a comparison group or simulation) and direction of the compiled evidence: **supportive** of intervention; **neutral** evidence for intervention; or **opposing** evidence for intervention). See: **http://emergency.medicine.dal.ca/ehsprotocols/protocols/toc.cf***

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Septic Shock

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF RECOMMENDATION FOR INTERVENTION	1 (strong evidence exists)	• Crystalloid Infusion • Dopamine			• Antibiotics • High flow oxygen • Hypertonic Saline • Titrated oxygen
	2 (fair evidence exists)		• Colloid Infusion		
	3 (weak evidence exists)		• Trendelenburg		

Cardiogenic Shock

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF RECOMMENDATION FOR INTERVENTION	1 (strong evidence exists)				• Crystalloid Infusion • Direct to PCI • Dopamine • Norepinephrine • Pericardiocentesis
	2 (fair evidence exists)				
	3 (weak evidence exists)				

*PEP is the **Canadian Prehospital Evidence-based Protocols Project.** Every clinical intervention is given a recommendation based on the strength of available research evidence (1 = randomized controlled trials and systematic reviews of RCTs; 2 = studies with a comparison group; 3 studies without a comparison group or simulation) and direction of the compiled evidence: **supportive** of intervention; **neutral** evidence for intervention; or **opposing** evidence for intervention). See: **http://emergency.medicine.dal.ca/ehsprotocols/protocols/toc.cf***

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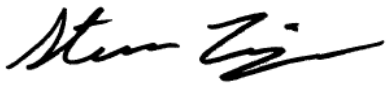
Signature of Program Director 	Signature of Program Document Coordinator 	

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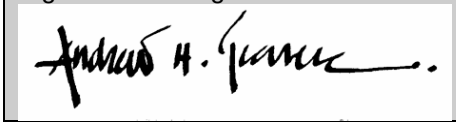
Signature of Program Director 	Signature of program Document Coordinator