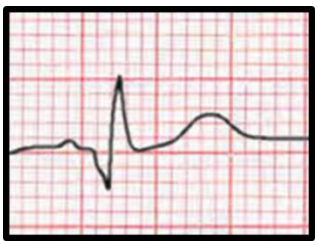
Northwest Community EMS System Continuing Education
August 2014: Shock

Objectives

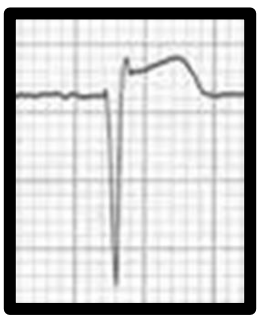
Upon completion of class, review of handouts, SOPs, & class credit questions, each participant will independently do the following to a level of expertise expected for their scope of practice w/ at least an 80% accuracy & no critical errors:

1. (C) Sequence and describe the pathophysiologic events leading to shock.
2. (C) Compare and contrast the pathophysiology and clinical presentations of compensated vs. uncompensated shock.
3. (C) Differentiate etiologies of cardiogenic, hypovolemic, obstructive and septic shocks.
4. (C) Correctly obtain and interpret assessment findings associated with shock.
5. (A) Consider and defend importance of inclusion of auscultated BP (in addition to automatic BP) in the determination of shock severity.
6. (C) Explain pathophysiology of shock signs and symptoms.
7. (C) Identify systemic inflammatory response syndrome (SIRS) findings as early indicators of sepsis.
8. (C) Formulate a field impression of specific types of shock based on assessment findings and patient history.
9. (C) Describe the drug profiles of pharmacologic agents and the intended impact of interventions used to treat specific shock signs and symptoms.
10. (C) Create a treatment plan for a field impression of cardiogenic, hypovolemic, obstructive and septic shocks, according to the NWC EMSS SOPs.
11. (C) Explain the significance and defend the importance of trending MAP and capnography readings for patients in shock.
12. (C) Analyze the protocol for sepsis and septic shock and incorporate sepsis alerts into hospital OLMC reports.
13. (C, A) Explain the advantages & disadvantages of humeral IO insertion site as opposed to a proximal tibial site for patients in shock.
14. (C)Explain procedural differences for IO insertion in the humerus compared to the tibia.
15. (C)Identify, on at least 2 adults, the correct insertion site for humeral IO.
16. (C) Discuss the ongoing monitoring of patients in shock to determine the effectiveness of resuscitation interventions and the need for additional care.

ECG Topic of the Month: Q Waves

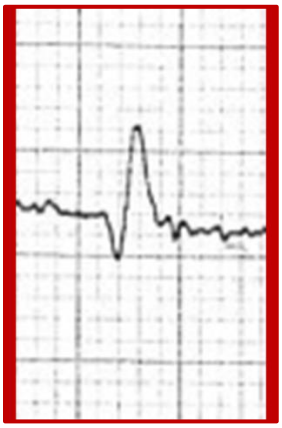


Q waves are an indication that myocardial tissue death (myocardial infarction) has occurred. Not all patients w/ MIs will develop Q waves. The size of pathological Q waves is dependent on the amount of tissue damage, so a Q wave may not be evident if the infarct results in minimal myocardial damage. Significant, or pathologic Q waves, must meet at least one of the following criteria: (1) width of 0.04 sec. or wider (one small box) and or (2) height equal to or greater than 1/3 the height of the QRS wave in that lead. The width of the Q wave is more significant as far as evidence of MI; one small square (0.04 ms) is pathological. Q wave development begins within the first 2 hours after MI and is usually complete in 24 hrs.



Insignificant (non-pathological) Q waves are commonly found in leads I, aVL, and V6. They are usually due to innervation in the septum. They are often a representation of the first vector of ventricular depolarization. These are called septal Q waves. Q waves are also noted when there is no R wave (upward deflection) between Q and the S waves. This makes it hard to tell if it is a Q wave or and S wave. These are called QS waves. Because the QRS in lead V1 is normally a negatively deflected complex, QS waves are common in that lead. If isolated to only V1, the wave is benign. If the configuration extends into leads V2 and V3, it is representative of an old anteroseptal infarct. Benign Q waves are also often found in lead III, so long as it is isolated to that lead. It is usually a narrow complex. If the Q wave is also noted

in leads II and aVF, it is significant for an old inferior MI.
Are the following pathologic or non-pathologic?



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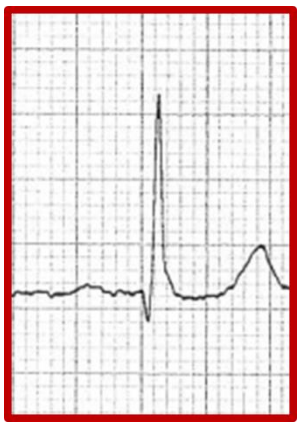

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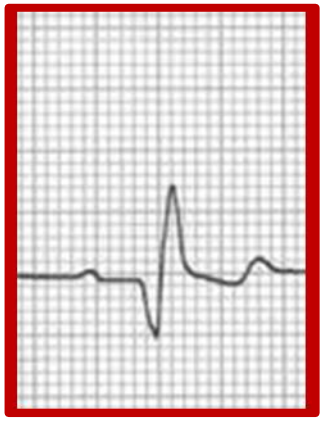

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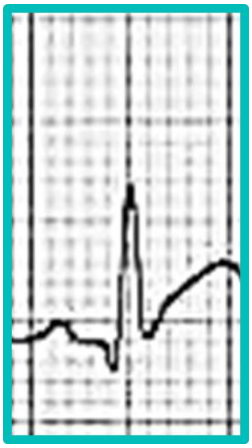

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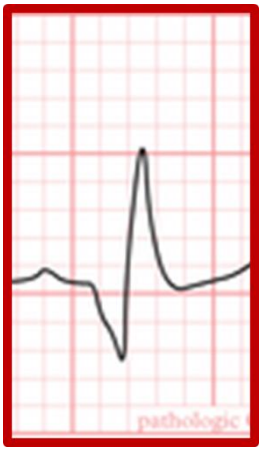

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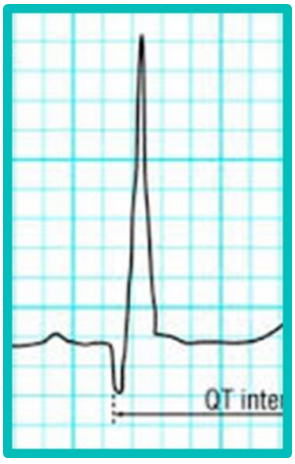

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Garcia, T.B. & Miller, G.T. (2004). Arrhythmia recognition; The art of interpretation. Sudbury, Jones & Bartlett.
Beasley, B.M. & West, M.C. (2001). Understanding 12 lead EKGs; A practical approach**.** Upper Saddle River, Prentice Hall.

Non-Traumatic Shock

Shock is about dysfunction in cellular **perfusion**. For cells to function normally, they must have a constant and sufficient supply of oxygen and fuel to support metabolism based on cellular demand along with a mechanism for the removal of waste products. This is perfusion. Without adequate perfusion, normal cellular activity cannot occur. Adequate perfusion requires (1) an effective pump (heart function must be adequate to move blood forward) (2) sufficient blood volume to fill the vessels, the ability to carry O2 to the tissues, release it to the cells, and to remove the waste products, and (3) an intact vasculature capable of dilating and constricting to distribute blood in response to regional demands (ANS influence) and to maintain minimum mean arterial pressure.

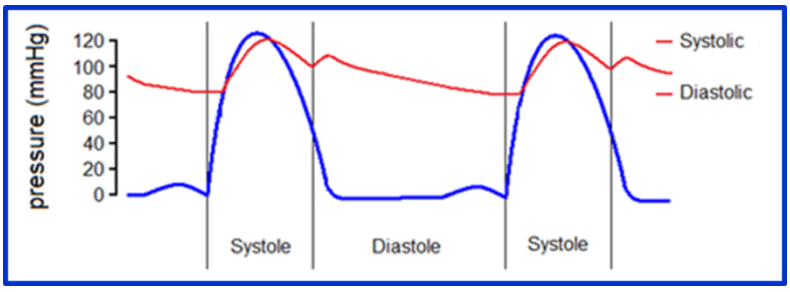
Normal cellular metabolism is achieved in a multi-step process known as **aerobic metabolism**. The primary energy source, glucose, is broken down into pyruvate. This step does not require oxygen, and produces little useable energy. In the next step, which DOES require oxygen, mitochondria metabolize pyruvate to produce CO_2 (waste), water and energy in the form of ATP. ATP is essential to fuel the active transport mechanism known as the Sodium-Potassium pump that maintains a normal volume of water inside and outside of the cells.

So, at the most basic level, **shock is precipitated by cellular hypoxia due to sustained hypoperfusion**. The common denominator, regardless of the cause, is failure of the circulatory system to deliver oxygen at a rate that meets or exceeds total O_2 consumption AND to remove waste products (CO_2 and other acids) from the cells. When these needs are not met for a sustained time, "oxygen debt" occurs, which forces the cells to resort to an alternative, less efficient form of metabolism (anaerobic). **Anaerobic metabolism** creates much more waste and much less energy than aerobic metabolism. The sodium potassium pump fails, allowing sodium to enter and remain inside the cells followed by an influx of water that floods the cells, resulting in cell death. Waste products and acids collect in the cells since perfusion is not adequate to transport it to the lungs for removal. The accumulated waste products create an acidic environment in which the cells cannot function normally.

Looking for clues of hypoperfusion

Blood Pressure

BP is measured in terms of the highest pressure occurring as a result of LV contraction (systolic BP) and the minimum pressure that occurs during continuous forward flow during cardiac rest/filling (diastolic BP). Note that at normal heart rates, approximately 1/3 of the mechanical cardiac cycle time is spent in the systole/pumping phase, and 2/3 (double the systole time) is spend in the relaxation / filling phase. **Mean arterial pressure** (MAP) is not simply an average of the two, but rather represents the pressure average throughout the entire cardiac cycle.



It is actually the MAP that propels blood into the arterioles, capillaries, venules, veins and back to the heart. This microcirculation is the delivery system to each and every cell. If MAP is insufficient to circulate blood to and from the cells, hypoperfusion occurs, and if not corrected, cell dysfunction and death ensue. The **normal MAP** is 70-110 mm Hg and the **minimum target** is at least 65 mm Hg in order to perfuse coronary arteries and the brain. The one exception is in a post-cardiac arrest situation where the MAP target is 90 -100 mmHg. The accuracy of the MAP value diminishes somewhat as the HR increases and the "desired" ratio of time spent in systole and diastole – 1:2 – becomes distorted.

Blood pressure as a shock indicator

Compensatory mechanisms allow significant reductions in central circulating blood volume and cardiac output to occur well before changes in Systolic BP (MAP) are seen! If known, consider the difference in current BP (MAP) and that which is normal for this pt. A drop of 30 mm Hg or more BELOW the patient's norm is significant and may signal hypoperfusion.

SNS Compensatory Mechanisms		
	Alpha	Beta
Heart		↑ rate ↑ pump function ↑ speed conduction
Bronchioles	Constrict	Dilate
Arterioles	Constrict	Dilate

If the Sympathetic Nervous System (SNS) is intact, reductions in cardiac output will be compensated for by vasoconstriction (first the veins then arterioles) which increases the peripheral vascular resistance (elevates diastolic BP). This allows the patient to maintain what seems to be a "normal" BP (MAP) for a time. The downside to this is that vasoconstriction results in smaller vessel diameter and *decreased blood flow* to the cells. The body is not able to vasoconstrict in neurogenic shock due to SNS dysfunction. A reduction in cardiac output coupled with vasoconstriction results in a **narrowed pulse pressure**. Pulse pressures generally range between 30 and 50 mmHg. Trending of pulse pressure changes and MAP is critical to ongoing patient monitoring. See Shock SOP p. 36.

Other clinical effects of SNS activation that should cause suspicion include increased RR; sustained increase in HR, full/bounding pulses (related to stronger pumping), and skin that is pale, cool, and moist. Look for these in the absence of obvious hypotension.

Automatic vs. Manual BP readings

Multiple sources state that automatic cuff BP readings may not correlate accurately with manual readings, particularly in patients with hypotension. Injury severity, degree of acidosis, and resuscitation end points were more accurately reflected by manual BP readings. Examples:

> Injury severity, degree of acidosis, and resuscitation volume were more accurately reflected by manual BP. Automated BP determinations were consistently higher than manual BP, particularly in hypotensive patients. Automated BP devices should not be used for field or hospital triage decisions. Manual BP determinations should be used until systolic blood pressure is consistently ≥110 mm Hg. PMID: 14608157 [PubMed - indexed for MEDLINE] J Trauma. 2003 Nov;55(5):860-3.

> "In the critically ill patient, non-invasive measurements of arterial pressure…must be interpreted with caution. Oscillometric devices can underestimate SBP by as much as 6-19%, and can overestimated DBP by as much as 5-27%. Noninvasive measurements of arterial pressure become less reliable in pts who have marked hypovolemia or abnormal cardiac function." EM Reports: Emergency Medicine: Pearls & Pitfalls: Vital Signs, MAP, Shock Index, and Circulatory Shock.

These findings provide convincing rationale for prehospital providers to obtain manual BP readings for the initial assessments and to stay with manual readings if the patient is hypotensive. Comparison should be made as to how closely aligned the manual and automated readings are to determine if it is safe to transition to automated readings.

Factors that influence the accuracy of BP readings

- Cuff size: width should be 40% of upper arm length and the cuff should easily overlap the arm circumference. A cuff that is too large will yield a falsely low reading, while a cuff that is too small will result in a reading that is falsely high. Use cuffs of the appropriate size and length and adapt where the cuff is applied in patients that are extremely obese. (See SOP)
- Accuracy of NIBP readings may be adversely affected if the patient has a rapid or irregular heart rhythm (due to beat to beat variability). One study found that 25% of pts with A-fib had auto BP readings that differed by > 10mm Hg, both falsely high and falsely low.

Take away points? Look at the WHOLE patient! Look for other shock indicators BEFORE and IN THE ABSENCE of ↓BP.

End Tidal CO_2 in Shock

Abnormalities in 3 systems – metabolic, respiratory, and circulatory – contribute to the low $EtCO_2$ levels seen in shock. Note that an increased RR is one of the earliest clinical responses to shock in an effort to blow off excess CO_2 to neutralize the acidosis. Tachypnea is easily detected by applying a capnography monitor. If noted, a careful and thorough assessment is indicated.

As stated above, in a metabolic acidosis, the ventilatory rate increases in attempt to eliminate excess CO_2, but because of decreased blood flow (hypoperfusion) to and from the cells, very little CO_2 actually gets delivered to the lungs to be exhaled, so $ETCO_2$ readings are low.

Factors Affecting ETCO2	
↑ ETCO2	↓ ETCO2
Metabolism: Pain Hyperthermia Shivering	Metabolism: Hypothermia Metabolic acidosis

Respiration: Resp insufficiency Resp depression COPD Analgesia/sedation	Respiratory: Alveolar hyperventilation Bronchospasm Mucus plugging
Circulation: Incr. cardiac output	Circulation: Hypotension Sudden Hypovolemia Cardiac arrest Pulmonary embolus
Medications: Bicarb	

Review NWC EMSS Shock SOP: p. 36

Use a central sensor for SpO_2 if pt has poor peripheral perfusion (ex: cold hands)

- Trend serial EtCO2 readings; low levels are a marker of hyperventilation, poor perfusion to lungs, and metabolic acidosis (<31)
- Trend pulse pressures and mean arterial pressure
    - Pulse pressures are easily calculated: SBP minus DBP = PP. Normal ranges 35-50.
    - Prior to decompensated shock, SBP is maintained above 100 and DBP increases which helps to maintain the MAP– watch for either PP < 30 or trends toward decreasing SBP (MAP) values
- Vascular access & fluids: IV fluid volumes depend on the etiology of shock

Stages of Shock

The stages of shock reflect the severity of disruption in tissue perfusion and the degree of cellular membrane damage. If precipitating factors are promptly reversed, compensatory mechanisms can usually restore perfusion. The longer a pt remains in shock, the longer vital organs are deprived of O2. Shock is reversible if perfusion is restored prior to onset of cell destruction. Once cellular destruction begins, shock is irreversible and organs will fail.

Compensated (Reversible) Shock

At the beginning of the shock event, there is a drop in cardiac output that adversely affects cellular function. Through compensatory mechanisms and neutralization of elevated lactate level, the body attempts to maintain hemodynamic stability. Cardiac output is restored and perfusion is maintained at near normal levels, so early clinical signs and symptoms are subtle. The net result is that cardiac output & tissue perfusion are restored *at the expense of non-vital organs.* Please refer to document *NWC EMS System Pathophsyiology & Management of Shock,* posted to the NWC EMSS website, for full documentation on the pathophysiology of shock.

Assessment findings in compensated shock:

- ↑ HR & pulse strength
- SBP ≥100; ↑ DBP
- ↓ pulse pressure
- ↑ RR & depth
- Skin: normal to pale, cool, moist
- Mental status: normal to anxious / restless
- Others: poor turgor, dry mucous membranes (hypovolemic), thirst

Decompensated /Progressive Shock – See SOP p. 36

Decompensated shock occurs when the circulatory system starts to fail in spite of the compensatory mechanisms discussed previously. It is at this point that systolic BP falls below 100. What were once protective mechanisms may now lead to complications.

Assessment Findings in Decompensated Shock

- SBP falls below 100
- Mild to severe hypoxia
- Altered mental status
- Skin: pallor, mottling, cool to cold
- Prolonged cap refill in children
- Weak peripheral pulses

Irreversible Shock

At this stage of shock, the patient is unresponsive to interventions. Even the protected organs are hypoperfused. Blood flow becomes sluggish, and clots form in the vessels, further impairing perfusion. Decreased perfusion to the lungs results in reduced surfactant production leading to atelectasis (alveoli close down and inner walls stick together making it difficult to reinflate on inspiration) and non-cardiogenic pulmonary edema. Myocardial depressant factor, released from a hypoxic pancreas, reduces the heart's pumping. As brain perfusion falls, SNS activity ceases, and mental status deteriorates. One by one, the organs fail, and the pt dies as a result of Multiple Organ Dysfunction Syndrome (MODS).

Assessment Findings in Irreversible Shock

- Profound hypotension
- Severe hypoxemia
- ↓ responsiveness
- Bradycardia
- Circulatory failure

Cardiogenic Shock: In cardiogenic shock, pump function is so compromised that the heart cannot meet metabolic needs, and compensatory mechanisms are no longer working. The hypotension in this form of shock decreases coronary perfusion as well, which further suppresses cardiac performance. Cardiogenic shock exists when rate, rhythm and volume problems have been addressed, yet the pt remains hypotensive.

Causes of Cardiogenic Shock:

- The left ventricle is responsible for the delivery of oxygenated blood to the entire body. Diminished pump function results in inadequate blood/oxygen supply to body's cells/organs. If unrelieved, alternative metabolism (anaerobic) takes over, creating large amounts of waste and very little energy. The cellular environment becomes acidotic, and cell function becomes abnormal (abnormal electrical activity/dysrhythmias and poor pumping).
- Decompensated HF refers to failure of compensatory mechanisms / therapies that had previously allowed the functionally impaired heart to support perfusion needs.
- Type II MI: Diffuse cardiac ischemia refers to generalized hypoperfusion of the myocardium, when there exists an imbalance in myocardial O2 supply & demand. Delivery of needed oxygen and fuel and removal of wastes is inadequate to the entire myocardium, not just a certain area distal to a clot, as in myocardial infarction. The entire myocardium, both pumping and conductive tissues, are affected. Cellular and organ dysfunction soon follow in the form of dysrhythmias and pump failure. This type of ischemia occurs in the setting of sustained hypotension/hypoperfusion. ECG changes are typically minimal, non-specific, or absent, and the pt may not experience chest pain or equivalents.

Causes of Type II MI	
Cause	**Mechanism**
Tachycardia	Causes hypoperfusion by (1) shortening diastole time (when myocardium gets perfused) and (2) incr O2 demand to support faster pumping assoc w/ fast HR
Bradycardia	Causes hypoperfusion by reducing the number of diastoles (O2 delivery to myocardium) occurring each minute
Anemia	Causes hypoperfusion by reducing the O2-carrying capacity of blood with each heartbeat (less O2 delivered with each beat as O2 is delivered bound to hemoglobin; if less than normal Hgb, then there are fewer "vehicles" on which to transport O2
Hypotension	Hypotension is often assoc w/ lower diastolic pressures & MAP. A BP of 90/60, which has a MAP of 70, just barely provides adequate pressure to perfuse the heart. Readings less than this put the pt at risk for inadequate perfusion.

Causes of Type II MI	
Cause	**Mechanism**
Coronary artery spasm	Spasm reduces the caliber of the vessel, thereby reducing the volume of blood it will accommodate. Less blood flow equates with less O2 delivered. If the spasm is widespread, the bulk of the myocardium is hypoperfused.
Coronary embolus	A clot in the proximal portion of one of the major epicardial vessels (RCA, L main, Circumflex, & LAD) will occlude flow to a large portion of myocardium. The closer the obstruction to the root of the vessel, the more muscle is hypoperfused. If there are obstructions in more than one of the main arteries, enough myocardium could be hypoperfused to cause global myocardial ischemia.
Hypertension	HTN is a direct result of the tone of the arteries into which the heart must pump blood. The more constricted (tone) the arteries, the harder the heart must work to eject blood. Depending on the tone of the vessels and the health of the myocardial circulation, the resulting myocardial O2 demand may exceed that delivered

Assessment Findings:

- SBP < 90 / MAP < 65 **OR** ↓30-60 mm Hg from baseline (Pts in early cardiogenic shock may not be hypotensive!)
- HR: ↑, but may fall as pt deteriorates
- Lt side HF: dyspnea, crackles, wheezes
- Rt side HF: periph edema, hepatomegaly, JVD
- Weak, thready pulses
- ↓ SpO2
- Agitation, confusion, apprehension
- Pale, cool moist skin
- ETCO2 ↓

Cardiogenic Shock (HF/Pulmonary Edema) SOP: see p. 21 in 2014 NWC EMSS SOP's

Non-hemorrhagic Hypovolemic Shock:

This type of hypovolemia is caused by a deficit in intravascular volume of either blood or plasma. Fluid "loss" may be "relative" as fluid leaves the compartment where it belongs, as in burns and large vessel rupture, or it may be due to actual fluid loss – for example, wound drainage, vomiting, diarrhea, or excessive urine output in DKA/HHNS/renal failure.

Assessment findings:

- ↓ BP
- ↑ HR
- ↑ RR
- Pale, cool moist skin
- Lungs: clear
- ETCO2: low
- Events/PMH findings suggestive of volume loss-related conditions

Shock SOP: please refer to SOP's specific to Burns (p 41), DKA (p 26), Dialysis (p 23), and Abd Pain (p 23)

NOTE: "Permissive hypotension" is ONLY used in pts w/ trauma – related hemorrhagic shock & vascular internal bleeding

Obstructive Shock:

This type of shock occurs when pressure in the thorax and or pericardium becomes high enough to "obstruct" venous return to the rights side of the heart (tension pneumothorax and pericardial tamponade), impairing cardiac filling, and thus reducing cardiac output. Pulmonary embolus affects preload (venous return) to the left heart by obstructing flow through a significant portion of the pulmonary vasculature. Reduced filling volume in the Lt heart results in low stroke volume cardiac output.

Tension Pneumothorax: All start with a simple pneumothorax. When air continues to collect in the pleural space under pressures that are higher than the central venous pressures within the vena cava & Rt atria, the central veins and RA collapse. That decreases the volume of blood entering the right side of the heart (preload). Reduced RV filling results in a reduction in the volume of blood ejected into the pulmonary arteries. This produces a chain reaction in that less blood is then forwarded to the LV so there is less to circulate to the body. The patient experiences a dramatic fall in the cardiac output. The high pleural pressure on the one side may shift the mediastinum onto the intact lung, further compromising ventilations. Cause of death: obstructive shock and hypoperfusion

Causes: Sudden increase in intrapulmonary pressure / positive pressure ventilations

Assessment findings:

- Severe pain w/ breathing
- Absent breath sounds on affected side
- Agitation, anxiety
- Distant heart sounds (after mediastinal shift)
- Extreme dyspnea
- Tachypnea
- *JVD
- Narrow PP
- Hypotension
- Asymmetric chest expansion
- Hyperextended hemithorax
- Tachycardia w/ weak, thready pulses
- Late signs: Cyanosis

*Note: If the pt is hypovolemic, JVD may not be present!

Tension Pneumo Intervention: SOP p. 42: The patient is dying from an over-pressurized pleural space and no cardiac output. Thus, the lifesaving intervention is to vent off the pressure. Needle pleural decompression (also known as needle thoracostomy) releases air to the atmosphere, restores acceptable intrapleural pressures, re-establishes venous return to the right heart, and hopefully restores cardiac output.

Pericardial Tamponade: May be difficult to differentiate from tension pneumo, as both conditions share some of the same exam findings. High intrapericardial pressure collapses VC, resulting in ↓ venous return to the Rt atrium & RV. The pericardial sac surrounding the heart fills w/ fluid, exerting sufficient pressure on the heart to prevent stretching & filling (preload) and contraction, resulting in decreased cardiac output.

For all patients, malignant diseases are the most common cause of pericardial tamponade. Other factors and conditions related to development of pericardial tamponade include cardiac rupture, anticoagulation, pericarditis, pericardial effusion, cardiac surgical procedures, and blunt or penetrating trauma.

Assessment Findings: **cardiac tamponade**

- *Hypotension; narrowed pulse pressure
- *JVD
- *Muffled heart tones
- ↑ HR
- Low voltage QRS & T waves
- Anxiety, agitation
- Dyspnea, chest tightness
- Recent MI, thoracic surgery
- *Breath sounds present bilaterally*

(* Beck's Triad)

Management: NS IV wide open to achieve SBP of **80 ONLY.** Higher pressures serve only to force blood/fluid into the pericardial sac, worsening the constrictive effect on the heart and vena cavae. This reduces venous return to the right heart and lowers cardiac output from the left heart. Thus the patient will decompensate more quickly.

Massive Pulmonary Embolus: Pulmonary emboli may be composed of coagulated blood, fat, air, bone marrow, bacteria, debris, or amniotic fluid. Smaller pulmonary emboli obstruct blood flow to lung tissue distal to the obstruction. The affected tissues are ventilated (oxygenated air enters alveoli) but not perfused (dead space) because there is no blood flow through alveolar capillaries in the affected area. Thus no gas exchange takes place and the SpO2 reading will be lower than normal. A massively large embolus or one that is seated strategically where it occludes flow to a large area of lung tissue (saddle embolus) will create obstructive shock because it prevents blood flow back to the LV. Smaller emboli create more of a shunting problem than a volume issue. When referring to a pulmonary embolus in obstructive shock, we are referring to a large embolus or one that occludes blood flow to a large area of lung tissue.

Assessment findings: S&S depend on (1) severity of blockage & resultant shunting (2) location of clot and (3) underlying health, age & presence of comorbid conditions. Hypotension depends on the severity of reduction in cardiac output.

Sixty-70% of the pulmonary bed needs to be obstructed to cause shock. Tachypnea will be out of proportion to fever & tachycardia. S&S range from no symptoms to severe S&S and sudden death.

- Hemoptysis (usually w/ smaller emboli)
- Sudden onset dyspnea, pleuritic chest pain (~50%)
- Hypoxemia
- ETCO2 ↓ w/ small blunted waveform
- Anxiety
- Tachypnea
- Tachycardia (pulses may be thready)
- Pale, diaphoretic skin
- Breath sounds usually clear
- May have localized wheezing or pleural friction rub
- Fever (43%)
- Cough (53%)

The pt may also have petechiae over arms & chest in the setting of profound hypoxia , hypotension. There may be indications of venous thromboembolism (VTE) (formerly DVT): calf redness, warmth, tenderness, swelling; palpation of a "thick cord"; severe pain upon dorsiflexion of foot w/ knee extended.

Risk Factors for PE:

- Virchow's triad:
  - Vessel wall trauma/damage (atherosclerosis): especially thorax, abdomen, pelvis, legs; atherosclerotic disease changes; local infection; diabetes; smoking
  - Venous stasis: lack of movement/restricted mobility of extremities: extended travel confined w/ knees bent, prolonged bed rest (> 3 days), recent major surgery; long bone fx requiring immobilization / casting; pelvic fx; poor peripheral circulation; varicose veins; sedentary life style; ↑ RBC's in emphysema thicken blood, predisposing to stasis; burns; hypercoagulability: smoking; pregnancy; BCP; cancer
  - Hypercoagulability: Use of birth control pills, pregnancy current or recent, hx PE, DVT; cancer; infections; AFib; sickle cell disease
- Hx VTE
- Pelvic/Long bone fx (fat embolus)

Management: Time sensitive!!! Prehospital interventions aim at maximizing oxygenation/ventilation to offset shunting. The patient may be a candidate for anticoagulation, fibrinolytics, or embolization at hospital.

- Supplemental O2
- IV: fluid challenges w/ goal SBP 90
- Monitor SpO2, ETCO2 (expect very small square waveforms with low numbers), ECG
- OLMC may order ASA
- ECG: anticipate cardiac arrest in pt w/ large embolus

Sepsis & Septic Shock:

Sepsis is a multi-factorial systemic response to infection, featuring derangements in coagulation, inflammatory response, cell function, and metabolism, culminating in cell death due to hypoperfusion.

Coagulation: Multiple abnormalities trigger inappropriate activation of the clotting cascade, with clots forming spontaneously. Eventually the body's smallest vessels get clogged – those that supply each cell. Entire organs and body systems may fail due to lack of perfusion as a result of these microvascular clots. Because the body's supply of platelets and clotting factors gets used up more quickly than they can be replenished, bleeding occurs as a result, and can be fatal.

Inflammatory Reaction: Effects include increased vascular permeability, impaired response to vasoconstrictors, and impaired myocardial pump function, resulting in circulating volume displacement and decreased cellular perfusion.

Cell dysfunction: Cells either over- or under-respond to a stimulus. For example, the cells lining the vessel walls lose their anticoagulant properties and become "sticky", so blood cells begin to clump in the affected areas. Excessive numbers of cells important in defending against infection die or are destroyed. Vessel walls also become "leaky" and

lose their ability to respond to the body's signals to vasoconstrict. Myocardial cell function is impaired, resulting in poor pump function.

Metabolism: Septic pts become hyperglycemic (a response to stress) and do not respond normally to insulin. Function of infection –fighting cells is impaired in this hyperglycemic environment. Normal cellular metabolism becomes impossible due to inadequate fuel and oxygen, so cells resort to anaerobic metabolism, which contributes to the development of acidosis.

The Four Stages of Sepsis

Sepsis' progression is subtle, rapid, and often deadly. It moves very quickly, and is often diagnosed too late.

Stage 1: Systemic Inflammatory Response Syndrome (SIRS)	Presence of at least 2 of the SIRS criteria: • T > 38C/110.4 F or < 36°C/96.8°F • HR > 90 • RR > 20
Stage 2: Sepsis	Stage 1 PLUS known or suspected infection source
Stage 3: Severe Sepsis	Stage 2 PLUS at least one sign of organ failure (this is where you MAY see hypotension): • SBP < 90 • MAP < 65 • Drop from baseline BP > 40 mm • Mental status changes • RR > 24 • Pulse ox < 92% ON O2 at 6L
Stage 4: Septic Shock	Stage 3 plus persistent hypotension UNRESPONSIVE to fluid resuscitation

Source: AJN: March 2009, vol. 109, No. 3, pp 40-45

Sepsis and EMS: EMS can play crucial role in survivability for these patients, as in AMI and stroke. Early recognition and initiation of prehospital care saves time and lives. Plentiful data exists supported by credible data/studies, showing that early recognition and therapy result in better outcomes and lower mortality for patients with sepsis.

EGDT: Stands for Early Goal Directed Therapy, the approach used by hospitals for suspected sepsis patients – similar to mobilization of resources and personnel for stroke and MI. The sepsis protocol involves time constraints as do protocol for AMI and stroke. Four components of EGDT: 1) early identification of pt w/ sepsis 2) optimization of oxygenation, ventilation, circulation 3) initiation of fluids, vasopressors, antibiotics 4) controlling source of infection.

Sepsis Protocol & Sepsis Alert: Please refer to Shock SOP on p. 36

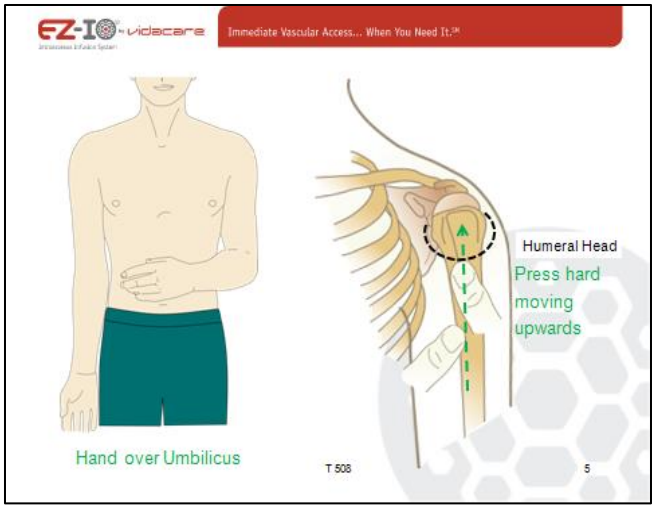
Proximal Humeral IO

Known benefits to the proximal humeral IO site include the following:

- Flow rates to the heart are superior via the proximal humerus
- Higher volumes of fluid may be infused through the proximal humerus
- Accessibility
- No compartment syndrome
- Less pain

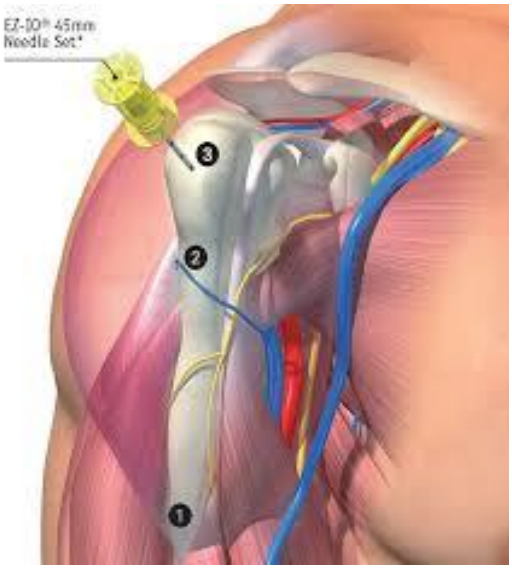
Site identification:

The site of optimal insertion is 1 cm above the surgical neck/notch in the greater tubercle. It is most easily palpated at a point midway along the length of the arm. Palpation of the bone requires firm pressure due to overlying structures.



Place the patient's hand over the umbilicus (medially rotates elbow and humerus) and adduct the arm (provides greater prominence of site). To locate the head, run thumb up humeral shaft, until you feel a "notch" or "groove" – the slight outward protrusion at surgical neck. The greater tubercle is above that and the site of insertion. Confirm the site by pronating and supinating the hand and feeling the greater tubercle rotate under your finger.

Insertion procedure is identical as for other approved sites. Note that the hub of the needle will sit at a slight angle when it is in place. Care must be taken to stabilize the arm to prevent needle dislodgement. Following insertion, the patient's arm should be immobilized to prevent movement above the level of the shoulders. Restrain in the adducted position is preferred.



Source: Vidacare EZIO

NWC EMSS Continuing Education
August 2014: Shock
Scenarios

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Breathing	
Circulation	
Disability	
Expose	
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