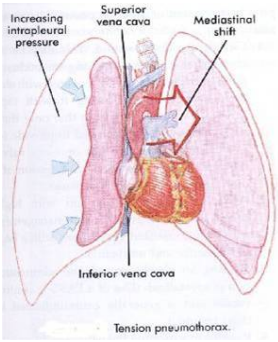
NWC EMSS Continuing Education
October 2019: Chest, Abd and Musculoskeletal Trauma
Resource Document

Source: 2018-2019 NCH Paramedic Program. Thoracic Trauma. Connie J. Mattera, M.S., R.N., EMT-P

I. Injuries that must be found at B (Breathing) as they jeopardize ventilations and/or gas exchange

A. Tension pneumothorax



1. **Etiology**
   a. Results from any of the causes of a simple pneumothorax
   b. Damage to lung parenchyma (tissue) usually from blunt trauma
   c. In ventilated patients, it may occur secondary to positive pressure ventilations resulting in a sudden increase in intrapulmonary pressure (barotrauma).
   d. Penetration of pleura by rib fracture
   e. Tracheobronchial tree injuries from shear forces

2. **Pathophysiology**
   a. Most commonly occurs from blunt trauma.
   b. Starts with a simple pneumothorax
   c. A closed pneumothorax progressively accumulates air within the pleural space on inspiration that cannot escape on expiration, creating a one-way valve.
   d. This accumulation produces an increase in intrapleural pressure (tension) that collapses the lung on the affected side and depresses the diaphragm.
   e. When pressure in the pleural space exceeds pressure in the atria and vena cavae, they collapse. This creates a mechanical obstruction of blood return to the right heart resulting in markedly decreased preload and cardiac output – **OBSTRUCTIVE SHOCK**
   f. Then…Rising intrathoracic pressure depresses diaphragm & shifts mediastinum away from affected side, further compressing vena cavae & compromising intact lung so opposite side becomes pressured as well
   g. **Life-threatening emergency due to cardiovascular compromise**. Must be suspected clinically as soon as possible.

3. **Assessment – classic findings**
   a. **Inspection**
      (1) Complaint of severe pain with breathing (pleuritic chest pain)
      (2) Restlessness, severe anxiety, agitation
      (3) Dyspnea, tachypnea, retractions and other signs of respiratory distress
      (4) Asymmetric chest movement, hyperdistended hemithorax on the affected side, bulging of intercostal muscles
      (5) **JVD:** Collapse of the superior vena cava and right atrium prevents blood from returning to the right side of the heart producing increased central venous pressure. Hypovolemia will prevent JVD.
      (6) Desaturation and cyanosis (late sign)
   b. **Palpation**

(1) Tachycardia, weak, thready pulses reflect reduced cardiac output

(2) Subcutaneous emphysema in face, neck and upper chest

(3) **Tracheal deviation** (late sign) – Trachea is solidly tethered and does not move easily above the sternal notch. Hard to detect without a chest x-ray.

c. **Percuss** for **hyperresonance** on affected side

d. **Auscultation**

(1) **Absent or decreased breath sounds on affected side**
(2) Distant heart sounds (if mediastinal shift)
(3) Displacement of apical impulse to left lateral chest wall if shift to the left

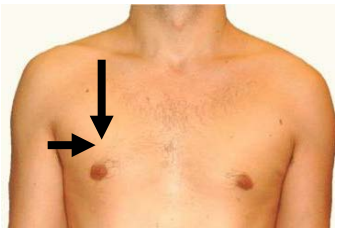
(4) **Hypotension** (narrowed pulse pressure)

4. **Emergency interventions - see lab manual**

a. Apply oxygen 12-15 L/NRM

b. **Needle pleural decompression** is a temporizing measure

(1) Equipment needed: 10 g IV catheter or use a commercial device; Chlorhexidine wipe.



(2) Procedural steps: Insert needle at a 90 degree angle to the chest wall in the 2nd or 3rd ICS in the midclavicular line to release air under pressure in pleural space. USE EXTREME CARE in selecting the correct site and inserting the needle using the correct angle so it penetrates into the pleural space.

An alternate site of insertion in the 4th-5th intercostal space in the midaxillary line is NOT approved in the NWC EMSS due to the possibility of diaphragm, liver or spleen penetration by blind needle insertion in a supine patient.

(3) If the needle hits a rib, gently go over the top of the rib to avoid injury to the neural vascular bundle running under each rib.

(4) Once in place, remove the needle and leave only the catheter in the chest. A retained needle has the potential to penetrate the heart or a ventilated lung.

(5) Release of pressurized air should relieve the patient's acute distress, improve ventilations, and re-establish venous return (and thus peripheral pulses) but it will not re-expand the lung (breath sounds will still be absent). Will need a chest tube at the hospital to expand the lung.

(6) Frequently reassess catheter patency. May need to repeat procedure with additional needle.

c. Venous access; IV NS up to 1 L

d. Time-sensitive patient – needs expeditious transport to a Level I trauma center

5. **Complications**

a. Pneumothorax if misdiagnosed prior to procedure

b. Hemothorax if a pulmonary, internal mammary or intercostal vessel is transected

c. Lung laceration

d. If tension pneumothorax presents unrecognized and therefore not treated, the patient's condition may deteriorate to pulseless electrical activity (PEA) and respiratory - cardiac arrest

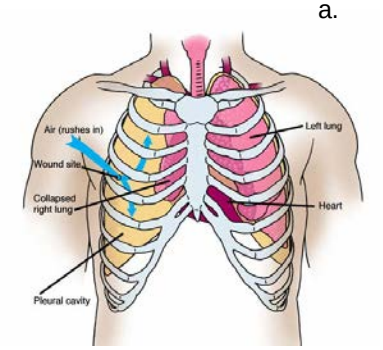
6. ***How can you tell the difference between a pneumothorax & tension pneumothorax*** (both have absent breath sounds)?

7. How can you tell the difference between a hemothorax & tension pneumothorax when both have unequal BS & low BP?

Open pneumothorax (sucking chest wound)

8. **Etiology**: Usually caused by penetrating chest trauma secondary to gun shot or stab wound, but can also be caused by an impaled object.

9. **Pathophysiology**



   a. Penetrating trauma through the chest wall creates an opening allowing air to enter the intrathoracic cavity that depends on negative pressures and intact pleural membranes to allow inspiration through the tracheobronchial tree.

      (1) Since chest wall and pleural integrity is lost, the involved lung paradoxically collapses on inspiration and expands minimally during expiration moving air in and out of the defect, producing a "sucking" sound.

      (2) **Critical diameter**: Air is gas, so it flows along the path of least resistance (high → low pressure). If the wound approximates 2/3 the diameter of the trachea, resistance to air flow through the respiratory tract may be greater than through the open wound, so air preferentially moves through the chest wall defect into the pleural space instead of the trachea to equalize intrathoracic and atmospheric pressure during ventilatory attempts.

      (3) Increased intrapleural pressure leads to lung collapse on the affected side with possible mediastinal shift

      (4) Ventilation/perfusion mismatch

         (a) Shunting
         (b) Hypoventilation
         (c) Hypoxia
         (d) Large functional dead space

      (5) Air may exit the wound during the exhalation phase producing a frothing or bubbling at the site

   b. A one-way flap valve may allow air in but not out resulting in an accumulation of pressure in the pleural space

   c. Direct lung injury may be present

10. **Morbidity/mortality**: Patient dies from inadequate ventilation and impaired gas exchange

11. **Assessment**

   a. **Inspect**

      (1) Visible chest wall defect; sucking sound on inhalation
      (2) Complaint of pain with breathing
      (3) Restlessness, dyspnea, tachypnea and other signs of ventilatory distress, hypoxia, and hypercarbia
      (4) Asymmetrical chest expansion, subcutaneous emphysema
      (5) Cyanosis (late sign)
      (6) Aphasia: Patient cannot speak if they are unable to move air through the trachea.

   b. **Palpate** for subcutaneous emphysema in neck and upper chest; tachycardia

   c. **Auscultate** for **absent or decreased breath sounds** on affected side (or both sides with significant injury).

12. **Emergency interventions**

   a. The *SMITH PAPYRUS*, written sometime around 3,000 B.C., contains the earliest recognized reference to "thoracic trauma". Work describes 58 patients (2 had thoracic injuries). Recommended treatment for penetrating injury was fresh meat poultice the first day, followed by grease, honey and lint on subsequent days (not bad advice!).

   b. If detectable, there is an immediate life-threat. Convert to a closed pneumothorax. Immediately cover with gloved hand. Then…

(1) Ask a cooperative patient to maximally exhale or cough

(2) Immediately apply occlusive dressing (Vented chest seal; Vaseline gauze, plastic wrap, defib pad or commercial device with flutter valve). Dressings should be at least 3 or 4 times the size of the defect.

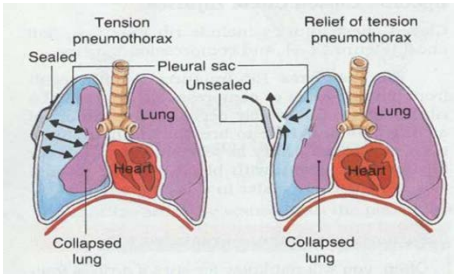
(3) Monitor VS, ventilatory/circulatory status, jugular veins after application of occlusive dressing.

c. Intubate if necessary and monitor ventilations

d. Oxygen 12-15 L/NRM; assist with BVM as necessary. Use positive pressure ventilations with caution in patients who have penetrating chest wounds. High ventilatory pressures may force air from an injured bronchus into an adjacent open pulmonary vein, producing systemic air emboli. This may account for many of the dysrhythmias and sudden deaths that occur in patients with severe penetrating chest wounds.

e. Administer sedatives cautiously per OLMC to allow for control of ventilations

f. Tension pneumothorax usually does not occur in the presence of an open pneumothorax but may develop if penetrating wound has a one-way flap or is sealed with an occlusive dressing. If patient becomes dyspneic and BP drops, assess for tension pneumothorax and temporarily lift or remove dressing to release pressure.



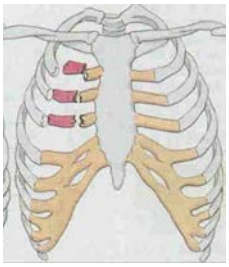
g. Assess need for needle pleural decompression if no improvement following removal of dressing

h. **No probing of wounds**

(1) Gives no information
(2) May create a false passage
(3) Precipitates hemorrhage
(4) May convert a closed to an open pneumothorax

B. **Flail chest**

1. Most severe form of blunt chest wall injury.



2. **Mechanism** of injury: usually due to blunt chest trauma

a. High speed MVC
b. Falls
c. Auto-pedestrian trauma
d. Motorcycle trauma
e. Severe compression trauma

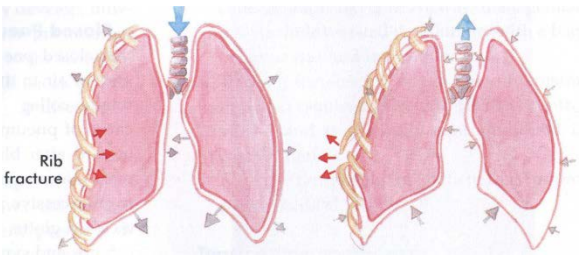
3. **Pathophysiology**

a. Flail chest occurs when two or more adjacent ribs and/or cartilages on both sides of an impact point are broken at two points resulting in a freely mobile or "floating" segment.

b. May be identified by location or size

(1) Anterior, posterior, or lateral
(2) Separation of the sternum from adjacent broken ribs or costochondral joints: sternal flail chest

c. Free segment moves separately and in the opposite direction (paradoxically) to the rest of the thoracic wall during the ventilatory cycle.



Bledsoe, 2006

d. Paradoxical motion of the flail segment interferes with the normal inspiratory/expiratory cycle due to the lack of bony support and changes in intrathoracic pressures.

e. Sub-atmospheric intrathoracic pressure during inspiration pulls the segment inward. Positive intrathoracic pressures during expiration move the segment outward.

f. The most significant life-threat accompanying flail chest is the insult to lung parenchyma that creates a **pulmonary contusion**. Contused lung produces more than the normal amount of interstitial and intra-alveolar fluid resulting in impaired gas exchange. Pulmonary contusion occurs in 30-75% of all blunt thoracic trauma (East, 2006) and is the major cause of respiratory compromise.

g. Local effects

(1) Laceration to lung tissue
(2) Hemorrhage-filled alveoli
(3) Reduced compliance leading to reduced ventilation
(4) Increased shunt fracture with decrease in pO_2 and increase in $AaDO_2$ (alveolar-arterial oxygen difference)
(5) Increased pulmonary vascular resistance
(6) Decreased pulmonary blood flow
(7) Injured and uninjured lung (ipsilateral and contralateral (EAST, 2006)

h. Pain from multiple rib fractures discourages breathing effort. Even if made initially, fatigue, CNS depression, or increased tracheobronchial secretions will eventually outweigh any patient efforts.

i. Hypoventilation, impaired oxygen diffusion, pulmonary physiologic shunting and venous admixture results in a decreased PaO_2, decreased lung compliance, decreased tidal volume and vital capacity, and decreased venous return with ventilation/perfusion (V_A/Q) mismatch. CO_2 retention results in hypercarbia. Impaired cough results in atelectasis.

4. **Morbidity/mortality**

a. Forces sufficient to produce a flail chest, are also sufficient to cause pneumothorax and serious injury to the underlying lung.

b. Mortality rates of 10% to 20% are typically accompanied by a significant pulmonary contusion. Mortality: 5-50% due to associated injuries

c. Mortality increased with

(1) Advanced age: make up about 10% of the cases but consume 30% of the clinical resources (East, 2006)
(2) Seven or more rib fractures
(3) Three or more associated injuries
(4) Shock
(5) Head trauma

5. **Assessment**

a. **Inspect:** Unclothed chest must be viewed from anterior, posterior, and lateral angles to detect presence of a flail segment.

(1) Evidence of chest wall trauma to soft tissues (abrasion, laceration or ecchymosis)
(2) Complaint of severe pleuritic chest pain at fracture sites, splinting
(3) Dyspnea, altered RR (>35 or < 8 min), cough, and other signs of respiratory distress
(4) Paradoxical chest movements - Not always apparent in an awake patient with muscle spasms, shallow respirations, and splinting secondary to pain. Seen as patient tires, usually within first 24 hours.
(5) Poor tidal volume (shallow breathing), pulse oximetry desaturation, and cyanosis (late sign)

b. **Palpate** (or auscultate) for subcutaneous emphysema in neck and upper chest, tenderness, crepitus, and pain at fracture sites, tachycardia.

c. **Auscultate** for absent or decreased breath sounds on affected side, respiratory stridor.

6. **Interventions**

a. **Goals of therapy**

(1) Promote oxygenation and gas exchange.
(2) Achieve and maintain adequate pain control.
(3) Achieve and maintain euvolemia.

b. Gain airway control; suction.

(1) Intubation and mechanical ventilation should be avoided (East, 2006). Mechanical ventilation should be used to correct abnormalities of gas exchange rather than to overcome instability of the chest wall.

(2) Clinical signs of progressive fatigue and deterioration should prompt intubation and mechanical ventilations.

(a) Respiratory rate > 35 or < 8 breaths/min
(b) PaO_2 < 60 mmHg at FiO_2 > 50%
(c) $PaCO_2$ > 50 mmHg at FiO_2 > 50%

c. **CPAP:** A trial of mask CPAP should be considered in alert, compliant patients with marginal respiratory status regimen (EAST, 2006). No external mechanical stabilization or splinting of the flail segment is indicated. Review procedure manual.

(1) **Why is CPAP helpful?**

(a) Prolongs O2 diffusion time by 50%
(b) Improves gas exchange
(c) ↓ work of breathing
(d) ↓ respiratory muscle fatigue
(e) ↑ functional reserve capacity
(f) ↑ cardiac output

(2) **Contraindications for CPAP:**

(a) Decreased LOC
(b) Unable to maintain patent airway
(c) Aspiration risk; inability to clear secretions
(d) Need for immediate intubation
(e) Need for ventilatory assistance
(f) ECG instability
(g) **Evidence of pneumothorax**
(h) Gastric distention
(i) ↑ ICP
(j) Facial trauma/burns or recent surgery to face/mouth that would complicate mask seal
(k) Epistaxis
(l) Pt unable to tolerate mask or pressure

(3) **Criteria to discontinue CPAP in the field**

(a) Inability to tolerate mask
(b) Need for tracheal intubation or assisted ventilations
(c) ↓ BP (SBP <90; DBP <60) (MAP < 65)

d. **Need early ventilatory support**

(1) Presence of shock
(2) Associated severe head injury
(3) Associated injury requiring surgery
(4) Previous pulmonary disease
(5) Age > 65 years
(6) Fracture of eight or more ribs

e. Monitor ECG

f. IVF as necessary with NS to maintain signs of adequate tissue perfusion. (East, 2006). Do not overhydrate.

g. **Pain management is essential** to promote ventilation and to prevent untoward effects of pain i.e., splinting, atelectasis, and hypoventilation. Some patients may desaturate purely from inadequate pain management. Use of narcotic agents may decrease the respiratory drive, worsen hypoxia, and cause ↓ BP and should be used with extreme caution in the presence of chest trauma. Balance risk with benefit in the field. Alternative pain interventions are used at the hospital like epidural analgesia and local nerve blocks.

h. No external splinting indicated

i. If patient suffers a cardiac arrest: an impedance threshold device (ResQPod) is contraindicated

7. **Complications**

a. ** Flail chest serves as a **red** flag for significant underlying intrathoracic injury, usually **pulmonary contusion**. Also suspect intrathoracic injuries such as hemothorax and pneumothorax. Suspect if SpO2 remains < 90 despite 15 L O2.

b. Pneumonia may occur secondary to a combination of factors: hypoventilation, intubation, aspiration, inadequate pain management, atelectasis, and pooling of secretions.

c. Prolonged tracheal intubation can lead to associated complications such as tracheal stenosis, vocal cord paralysis, and vocal cord ulceration.

d. Long-term symptoms include complaints of dyspnea and chest pain.

II. **C: Circulation = Injuries that jeopardize circulation/perfusion**

A. **Assess for hemodynamic stability**

1. **Mental status**: Restlessness, progressive agitation, mental confusion and irrational or uncooperative behavior

2. **Pulses:** Presence/absence, rate (generally fast or slow), quality (full/thready), rhythmicity (regular/irregular), location (carotid, femoral, or radial), symmetry, deficit, or loss on inhalation (pulsus paradoxus). Tachycardia is not specific to one cause, but may give an indication of shock.

3. **Skin perfusion**: Color/temperature/moisture. Look for pale, cool, diaphoretic skin due to sympathetic nervous system response.

4. Evaluate neck veins

5. Monitor ECG rhythm

1. **Heart tones**

a. Heart sounds are best noted over the following areas:

(1) Mitral valve: 5th left intercostal space (LICS) in the midclavicular line
(2) Tricuspid valve: lower left sternal border at the 4th intercostal space
(3) Aortic valve: 2nd right intercostal space at the sternal border
(4) Pulmonic valve: 2nd left intercostal space at the sternal border

a. Heart sounds are often **difficult** to assess while examining the multiply traumatized patient. However, muffled or distant heart tones are noteworthy because this finding may indicate cardiac tamponade or a tension pneumothorax with significant mediastinal shift.

6. Shift of apical impulse

B. Because many of the organs in the chest are so vascular, hemorrhagic shock is a common complication. The lungs are a low pressure system. Continuous hemorrhage indicates rupture of major vessel.

1. Chest trauma patients in shock had a mortality of 7%. If respiratory distress was also present, along with shock, the mortality increased to 73%.

2. Most frequent sources of bleeding causing shock in thoracic trauma

a. **Blunt chest trauma**

(1) Pelvic or extremity fractures (59%)
(2) Intraabdominal injuries (41%)
(3) Intrathoracic bleeding (26%)

In addition, 15% had myocardial contusion and 7% had SCI which can contribute to hypotension from decreased myocardial contractility and diminished sympathetic tone.

b. **Penetrating chest trauma**

(1) Intrathoracic bleeding (74%) often from multiple sites:

(a) Lung (36%)
(b) Cardiac, usually with tamponade (25%)
(c) Great vessel (14%)
(d) Intercostal/internal mammary arteries (10%)

(2) Other contributing sources: 40%

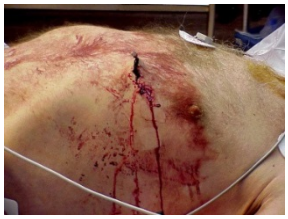
C. **Pericardial tamponade**

1. **Definition**: Tamponade comes from the French word, "tampon", meaning "to plug". Pericardial tamponade literally means a plug in the pericardial sac. It practically means an accumulation of blood and/or clot in the pericardium causing an increase in intrapericardial pressure.

2. **Etiology**: Can occur with blunt or penetrating trauma, however, penetrating is more common from small projectiles (ice pick or stiletto knife). Because blunt trauma causes such a large tear, the patient generally exsanguinates before the lesion can seal over.

3. **Epidemiology**

a. **Incidence**



(1) Occurs in less than 2% of chest trauma

(2) Tamponade occurs in 10% of patients with blunt chest trauma (Yamamoto et al, 2005).

(3) Approximately 80-90% of patients with stab wounds to the heart show evidence of tamponade.

(4) Occurs in 93% of all right ventricular wounds; 43% of left ventricular wounds (Chappell, p.14)

(5) Can occur with acute MI and myocardial rupture

(6) Rare – CPR

b. **Morbidity/mortality**

(1) Estimated mortality 15-60%
(2) Gunshot wounds carry higher mortality than stab wounds
(3) Lower mortality if isolated tamponade is present
(4) Clinical severity depends on pericardial compliance, rate of fluid accumulation and amount of fluid in pericardium

4. **Anatomical considerations**

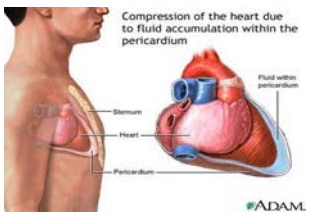
a. The pericardium is a tough, fibrous sac, enclosing the heart and attaching to the great vessels at the base as they leave the heart

b. Visceral and parietal layers serve to anchor the heart, restricting excess motion during acceleration or repositioning of the body. They also prevent kinking of the great vessels.

c. The space between the layers is a “potential space”

d. Parietal, fibrous portion is non-distensible. Space is normally filled with 30-50 mL of straw-colored fluid secreted by the visceral pericardium. Fluid provides lubrication, lymphatic drainage and immunologic protection for the heart.

5. **Pathogenesis of pericardial tamponade**



a. Bleeding from the myocardium or coronary arteries accumulates in the pericardial sac. The pericardium has a non-linear pressure compliance curve. In chronic disease, there may be a slow, progressive accumulation of fluid that may distend the sac from 1000-1500 mL yet maintain adequate output.

b. In traumatic injury, rapidly accumulating amounts of > 50 mL can over a period of minutes to hours increases intrapericardial pressure and overcomes the compliance curve as

there is no time for the tissues to stretch and accommodate for the excess fluid. The most compressible structures within the sac collapse - atria, great veins.

c. The combination of pressure and vascular collapse decreases venous return to the right side of the heart (preload), restricts diastolic expansion and filling and reduces stroke volume.

d. Initially, mechanisms such as an increased heart rate, increased myocardial contractility, and an increase in ventricular filling pressure are used to compensate for the decrease in stroke volume (CO = SV X HR).

e. Once the limits of compensation are reached, further increases in pericardial volume cause a severe impairment of cardiac functioning.

f. Myocardial perfusion decreases due to pressure effects on the walls of the heart and decreased diastolic pressures.

g. Ischemic dysfunction may result in infarction

h. Because of the dynamics of the pressure compliance curve, removal of as little as 20 to 50 ml of blood may drastically improve cardiac output

i. **Same mechanism of death as a tension pneumothorax** = Inadequate venous return and decreased CO.

6. **Assessment**

a. Clinical presentation depends on the interaction between pericardial compliance, rate of fluid accumulation, and the amount of fluid present in the pericardium.

b. **Beck's triad:** advanced stages seen in only 30% of patients

(1) **JVD** (first sign): Kussmaul's sign (neck veins fill during inspiration, empty during expiration)

(2) Decreased arterial pressure (Systolic BP less than 100 mm Hg); pulsus paradoxus and narrowed pulse pressure

(3) Muffled (subjective) heart sounds

c. **Evidence of shock/hypoxemia**

(1) Thready/absent peripheral pulses (PEA): peripheral pulse 0 (absent) or 1+ (thready of a 4+ scale). Cardiac arrest may occur secondarily to unrecognized pericardial tamponade. Patients who survive arrest may sustain other complications such as sepsis, anoxic encephalopathy, or ischemic bowel if the arrest is prolonged
(2) Diaphoresis
(3) Dyspnea and other signs of respiratory distress; cyanosis of upper half of body
(4) Altered mental status, agitation
(5) Tachycardia, tachypnea
(6) Pulsus paradoxus: Systolic BP decreases > 10 mmHg with inspiration

d. Continuous ECG monitoring – assess and record tracings

e. May be difficult to differentiate between a tension pneumothorax and pericardial tamponade in the field as they share many of the same signs and symptoms.

7. **Emergency interventions**

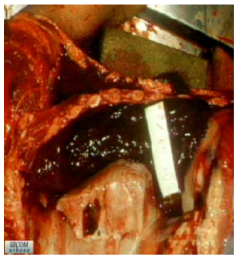
a. Gain airway control; Oxygen based on level of hypoxia
b. IV **NS** to achieve a SBP of 80.
c. Monitor for PEA: Treat per Traumatic Arrest SOP while enroute.
d. Pericardiocentesis is a controversial temporizing intervention due to the risk of possible cardiac damage. It may elicit marked improvement in the patient who is hemodynamically compromised if enough blood is removed from the pericardial sac to allow right heart filling again. This procedure is NOT done in the NWC EMSS.

e. Prepare resuscitative equipment

D. **Massive hemothorax**

1. **Definition**: Accumulation of 1500 mL or more blood in the pleural space (Class III or IV hemorrhage). Pleural spaces can hold the entire blood volume.

2. **Etiology**: Blunt or penetrating injury with trauma to systemic or pulmonary vessels, lung, or heart



   a. **Bleeding sources on left side**, in decreasing order of frequency

      (1) Rib fracture
      (2) Pulmonary parenchyma
      (3) Aortic isthmus
      (4) Spleen
      (5) Heart
      (6) Intercostal artery: Can easily bleed 50 ml/min
      (7) Supra-aortic vessel
      (8) Major pulmonary vessel
      (9) Diaphragm

   b. **Bleeding sources on right side** in decreasing order of frequency

      (1) Rib fracture
      (2) Pulmonary parenchyma
      (3) Liver
      (4) Intercostal/internal mammary artery
      (5) Supra-aortic vessel
      (6) Pulmonary vessel
      (7) Aortic isthmus
      (8) Heart
      (9) Diaphragm

3. **Morbidity/mortality**

   a. Life-threatening injury that frequently needs urgent chest tube insertion and/or surgery at the hospital

   b. Hemothorax associated with great vessel or cardiac injury

      (1) 50% will die immediately
      (2) 25% live five to ten minutes
      (3) 25% may live 30 minutes or longer

4. **Pathophysiology**

   a. Blood accumulates in pleural space causing partial or total lung collapse with possible mediastinal shift and impaired venous return

   b. Patient presents in hypovolemic shock and in respiratory distress. Cause of death = exsanguination leading to cardiac arrest.

   c. Ventilatory insufficiency depends on the amount of blood in pleural space

5. **Assessment**

   a. **Inspection**

      (1) Signs of hypovolemic shock (pale, cool, moist skin)
      (2) Dyspnea, tachypnea, and other signs of respiratory distress; desaturation on pulse oximeter
      (3) Asymmetric chest expansion, complaint of chest tightness, pleuritic chest pain
      (4) Ecchymoses over affected lung
      (5) Hemoptysis or bloody sputum
      (6) Neck veins should be flat

   b. **Palpation**

      (1) Trachea should not be deviated
      (2) Diminished pulse quality, tachycardia

   c. **Percuss** for dullness on affected side

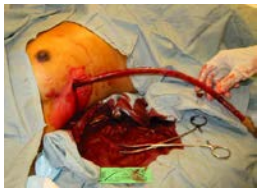
d. **Auscultate** for decreased or absent lung sounds on affected side; BP for hypotension, narrowed pulse pressure

6. **Emergency interventions**

a. Gain airway control

b. Oxygen based on degree of hypoxia; assist ventilations as necessary

c. Insert large bore peripheral IV line



d. Administer NS to correct hypotension to minimum acceptable levels (SBP 80 with penetrating trauma and 90 with blunt trauma). If hypotension lasts <30 minutes, mortality may only be 11%. If hypotension is prolonged: mortality rises to 40%-50%. If patient has underlying disease & is 65 years or older, the mortality with hypotension for > 30 minutes may be over 90%.

e. Hospitals will insert chest tubes, perhaps autotransfuse the patient and determine if rapid operative intervention is necessary.

A. **Thoracic aorta dissection/transection**

1. **Etiology**

a. Most commonly injured from **blunt trauma**; usually high speed MVCs with lateral impacts and sometimes falls from a height.

b. Produces 15% of all blunt trauma deaths.

c. Penetrating injuries may occur at any point and usually result in massive hemorrhage. Patients do not respond to CPR or volume replacement until continuing loss is controlled.

2. **Morbidity/mortality**

a. 80-85% die at scene
b. Of those who survive, 10-15% survive to arrival at hospital
c. 33% die within six hours
d. 33% die within 24 hours
e. 33% survive three days or longer

3. **Pathogenesis**

a. Impact produces increased intraluminal pressures

b. Points of attachment

(1)	Isthmus at ligamentum arteriosum	85%
(2)	Aortic annulus	9%
(3)	Diaphragm	3%
(4)	Other	3%

c. When the body is in motion and comes to a sudden halt, shearing forces or stress on fixation points cause tears of the intimal layer at points of attachment or thinning which allows it to separate from the media.

d. Media and adventitia are more elastic and may not tear initially

e. Blood dissects between the two layers causing a bulge on the vessel (false aneurysm).

4. **Assessment**

a. In those who are not immediately exsanguinating, the physical exam may be unremarkable. There are no specific symptoms… patients present with a vast spectrum of clinical findings. Maintain a high index of suspicion.

b. **Inspection**

(1) Complaint of retrosternal chest or interscapular pain (80%)
(2) Ischemic pain of the extremities
(3) Signs of hypovolemic shock; pallor
(4) Severe dyspnea, stridor
(5) Decreasing level of consciousness, restlessness, apprehensiveness
(6) Dysphagia (from hematoma-induced esophageal compression)
(7) Hoarseness (from hematoma-induced laryngeal compression)

c. **Palpation**

(1) Pulse differential between arms or greater pulse amplitude in arms than in legs (may reflect ruptured descending aorta), decreased or absent femoral pulses

(2) Tracheal shift

(3) Tachycardia.

d. **Auscultation**

(1) Hypotension - 25% due to leakage and hypovolemia

(2) Hypertension - 25% due to stretching of sympathetic nerves in the aorta near the ligamentum arteriosum

(3) Pressure differential between right and left arms (may reflect ruptured subclavian artery on side with lower pressure)

(4) Harsh systolic murmur over precordium or interscapular region due to turbulence as the blood exits the heart and passes the disrupted blood vessel wall.

e. **Be suspicious if:**

(1) + physical exam; decreased BP to lower limbs;
(2) 1st-2nd rib fracture;
(3) sternal fracture; or
(4) significant deceleration > 35 mph.

5. **Clinical S&S**

a. Respiratory distress
b. Signs of pericardial tamponade
c. Physical evidence of major chest trauma e.g., steering wheel imprint on chest
d. Pulse differential between arms or between upper and lower extremities: decreased or absent femoral pulses
e. Upper extremity hypertension or BP differential between arms
f. Enlarging hematoma at the thoracic outlet
g. Interscapular murmur
h. Palpable fractures of sternum and/or thoracic spines
i. Trauma which may be suspicious for occult injuries
j. Scapula fracture
k. Multiple left rib fractures
l. Flail chest
m. Clavicle fracture in the multi-system injured patient
n. First rib fracture

6. **Emergency interventions**

a. Airway control

b. Monitor for signs of decreased tissue perfusion/hypovolemic shock

c. IVF to achieve BP just up to 80-90 mm Hg. Do not hydrate too quickly - raising the BP may complete the separation or rupture the remaining vascular walls. Permissive hypotension is preferred.

d. Monitor the patient's response to fluid administration; VS

e. Monitor for dysrhythmias and alterations in consciousness

f. ***Time sensitive patient***. They need immediate surgical intervention. Transport ASAP to a hospital with cardiothoracic surgery capabilities.

7. **Complications**

a. **Paraplegia**: vascular supply to cord disrupted in thoracic segments
b. Bowel ischemia
c. Renal failure
d. Anoxia
e. Brain injury
f. Left ventricular failure

B. **Penetrating wounds of the great vessels**

1. **Usually involve**

a. Chest
b. Abdomen
c. Neck

2. Wounds are accompanied by

a. Massive hemothorax
b. Hypovolemic shock
c. Cardiac tamponade
d. Enlarging hematomas

3. Hematomas may cause compression of any structure

a. Vena cavae
b. Trachea
c. Esophagus
d. Great vessels
e. Heart

4. Emergency interventions

a. Manage hypovolemia
b. Relief of tamponade if present
c. Time-sensitive patient; expeditious transport

C. **Blunt cardiac injury (old myocardial contusion)**

1. Most controversial of all injuries

2. **Incidence:** 13-75% of trauma patients experience blunt cardiac trauma. Disparity explained due to lack of a "gold standard" diagnostic test at the hospital. It occurs more frequently in younger patients.

3. **Morbidity**: Rarely fatal alone, but may cause significant morbidity. Mortality: 8-20%; deaths secondary to dysrhythmias or ventricular failure. May be the most common unsuspected visceral injury found after fatal crashes.

4. **Mechanism of injury**

a. 90% occur due to MVCs. Speeds of 20-35 mph can cause contusion w/o external chest trauma

b. Extent of injury is related to the magnitude of the force, duration over which force is applied, and the rate of change over time.

c. **Forces**: Compression (RV absorbs impact against sternum ("clapper against a bell"), acceleration/deceleration, intra-abdominal cavity compressed and kinetic energy directed upward (hydraulic ram effect).

5. **Associated injuries** causing increased index of suspicion

a. Sternal fractures
b. Anterior flails
c. Aortic transection
d. Pelvic fractures
e. Cardiogenic shock if contusion > 40% of ventricular surface

6. **Pathophysiology**

a. Areas of damage are well demarcated
b. Hemorrhage with edema and fragmented myocardial fibers
c. Cellular injury
d. Vascular damage may occur
e. Pericardial tamponade: hemopericardium from lacerated epicardium or endocardium

f. **Decreased ventricular function**

(1) **RV most frequently injured** due to proximity to chest wall

(2) LV dysfunction secondary to increase in RV afterload with subsequent shift of the intraventricular septum to the left - causing decreased LV compliance and stroke volume.

g. **Dysrhythmia secondary to**

(1) activation of ectopic pacemakers;
(2) re-entry pathways;
(3) hypoxia/ischemia; or
(4) alcohol ingestion.

7. **Sequelae (complications) of blunt cardiac trauma – much like AMI**

a. Dysrhythmias usually absent in children. The location of the injury influences the type of dysrhythmia that occurs.
b. VSD: ventricular septal defect
c. Valvular disruption
d. Coronary artery occlusion
e. Ventricular aneurysm and myocardial rupture
f. Fibrinous reaction at contusion site may lead to delayed pericardial rupture with cardiac herniation

8. **Clinical presentation**

a. Chief complaint is often retrosternal chest pain or shortness of breath: typically sharp and well localized but may mimic ischemic pain. Often difficult to distinguish from chest wall pain.

b. **Inspect** for ecchymosis on anterior chest, complaint of retrosternal angina, and signs of hemodynamic instability and cardiogenic shock (but be aware that patient may be asymptomatic).

c. Palpate for point tenderness, crepitus over sternum or ribs

d. **Auscultate** for crackles, heart tones and pericardial friction rub; S3 gallop

e. **ECGs**: Abnormal in 40%-80% of contusions, but abnormal in 50% of patients without contusions with chest trauma.

(1) Cardiac injury causes alterations in cardiac depolarization and repolarization and cardiac ischemia. ECG changes are often noted within 24 hours and return to normal in a much shorter time than those produced by myocardial infarction. Most resolve spontaneously, without treatment. One of the limitations of the conventional ECG is that the recordings are dominated by the larger mass of the left ventricle, while the more anteriorly placed RV is the more commonly injured chamber. The ECG is therefore not a sensitive or specific indicator of MC. Use only to increase index of suspicion. There is no typical ECG pattern for cardiac contusion.

(2) Dysrhythmias: 90% present at impact. Should revert to normal in 3-4 months.

(a) Persistent tachycardia
(b) Death in field caused by VT or VF
(c) Atrial flutter/fib
(d) PACs, PVCs - frequently resolve by arrival at hospital

(3) Conduction abnormalities: trend toward overdiagnosis

(a) RBBB
(b) AV blocks
(c) Short PR syndrome
(d) Left or right atrial enlargement
(e) Non-specific ST-T wave changes - 80%. Must prove it was pre-existent or assume it was caused by injury
(f) Prolonged QT interval

9. **Emergency interventions**

a. Gain airway control

b. Oxygen therapy based on clinical presentation

c. Monitor HR, and peripheral pulses, and BP. Notify medical control of

(1) changes in mental status,
(2) systolic BP below 100 mmHg,
(3) absent or thready peripheral pulses.

d. Continually assess patient for signs of

(1) pulsus paradoxus,
(2) neck vein distention,
(3) muffled heart sounds, and/or
(4) hypotension.

e. Administer IVF to maintain BP at 90

f. Pressor agents such as dopamine in high doses for hypotension: Be familiar with dose and administration calculations prior to the time the patient requires pressor support.

g. Antidysrhythmic agents as needed

Physical assessment findings in cardiac tamponade			
Assessment	**Normal Findings**	**Findings in Tamponade**	**Scientific Explanation**
Neck veins	Flat when pt. is elevated to a 30°-45° angle.	Distended when pt. is elevated to 30°-45° angle.	The vena cavae, atria and ventricles are compressed by blood in the pericardial sac. Venous return is impaired due to elevated pericardial pressure.
Heart sounds	Distinct S1, S2	Muffled S1, S2	Blood accumulation in the pericardial sac decreases the conduction of the heart sounds.
Arterial blood pressure	Systolic pressures > 100 mmHg	Systolic pressures < 100 mmHg	The combination of ↓ CO and poor myocardial contractility ↓ arterial pressure and tissue perfusion. Peripheral vasoconstriction will ↑ Diastolic BP to narrow pulse pressure.
Peripheral pulses	Full, easily palpable	thready or absent	Peripheral vasoconstriction w/ ↓ CO make pulses difficult to palpate.
Nailbeds	Pink w/ normal capillary refill	dusky or pale w/ delayed/absent cap refill	Peripheral vasoconstriction shunts blood away from capillary beds.
Pulsus paradoxus	PP < 10 mmHg. No ↓ in systolic BP during inspiration	PP > 10-12 mmHg	During inspiration, blood pools in the pulmonary circulation. Therefore, during inspiration, left ventricular preload is reduced, ↓ CO and peak systolic pressures.
Respiratory Rate	Adult: 12-20/min.	Tachypnea w/ respiratory alkalosis	Compensatory mechanism for ↓ CO.
Integument: Skin color	No mottling or abnormal discoloration	Pale or cyanotic	Peripheral vasoconstriction.
Skin moisture	Dry	Diaphoretic	Sympathetic response
Skin temperature	Warm	Cool	Peripheral vasoconstriction
ECG findings	All waveforms are of normal morphology and timing.	Electrical alternans, ↑ ST segment, T wave changes	Heart is swinging within the pericardium and myocardial ischemia is present.
Mental status	A & O X 3	Decreased LOC	Decreased cerebral perfusion

---

Source: 2018-2019 NCH Paramedic Program. Abdominal Trauma. Connie J. Mattera, M.S., R.N., EMT-P

I. **Abdominal exam**: A thorough abdominal exam needs to be done to determine evidence of injury and/or peritoneal irritation. Large amounts of intra-abdominal bleeding may occur without much external evidence. A positive exam is the most reliable method of diagnosing intra-abdominal trauma **but a negative exam does not rule out injury**.

A. The physical exam may be unreliable in patients who have sustained significant abdominal trauma and have impairment from a head injury, drugs, alcohol, etc., and/or other pain sites (distracting injuries) i.e., femur fracture.

B. Overview

1. Inspection (scars, masses, ecchymosis, distention)
2. Auscultation (bowel sounds, bruits)
3. Percussion (organ enlargement, dullness)
4. Palpation (tenderness, guarding, rebound, referred pain, masses)

C. **Specific abdominal assessment;** serial exams necessary

1. **Inspection**: anterior and posterior torso from lower chest to perineum

a. **Observe for gross abnormalities: DCAP BLS**

b. Lesions: focal injury sites - contusions, ecchymoses, abrasions, penetrating wounds (entry and/or exit wounds), lacerations.

c. Presence of impaled objects: Note characteristics of object, more than one type? Spine injury MOI?

d. **Penetrating wound landmarks**

(1) Anterior Superior - 4th ICS
(2) Anterior Inferior - Inguinal crease
(3) Posterior Superior - 6th ICS
(4) Posterior Inferior - Gluteal folds

e. **Thoracoabdominal stab wounds**

(1) Below the nipples but above the costal margins

(2) Suspect injury to diaphragm and underlying abdominal organs

(3) Consider phase of respiratory cycle at time of wound

(a) Inspiration = pulmonary injury

(b) Expiration = abdominal injury

(c) Consider sex of assailant

(i) Females stab downwards

(ii) Males thrust upwards

f. **GSW:** The internal path of a penetrating object may not be apparent from the external wound. Must also consider collateral damage from deflected missiles, shock waves and/or cavitation. Example, **Tangential GSW to abdomen**: GSW strikes the abdomen but does not enter the abdominal cavity, includes wounds of the abdominal wall, flanks, pelvis, and back.

g. Scars (previous abdominal surgery, trauma)

h. Protrusion of abdominal contents (evisceration)

i. **Contour**

(1) Flat

(2) Rounded

(3) Scaphoid

(4) Distended

(5) Pregnant

j. **Symmetry**; irregular shape or distribution may indicate an accumulation of fluid, blood or gas

k. Visible pulsations

l. **Skin color/discolorations**

(1) **Cullen's Sign:** Bluish discoloration around the umbilicus suggestive of retroperitoneal hemorrhage.

(2) **Grey-Turner's Sign:** Discoloration of the flanks believed to be due to the infiltration of extraperitoneal tissues with blood.

(3) **Seatbelt sign;** fit across abdomen; bruising

m. Pelvic area and perineum; bleeding (rectal, vaginal, wounds), drainage, edema, ecchymosis.

2. **Auscultation**

a. Stimulation of bowel sounds may be initiated by palpation, therefore, auscultation is performed prior to palpation.

b. Normal bowel sounds occur every 5-15 seconds. It is recommended that the evaluator listen for one minute in one quadrant to determine that bowel sounds are diminished. Warm stethoscope head and place gently on abdomen.

c. Decreased or absent bowel sounds generally indicate peritoneal irritation or paralytic ileus from enteric contents, bile, pancreatic juice, or intraperitoneal blood. Bowel sounds in chest: diaphragmatic rupture. Extra-abdominal injuries (pelvic, spinal, and rib fractures) may also cause ileus.

d. **Vascular sounds**: A bruit can be noted if an artery is partially occluded.

3. **Percussion**: Generates slight motion of the peritoneum which may amplify subtle rebound tenderness after injury. Done to detect air, fluid or tissue. May find a markedly distended stomach or the subtle lack of dullness over a liver under free intraperitoneal air.

a. Dull sounds: organ margins, accumulation of free fluid after rupture

b. Tympanic sounds: air, gas

c. Shifting dullness: *Balance's Sign* - indicates splenic injury

4. **Palpation**: assess location and magnitude of pain, involuntary muscle guarding, tone of the abdominal wall, flank and back muscles.

a. Start with **light** palpation, indenting the abdominal wall ½ inch, using the palmar surface of the fingers or side of the hand, beginning at a site distant to the area described as tender/painful.

b. Move from one quadrant to the next assessing for the following:

(1) Point tenderness; local vs. diffuse

(2) Guarding: voluntary vs. involuntary

(3) Rigidity

(4) Pulsatile masses

c. Abdominal tenderness or involuntary guarding elicited by light palpation should alert suggests potential intraabdominal insult. However, voluntary guarding may occur secondary to noxious stimuli (tickling, coldness, fear of pain) and may not occur secondary to intraabdominal trauma.

d. Evolution of peritonitis S&S based on nature of irritant

(1) Blood will not cause immediate peritonitis

(2) Chemical peritonitis may be abrupt from stomach acids

(3) Bacterial peritonitis may take hours to develop

e. **Deep palpation (3")**: usually performed by a physician

f. **Rebound tenderness**: Elicited by having the examiner palpate deeply into an area remote from the area of tenderness and then rapidly removing the hand. The presence of pain following the removal of pressure is a sign of peritoneal irritation. This approach should be done by physicians. Better ways for EMS to test for rebound:

(1) Cough tenderness

(2) Percussion tenderness: **tap the patient on the bottom of the foot** and assess for an increase in abdominal pain

(3) Heel drop test (Markel sign): Have patient stand on toes and rapidly drop to heels (adaptation of percussion tenderness)

D. **Signs** of abdominal injury

1. Bruises, lacerations; entry or exit wounds; stab wounds
2. Vomiting
3. Hypovolemic shock
4. Distention
5. Local or diffuse tenderness
6. Rapid, shallow respirations

E. **Symptoms** of abdominal injury

1. Pain: abdominal or referred (Kehr's sign)
2. Nausea, anorexia
3. Patient affect
    a. Anxiety from pain, hypoxia or hypoperfusion
    b. Desire to remain still and not move; quiet, non-complaining patients may have severe injuries
    c. Lots of patient movement indicates less chance for peritonitis
    d. Hypovolemia changes LOC

F. **Signs of peritoneal irritation** (peritonitis) and a possibly acute abdomen

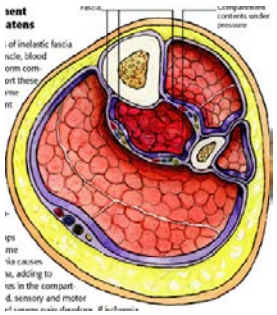
1. Board-like abdomen described as increased **rigidity of the abdominal wall** or palpable abdominal rigidity
2. **Involuntary guarding**
3. Severe focal pain
4. **Rebound tenderness**
5. Silent or absent bowel sounds
6. Abdominal pain associated with peritoneal signs in children: Rebound tenderness (including tenderness to percussion) or guarding suggests peritoneal inflammation. Children with peritonitis will often avoid motion and keep their hips flexes to relieve tension on the abdominal musculature. The abdomen may be distended with decreased or absent bowel sounds.

Source: 2018-2019 NCH Paramedic Program. Musculoskeletal and Soft Tissue Trauma. Connie J. Mattera, M.S., R.N., EMT-P

I. **Complications of musculoskeletal injuries**

A. **Neurovascular injuries:** The skeletal system normally protects the neurovascular structures in the limbs from injury. Critical structures lie deep in the limb, close to the skeleton. Ex: brachial plexus lies in the axilla and on the inner aspect of the arm; shielded from injury by the shoulder girdle. At time of injury, displaced fracture fragments or bone ends at a dislocated joint may lacerate or impale nerves and vessels or compress and entrap these structures in the fracture site. Neurovascular structures are more securely tethered to the soft tissues around joints and are less likely to escape injury if there is significant joint displacement.

1. **Compartment syndrome:** Bleeding or swelling occurs in a closed compartment surrounded by inelastic membranes. Pressure is transmitted to the vessels and nerves. Circulation and sensation are compromised due to pressure. A common misconception is that open fractures are safe from compartment syndrome, and this is not true.



    a. **Precipitating factors for compartment syndrome**
        (1) External: Constrictive bandages; casts; PASG
        (2) Internal
            (a) Increased compartmental content
            (b) Bleeding within the compartment from direct vessel injury or an abnormal bleeding tendency
            (c) Edema from ischemic swelling, exercise, trauma, burns (thermal vessel damage, vascular thrombosis, tissue destruction leads to leakage of plasma colloids and proteins into the interstitial spaces)
            (d) Bleeding in upper extremities is most likely to occur following a crush injury or dislocation, displaced fracture of the elbow, or of the proximal or **middle forearm.**
            (e) **Edema**

b. **Pathophysiology**

(1) **Decrease in compartment size or increase in compartment contents**

(2) **Increased intracompartmental pressure**

(3) **Vasospasm; arterial occlusion**

(4) **Increased capillary permeability**

(5) **Decreased tissue perfusion**

(6) **Intramuscular edema**

(7) **Increased intramuscular pressure**

(8) **Progressive death of muscle and nerve tissue**

c. **Signs & symptoms of compartment syndrome**

(1) Throbbing pain localized pain to compartment involved. Pain associated with neurovascular compromise is usually described as burning or searing and is poorly localized. Pain will appear out of proportion to the injury with no relief with pain medication. Pain with passive movement occurs as a result of gently stretching the ischemic muscle. These areas can be tested by (1) thumb and finger extension and flexion or abduction and adduction, (2) toe extension and flexion, and (3) foot inversion and dorsiflexion.

(2) **Pallor**

(3) **Paralysis; weakness of muscle**

(4) **Pulse -** Distal pulses and capillary refill will still be present except in the event of direct vessel injury, as the radial artery runs outside of the forearm compartment. It is theorized that the ischemia causing the tissue damage is due to closure of the arterioles and venous collapse. The larger arteries remain open until compartmental pressures are extremely high; therefore pulses will usually be palpable on physical examination**.**

(5) **Paresthesias;** burning sensation distal to injury or decreased 2-point discrimination over involved compartment, not localized.

(6) **Increased pressure or tension in muscle compartment**

d. **Treatment:** Need to maintain perfusion as well as possible. DO NOT apply cold, ice or elevate the extremity if compartment syndrome is possible. Fasciotomy at hospital.

B. **Crush syndrome:** Occurs after prolonged compression force impairs muscle metabolism and circulation. The syndrome is precipitated after extrication or release of an entrapped limb. The degree of injury will depend on the extent of tissue compressed and the duration and force of compression. Consider this syndrome not only in trauma victims, but in victims that have been lying on an extremity for extended periods of time (4-6 hours of compression). Example: Drug overdose or stroke victim who has not been found for an extended period.

1. **Injury t**o muscle is caused by vascular compromise: irreversible muscle damage after 4-6 hours; direct force; and/or direct prolonged pressure.

2. **Pathophysiology**

a. As blood flow to tissue decreases, toxins from cellular metabolism begin to build up.

b. Swelling occurs in response to the injury, and increases pressure in the compartment. Fluids and waste are now unable to move freely in or out of the area.

c. When the individual is finally extricated or the limb is released, blood moves into the area that was crushed, and toxins are released into the systemic circulation. The toxins are lactic acid, potassium, myoglobin, and uric acid in quantities not normally found in bloodstream.

3. **Results of toxin release**

a. **Plasma** leaks out of capillaries due to crush injury and swelling. This causes third space fluid loss that can lead to hypovolemia and shock.

b. Decreased blood pH due to increased **lactic acid,** resulting from anaerobic metabolism. When the pH is decreased, the heart is vulnerable to fibrillation, which is resistant to treatment due to the acidosis.

c. **Potassium** leaks out of the damaged cells into the extracellular fluid. Increased potassium makes the heart prone to asystole. Hyperkalemia causes muscle weakness or paralysis and is an electrical depressant.

d. **Myoglobin** is released from damaged muscle tissue. As it accumulates in the systemic circulation, the kidneys are unable to filter it all as waste. Glomerular filtration is blocked by the large protein molecules and renal function is impaired.

4. **Treatment:** Focused on combating the effect of the toxins systemically when released. Initiate treatment as soon as possible to dilute the toxins.

a. **ABCs essential:** 15 L O_2 may be needed to combat dust-related airway problems. In acidosis the RR will increase, so supplemental O_2 is essential.

b. Obtain baseline ECG before release if possible; continue ECG monitoring after release. Observe for the following:

(1) Peaked T waves

(2) Widened QRS

(3) Prolonged PR interval

(4) VT; VF; or asystole

c. Start IV NS TKO prior to compression release. Run wide open after release of compressed body parts to fill the vascular space, dilute urine and assist kidneys in excretion of lactic acid and myoglobin. Give 200 mL IVF challenges in elderly – monitor for fluid overload.

d. Assess for hyperkalemia w/ cardiotoxicity: Peaked narrow T waves w/ shortened QT to flattened or absent P waves, prolonged PRI, wide QRS, sine-wave pattern (QRS merges w/ T wave), asystole. If present:

(1) SODIUM BICARBONATE 50 mEq slow IVP over 5 min followed by 20 mL NS IV flush

(2) No IV: ALBUTEROL 5 mg continuous neb up 20 mg (throughout transport) [BLS]

(3) OLMC may order use of both

e. If HR > 100, restless, ↑RR, wide QRS, long PR interval, or peaked T waves after above: IV NS up to total of 3 L over 1st 90 minutes following release of compression unless contraindicated. (Ensure clear lung sounds, no shortness of breath)

f. DO NOT apply PASG or other compressive device. Assess for compartment syndrome; if present, do not elevate or cool limb.

C. **Suspension injuries:** The major risk with these patients is orthostatic intolerance (shock) while suspended. At risk for Reflow Syndrome after rescue: Occurs when toxins that accumulated in pooled blood suddenly return to body after pt lies flat following suspension release. Observe for significant HYPERKALEMIA as noted above under Crush Syndrome.

Keys for EMS Treatment

1. A patient who is experiencing pre-syncopal symptoms or who is unconscious while suspended in a harness should be rescued as soon as safely possible.

2. If you cannot immediately release a conscious patient from a suspended position, instruct them to elevate their legs and contract their leg muscles periodically.

3. Watch for signs and symptoms of pre-syncope: light-headedness, nausea, sensations of flushing, tingling or numbness of the arms or legs, anxiety, visual disturbance or a feeling they're about to faint.

4. After rescue, **do NOT allow the patient to lie flat** (unless CPR is required). If conscious: Position sitting up with legs bent at the hips and knees for at least 30 min. If unconscious, place on side w/ knees drawn up to chest.
5. **Do NOT allow the patient to stand up**. Risk of syncope and rapid weakness should be anticipated.
6. For a semi-conscious or unconscious person who has already been placed in a horizontal position, follow standard guidelines. Do not raise an unconscious or pre-syncopal patient back to a sitting or standing position.
7. Maintain a patent airway and follow standard procedures for ABCs.
8. Start IV NS TKO prior to suspension release if possible. Run wide open after release up to 1 L.
9. Hypoglycemia should be corrected per SOP.
10. Monitor the ECG for electrical abnormalities, such as hyperkalemia (peaked T waves, prolonged QT intervals, widened QRS complexes).
11. Monitor the blood pressure and the onset of crush syndrome.
12. Consider additional drugs (IV bicarbonate or albuterol) if hyperkalemia suspected (per crush syndrome SOP).