Scenario: EMS is dispatched to a 2-car MVC with head on collision. The posted speed limit is marked at 40 MPH. Upon EMS arrival to the scene an unrestrained adult driver is found inside the vehicle with noted + steering wheel deformity. The patient is A & O X 3 but appears restless and agitated.

On assessment the following is noted:

Airway:	Patent
Breathing:	RR: fast; labored with asymmetric chest expansion and use of accessory muscles but no paradoxical movement; left side appears hyperinflated and does not move. Breath sounds absent on left, diminished on right; no adventitious sounds. No open wounds; trachea midline. SpO ₂ 86%, EtCO ₂ 27.
Circulation:	JVD present; radial pulses absent; carotid pulses fast, weak and thready; equal bilaterally. C / O severe chest pain & difficulty breathing
Disability/LOC:	Eyes open spontaneously, pt is awake, alert & oriented to voice & is able to move all extremities to command; PERL, EMS notes an abrasion to L anterior chest; the pt is A & O but restless & agitated.
Pain:	9/10
VS:	BP 84/60, P 116, R 24

	Questions	Answers
1.	What two immediate life-threats should be suspected based on this presentation?	
2.	Which one is most likely based on the mechanism of injury? What is the pathophysiology and the classic clinical findings of this injury?	
3.	What is the mechanism of death in this injury?	
4.	What temporizing life-saving procedure must be performed immediately?	
5.	What equipment will you need?	
6.	What landmarks must you find?	
7.	At what angle is insertion performed?	
	If you hit bone, should you go over or under? Why?	
8.	What should happen after penetration into the pleural space?	
9.	Will this procedure re-expand the collapsed lung? Why or why not?	
10.	What is the difference between a simple pneumothorax and a tension pneumothorax?	

NWC EMSS CE November 2015

Case #2

Scenario: EMS is dispatched for an adult who fell. Upon arrival to the scene, a neighbor greets you and states that they saw the person cleaning gutters earlier. EMS sees an adult pt (50 M) lying on cement driveway supine outside home. Upon scene size up a ladder is found on the ground outside a 2 story (~20 ft.) family home; bushes in front appear damaged. Upon arrival to the pt, EMS finds a person as stated with bleeding from left forehead; 10" diam of blood on ground and appears in distress moaning and localizes pain.

On primary assessment you note the following:

Airway:	Gurgling sounds noted in airway w/ bloody secretions
Breathing:	Breathing faster than normal, shallow and labored effort (diminished BS on L side).
Circulation:	Pulse is fast and regular; but radials are weak. Capillary refill is 3 seconds. Neck veins are flat and skin is dusky, cool and moist to the touch.
Disability/LOC:	Eyes are open to pain, incomprehensible sounds made & localizes to painful stimuli. Pupils PERL; blood glucose level is 86.
Pain:	8/10
Secondary assessment:	
VS:	BP 94/64, P 116, R 24
Head:	airway clear w/ suctioning; no bruising to face.
Pupils:	PERL
Neck:	JVD, trachea midline

Chest: abrasion & tenderness L lat area; + distress; + crepitus to palp w/paradoxical movement

Abdomen: abrasion noted to the LUQ/L flank area; pt moans to palpation

Pelvis: unremarkable

Ext: L LE w/deformity; otherwise + movement x 4

	Questions	Answers
1.	What 3 chest injuries should be suspected based on this presentation and mechanism of injury?	
2.	What is the most likely chest injury based on the mechanism of injury and pt presentation?	
3.	What is the definition for that injury?	
4.	What are the other injury concerns based on pt presentation?	
5.	What criteria is needed for the pt to be placed on CPAP?	
6.	Once treatment includes CPAP, what area of re-assessment if key?	
7.	Where should this pt be transported based on presentation and injury?	

Case #3

Scenario: EMS is called to a house for an adult with chest pain from a penetrating injury. PD is on scene stating that the scene is safe and that there was an attempted home burglary in which the burglar stabbed the homeowner in an attempt to escape scene. Upon entering the house, the pt is sitting on the sofa holding his chest in distress. The t-shirt is noted to have a minor amount of bright red blood in a circular fashion to the slight L of lateral position mid chest. He states that his chest feels like there is a burning sensation and is in respiratory distress. No penetrating objects remain.

On primary assessment you note the following:

Airway: Breathing:	Patent Dyspneic; RR rapid, shallow and labored with no movement of L lateral chest wall; breath sounds absent bilaterally. RA SpO2 89%; EtCO2 30
Circulation:	Radial pulses becoming non-palp w/ inspiration, carotids fast, weak and thready. Skin is dusky, cool & clammy. No uncontrolled hemorrhage but + bubbling to chest wound, + JVD
LOC:	eyes open spontaneously; voice is oriented and moves extremities to commands. Pupils are PERL.

Secondary assessment

VS: HEENT:	BP: 96/72; P: 136; RR: 32 shallow and labored. Pt states "I can't catch my breath." airway remains open; no DCAP-BTLS-TIC PMS to head or neck
Neck:	Trachea is midline; jugular veins flat
Chest:	1 ½" opening over left medial chest wall; pain on palpation with blood bubbly w/resps. Heart sounds muffled.
Abdomen:	Soft and non-tender.
Skin:	Dusky nail beds; circumoral cyanosis. Cool, pale, diaphoretic.
Neuro:	GCS 15; PERL; SMV intact X 4
Pain:	10/10

	Questions	Answers
1.	What 2 chest injuries should be suspected based on this presentation and mechanism of injury?	
2.	What are the classic clinical findings of these injuries?	
3.	Identify the life threat?	
4.	How should this patient be treated? What life saving treatment should be done for these injuries?	
5.	What equipment is needed to perform this treatment?	
6.	What is the ongoing danger to the pt from this injury? What is the mechanism of death?	
7.	What should be done if after treatment with BP rising, then the pt again becomes hypotensive?	
8.	Where should this pt be transported based on presentation and injury?	

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Case #4

Scenario: You are called to a restrained driver in a MVC on expressway. There is 15-20 inches of metal intrusion in at the dashboard due to a frontal impact. The windshield is broken and the steering wheel is bent. The patient is complaining of severe substernal chest pain. He is holding his arm against his chest to splint when he breathes.

On primary assessment you note the following:

Airway:	Patent
Breathing:	Dyspneic; RR rapid, shallow and labored with redness and abrasions to the chest wall; no
	paradoxical movement, SpO2 90%; Breath sounds present but diminished bilaterally.
Circulation:	Radial pulses equal; rapid, weak and thready. Skin pale, cool, clammy.
LOC:	Awake; responds to verbal stimuli

Secondary assessment:

VS:	BP: 92/50; P: 116; RR: 26 and shallow
HEENT:	All WNL
Neck:	Trachea is midline; jugular veins flat
Chest:	Contusion over sternum on chest wall; pain noted on palpation.
	ECG: ST with multi-focal PVCs
Abdomen:	Soft and non-tender.
Skin:	Cool, pale, diaphoretic.
Neuro:	GCS 14; PERL; SMV intact X 4
Pain:	9/10

	Questions	Answers
1.	What chest injuries should be suspected based on this mechanism of injury?	
2.	Which one would be most likely? Why?	
3.	How should this pt be monitored?	
4.	How should you treat this patient?	
5.	What 2 treatment modalities are indicated if the pt becomes/remains hypotensive?	
6.	Where should this pt be transported based on presentation and injury?	

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Case #5

Scenario: A 50 y/o restrained driver of a single vehicle crash who drove off the road and laterally hit into a tree at 40 mph on the drivers side. Upon arrival, assessment reveals the car to be a vintage model with only the lap belt available. Therefore the patient is slumped sideways in to the center of the vehicle, moaning.

On primary assessment you note the following:

Airway:	patent
Breathing:	labored; rapid rate. Breath sounds normal and equal bilaterally.
Circulation:	Radial pulses rapid and weak; skin pale and cool to touch.
LOC:	Eyes closed; responds to verbal stimuli by moaning; not moving extremities to command. Pupils
	PERL, sluggish to respond.

Secondary assessment:

VS:	BP: 88/54; P: 110; RR: 24.
HEENT:	Multiple abrasions to the lateral aspects of the pt's head with lac to L forehead and bleeding. Pupils as noted above; bleeding coming from mouth with loose teeth.
Neck:	Trachea midline; jugular veins flat.
Chest:	No injury noted to chest with equal chest expansion; no paradoxical movements. EKG: ST with PVCs.
Abdomen:	abdominal exam with point tenderness to palpation to R and LLQ with positive guarding and tenderness to palpation (moans and localizes pain).
Extremities:	multiple abrasions but no entrapment or extrication needed.

Questions		Answers
1.	What abdominal injuries should be suspected based on this presentation and mechanism of injury?	
2.	Which abdominal injury is the patient at greatest risk?	
3.	Why? Does this injury to the abdomen create an immediate life-threat?	
4.	How should you treat this patient?	
5.	What is the ongoing danger to the patient from this injury?	
6.	Identify 3 internal organs in the LQs that could be injured from a low lying lap belt?	
7.	Identify 3 internal organs in the UQs that could be injured from a high lying lap belt?	
8.	Where should this pt be transported based on presentation and injury?	

NWC EMSS CE November 2015

Thoracic Trauma as written for the

NWC EMSS Paramedic Education Program

Connie J. Mattera, M.S., R.N., EMT-P

I. Epidemiology of chest trauma

A. Incidence

- 1. Chest injuries are common occurrences following blunt and penetrating trauma.
- 2. Isolated chest trauma is uncommon; the majority of these patients will have additional injuries.
- 3. Prevention efforts a major key
- 4. Thoracic trauma accounts for ¹/₃ all trauma admits
- 5. Thoracic injury may involve:
 - a. Chest wall
 - b. Thoracic great vessels
 - c. Heart, lungs, pleura
 - d. Diaphragm, esophagus
 - e. Trachea and bronchus
- 6. Blunt chest trauma
 - a. Occurs in both rural and urban settings
 - b. Motor vehicle crashes are responsible for **70-80%** of blunt thoracic trauma plus falls, sports and crush injuries

7. Penetrating chest trauma

- a. Usually associated with an urban setting
- b. Commonly due to gun shot wounds and knife wounds
 - (1) **Low velocity gunshot wounds**: Hand guns 12-25% mortality. Sterile, wound only along track of missile.
 - (2) **High velocity gunshot wounds**: Military and hunting rifles. Cavitation may create tissue damage 15 times the diameter of the bullet. All these wounds will require operative debridement at the hospital.
 - (3) **Shot gun wounds**: Result in varying wound types depending on pellet size, choke, and distance from the victim.
 - (4) **Stab wounds**: 75% of penetrating chest wounds resulting from knives, ice picks, sticks, arrows, portions of automobiles or other projectiles.
 - (5) Impalement injuries
- 8. Isolated chest trauma is uncommon (16%); 84% of these patients will have additional injuries

B. Morbidity and mortality

- 1. Thoracic injuries are the second leading cause of trauma mortality occurring in 15-25% of all trauma-related deaths (about 12,000 per year in the U.S.). Most deaths are secondary to heart and great vessel trauma causing exsanguinating hemorrhage and respiratory failure.
- 2. Thoracic injuries are second only to head trauma in mortality rates. Overall mortality rate is 3% to 18%.
- 3. Chest injuries are often associated with abdominal injuries and are a significant contributor to fatal outcomes in an additional 25%-50% of cases. They are the leading cause of preventable trauma death.
- 4. Mechanisms of injury causing death from thoracic trauma
 - a. MVC
 - (1) Account for largest number of trauma deaths
 - (2) Over 50% have one or more drivers legally intoxicated
 - (3) Motorcycle death rate more than 15 times greater then auto crash
 - b. Falls: More than ½ in elderly
 - c. Penetrating injuries; seen more frequently in urban areas due to violent crime
 - d. Crush injuries

- 1. **Acceleration-deceleration**: Skeletal body starts or stops moving more quickly than the internal organs. This type of motion frequently causes more damage to relatively fixed structures, (aorta), than to non-fixed organs, such as the heart and lungs.
- 2. **Compression**: Occurs when the external force applied is greater than the resistance of the skeletal body.
- D. Basic approach to major thoracic trauma remains unchanged, but the treatment of several injuries have undergone an evolution in the recent past:
 - 1. Pericardial tamponade
 - 2. Aortic transection
 - 3. Blunt cardiac injury
 - 4. Pulmonary contusion and flail chest
- E. **Prevention** efforts have the potential to reduce the incidence of thoracic injuries:
 - 1. Firearm safety
 - 2. Sports training
 - 3. Seatbelt use, passive restraint systems
 - 4. Decreased speed limits
 - 5. Community/legal activity regarding drunk driving, etc.
 - 6. Violence prevention education i.e., conflict resolution skills

Review thoracic anatomy from Respiratory A&P and Cardiac A&P lectures

III. General pathophysiology of chest injuries

II.

Α.

- Impairments in ventilatory efficiency
 - 1. Pain restricting chest excursion
 - 2. Air or blood entering the pleural space
 - 3. Chest wall fails to move in unison
 - 4. Ineffective diaphragmatic contraction
- B. Impairments in gas exchange
 - 1. **Hypoxia**: Results from inadequate O2 delivery to tissues
 - 2. Pulmonary ventilation/perfusion mismatch: i.e.- contusion, hematoma, alveolar collapse
 - 3. **Changes in intrathoracic pressure relationships**: tension/open pneumothorax or severe hemothorax
 - 4. Atelectasis
 - 5. Contused lung tissue
 - 6. **Respiratory acidosis, hypercarbia**: most often results from inadequate ventilation caused by changes in intra-thoracic pressure relationships and depressed level of consciousness
- C. Disruption of respiratory tract

D. Impairments in cardiac output

- 1. **Hypovolemia**: Inadequate intravascular volume due to blood loss
- 2. Increased intrapleural pressures reduce venous return
- 3. Blood in pericardial sac reduces preload
- 4. Decreased stroke volume due to blunt cardiac injury
- 5. Myocardial valve damage
- 6. Vascular disruption
- 7. **Metabolic acidosis**: caused by hypoperfusion to tissues

IV. Primary assessment pearls

- A. Clinically evident, immediately life-threatening injuries should be considered, found and resuscitated as soon as a deficit is discovered. AVOID HYPOXIC INJURY.
- B. If agitation or altered mental status is present, assume that the patient has an airway, breathing, and/or perfusion problem especially if objective criteria support these findings, e.g., decreased oxygen saturation, change in capnogram, or pulse deficits. Do **not** initially attribute abnormal finding to drugs or ethanol abuse until life-threatening problems have been ruled out.
- C. If cervical spine status is unclear, spine motion restriction must be maintained if a mechanism of injury suggests potential c-spine injury.

D. Etiology of inadequate ventilations/impaired gas exchange

1. Ventilation deficiencies: Pulmonary, musculoskeletal, or neurologic

2. Diffusion deficiencies: Pulmonary contusion, previous disease

E. Inspection

- 1. Visually inspect the thorax for appearance, contour, symmetry of excursion, and any gross abnormalities, i.e., deformity, contusions, abrasions, penetrating wounds, bruising, lacerations, subcutaneous or tissue edema, paradoxical movements, retractions, or impaled objects, etc.
- 2. Determine adequacy of ventilations
 - a. General respiratory rate, depth; and effort (tachypnea, bradypnea)
 - b. Work of breathing; use of accessory muscles; nasal flaring
 - c. Capnography number and waveform

3. Determine oxygenation status

- a. Clinical presentation i.e. mental status, skin color (cyanosis of the lips or nail beds) etc.
- b. Pulse oximetry (SpO₂)

4. Neck veins

- a. Normal anatomic location of neck veins
 - (1) External jugulars are above the clavicle and cross over the sternocleidomastoid muscles.
 - (2) Internal jugulars run parallel to the sternocleidomastoid muscles near the carotid arteries.
 - (3) If a patient is positioned at a 45° angle, the venous pulses should **not** ascend more than one to two cm above the clavicle.
- b. Markedly distended neck veins occur when blood cannot drain into the right atria. In the presence of chest trauma, JVD may indicate tension pneumothorax or cardiac tamponade.
- Observe for the type, amount, and nature of secretions

5. C F. Palpation

- 1. Point tenderness
- 2. Loss of chest wall integrity; instability
- 3. Crepitus; subcutaneous emphysema
- 4. Edema
- 5. Tracheal position: Deviation is difficult to appreciate clinically

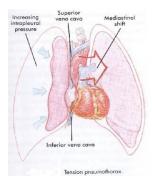
G. Percussion

- 1. Hyperresonance / tympany (pneumothorax)
- 2. Dull or flat tone (hemothorax)
- H. Listen without a stethoscope for noisy ventilatory efforts, air being sucked in or out of an open chest wound.
- I. Auscultate immediately if in ventilatory distress; Listen posteriorly, laterally, and anteriorly
 - 1. Breath sounds present or absent; unilateral or bilateral deficits
 - 2. If decreased, attempt to discern etiology; treat appropriately
 - 3. Adventitious sounds: treat during focused exam phase
 - 4. Presence of bowel sounds in the chest may signify a ruptured diaphragm

V. 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
 - 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. Hemodynamic dysfunction produces hypoxia and obstructive shock.
- h. 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 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 to right)
 - (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
 - Equipment needed: 10 g IV catheter or use s 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 needle insertion in the 4th-5th intercostal space in the midaxillary line is NOT approved in the NWC EMSS due to the possibility of diaphragm and liver/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?
- B. **Open pneumothorax** (sucking chest wound)

(1)

is

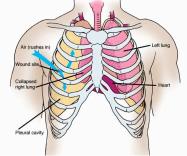
lost,

- 1. **Etiology**: Usually caused by penetrating chest trauma secondary to gun shot or stab wound, but can also be caused by an impaled object.
- 2. 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.

Since chest wall and pleural integrity

involved

the



paradoxically collapses on inspiration and expands minimally during expiration moving air in and out of the defect, producing a "sucking" sound.

lung

- (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
- Morbidity/mortality: Patient dies from inadequate ventilation and impaired gas exchange
- 4. Assessment

3.

- a. Inspect
 - (1) Visible chest wall defect; sucking sound on inhalation
 - (2) Complaint of pain with breathing

NWC EMSS CE November 2015

- (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).

5. Emergency interventions

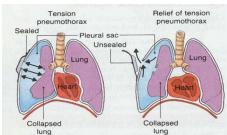
- a. The *SMITH PAPYRUS*, written sometime around 3,000 B.C., contains the earliest recognized reference to "thoracic trauma". Work describes 58 patients (2 had thoracic injuries). Recommended treatment for penetrating injury was fresh meat poultice the first day, followed by grease, honey and lint on subsequent days
- 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 (Vaseline gauze, plastic wrap, defib pad or commercial device). Dressings should be at least 3 or 4 times the size of the defect.
 - (3) May tape on 3 sides to create flutter valve
 - (4) 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

C. 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

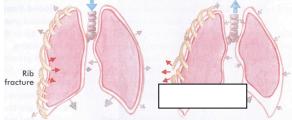




- Flail chest occurs when two or more adjacent gibs another cartilages on both sides a. of an impact point are broken at two points resulting in a freely mobile or "floating" segment.
- b. May be identified by location or size
 - (1) Anterior, posterior, or lateral
 - (2) Separation of the sternum from 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.

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.



- Subatmospheric intrathoracic pressure during inspiration pulls the segment e. 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 pO₂ and increase in AaDO₂ (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 PaO₂, 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
- Mortality increased with c.
 - (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.

Page 13

- (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

а.

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.
 - 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) $PaO_2 < 60 \text{ mmHg at } FiO_2 > 50\%$
 - (c) $PaCO_2 > 50 \text{ mmHg at } FiO_2 > 50\%$
- 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.
 - (1) Why is CPAP helpful?
 - (a) Prolongs O2 diffusion time by 50%
 - (b) Improves gas exchange
 - (c) \downarrow work of breathing
 - (d) \downarrow respiratory muscle fatigue
 - (e) \uparrow functional reserve capacity
 - (f) \uparrow 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
 - (I) Pt unable to tolerate mask or pressure
 - Criteria to discontinue CPAP in the field
 - (a) Inability to tolerate mask
 - (b) Need for tracheal intubation or assisted ventilations
 - (c) \downarrow BP (SBP <90; DBP <60) (MAP < 65)
- d. Need early ventilatory support

(3)

- (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.

VI. 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%.

NWC EMSS CE November 2015

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.

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

b.

- 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.

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

- (4) Can occur with acute MI and myocardial rupture
- (5) Rare CPR

b. Morbidity/mortality

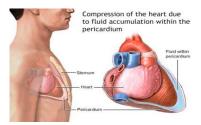
- (1) Estimated mortality 15-60%
- (2) Gunshot wounds carry higher mortality than stab wounds
- (3) Lower mortality if isolated tamponade is present
- (4) Clinical severity depends on pericardial compliance, rate of fluid accumulation and amount of fluid in pericardium

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 nonlinear 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; 12-15 L oxygen
- b. IV **NS** to achieve a minimum 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.
 - Prepare resuscitative equipment
- D. Massive hemothorax

e.

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- 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
- 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



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- b. 12-15 L oxygen; 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 less than 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.

E. Myocardial rupture

- 1. Associated with immediate trauma or delayed for 2-3 weeks
- 2. Associated with blunt trauma as the heart is compressed between the sternum and vertebrae
- 3. Penetrating trauma: Rib, missile, sternal bone
- 4. History of trauma with a presentation of
 - a. Heart failure
 - b. Cardiac tamponade
- 5. Immediate onset of heart failure following trauma
 - a. Rupture of cardiac valves
 - b. Intraventricular septal rupture
- 6. Management is supportive
- F. Commotio cordis: Blow to the chest that produces cardiac arrest. This may be more common than once thought. Any blow to the chest, regardless of its intensity or velocity or force is capable of producing cardiac arrest, especially in younger children whose rib cages are narrow and have underdeveloped chest muscles. Patients have experienced death due to a blow from softballs, baseballs, hollow toy baseball bats, snowballs, chest blows during shadow boxing, playing with a family dog or trying to remedy hiccups (Cooke, 2002).

G. Traumatic asphyxia

- 1. Pathophysiology
 - a. Sudden compressional force squeezes the chest
 - b. Vascular pressure increases in the head, neck, and kidneys
 - c. Jugular veins engorge, capillaries rupture
- 2. Clinical presentation

a.

b.

- Inspection
 - (1) Blunt chest trauma, may have flail
 - (2) Profound shock
 - (3) Cyanosis of face, neck, and shoulder
 - (4) Swelling or hemorrhage of the conjunctiva; may exhibit exophthalmos (protruding eyes)
 - (5) Swollen, cyanotic tongue and lips
 - (6) JVD
 - (7) Skin below area remains pink
 - (8) Bloody vomiting (hematemesis)
- Auscultation: Hypotension when pressure is released

3. Emergency interventions

- a. Gain airway control
- b. 12-15 L oxygen via BVM or transport ventilator.
- c. Spine motion restriction on a long spine board
- d. Time sensitive patient; begin expeditious transport
- e. Venous access enroute; fluid administration per ITC

VII. Secondary assessment

A. Once the initial assessment is completed and life-threatening injuries have received resuscitative intervention, proceed with focused history and exam. With chest trauma, **any** negative change



from the initial assessment is a sign of possible trouble.

- Β. Full set of vital signs
 - Pulse: Rate, quality, rhythmicity, deficits 1.
 - 2. **Blood pressure**
 - Narrowed pulse pressure a.
 - b. Hypertension
 - Hypotension c.
 - d. Pulsus paradoxus
 - 3. Respirations: Rate, pattern, depth
- C. **SAMPLE** – history
 - 1. **S**ymptoms
 - 2. Allergies
 - 3. **M**edications
 - 4. Past medical history
 - 5. Last oral intake/LMP
 - 6. Events surrounding the incident – mechanism of injury
- D. Continue to use maneuvers of inspection, palpation, percussion and auscultation to detect additional injuries.
 - Chest wall injuries are characterized by pain, ineffective ventilation, and secretion 1. retention
 - 2. Injuries to the lung include pulmonary contusions and hematomas. Both cause bleeding into the alveoli which impairs gas exchange.
 - 3. Pleural space injuries: pneumothoraces, hemothorax, and tracheobronchial tears

Ε. Injuries to suspect/find now

- 1. Aortic transection
- 2. Penetrating wounds of the great vessels
- 3. **Bronchial disruption**
- 4. Esophageal injury; traumatic diaphragmatic hernia
- 5. Blunt cardiac injury (myocardial contusion)
- 6. **Pulmonary contusion**
- 7. Simple pneumothorax
- 8. Hemopneumothorax
- 9. Fractured ribs
- 10. Sternal fracture

F. Thoracic aorta dissection/transection

- 1. Etiology
 - Most commonly injured from **blunt trauma**; usually high speed MVCs with lateral a. impacts and sometimes falls from a height.
 - b. Produces 15% of all blunt trauma deaths.
 - Penetrating injuries may occur at any point and usually result in massive c. hemorrhage. Patients do not respond to CPR or volume replacement until continuing loss is controlled.

Morbidity/mortality 2.

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

3. Pathogenesis а.

- Impact produces increased intraluminal pressures
- b. Points of attachment
 - (1)Isthmus at ligamentum arteriosum 85%
 - (2) Aortic annulus 9%
 - (3) Diaphragm 3% 3%
 - (4) Other
- When the body is in motion and comes to a sudden halt, shearing forces or stress C. 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
 - I. 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.

3.

5.

- 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

G. 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
 - 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

H. 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).
- 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 for a 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 preexistent or assume it was caused by injury

NWC EMSS CE November 2015

(f) Prolonged QT interval

NWC EMSS CE November 2015

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.
 - Antidysrhythmic agents as needed

I. Pulmonary contusion: see flail chest

g.

- 1. Incidence: Present in 30 to 75 percent of patients with significant blunt chest trauma
 - 2. Mechanism of injury
 - a. Commonly associated with rib fracture
 - b. High velocity missile with blast effect
 - c. High energy shock waves from explosion
 - d. Rapid deceleration
 - e. Low velocity: projectiles like an ice pick
 - 3. Mortality and morbidity
 - a. Missed due to high incidence of other associated injuries
 - b. Mortality between 14-20%
 - 4. Pathophysiology
 - a. Three physical mechanisms
 - (1) Implosion effect
 - (a) Overexpansion of air in lungs secondary to positive-pressure concussive wave
 - (b) Rapid excessive stretching and tearing of alveoli
 - (2) Inertial effect

Strips alveoli from heavier bronchial structures when accelerated at varying rates by concussive wave

- (3) Spalding effect
 - (a) Liquid-gas interface is disrupted by shock-wave
 - (b) Wave releases energy
 - (C) Differential transmission of energy causes disruption of tissue
- b. Alveolar and capillary damage
- c. WBC and platelet aggregation in pulmonary vessels leads to release of vasoactive substances
- d. Loss of pulmonary capillary integrity with increased membrane permeability; movement of water and plasma proteins into alveolar and interstitial spaces = interstitial edema and congestive atelectasis
- e. Surfactant dilution results in decreased lung compliance
- f. The amount of oxygen delivered across the pulmonary capillary bed in the injured segment is decreased resulting in hypoxemia and carbon dioxide retention.
- g. Hypoxia causes reflex thickening of mucous secretions
 - (1) Bronchiolar obstruction
 - (2) Atelectasis
- If the contusion is large, the body compensates by vasoconstricting pulmonary blood flow and increasing cardiac output to shunt blood around the area of minimal oxygenation. There is deceased functional reserve capacity due to this

pulmonary shunting which causes mixed venous blood to be returned to the heart resulting in further hypoxemia.

5. Assessment

- a. The adverse effects of pulmonary contusion usually do not become clinically evident until 24 hours after injury.
- b. Inspection
 - (1) Signs of respiratory distress, dyspnea, tachypnea, restlessness, apprehension
 - (2) Ineffective cough
 - (3) Hemoptysis
 - (4) Possible chest wall abrasions or contusions
 - (5) Increased pulmonary secretions, decreased pulmonary compliance, desaturation on pulse oximeter, and cyanosis
- c. Palpate for possible tenderness over chest wall, tachycardia
- d. Auscultate for crackles and wheezes, areas of decreased breath sounds

6. **Emergency interventions**

- a. Gain airway control
- b. 15 L oxygen; assist ventilations as necessary
- c. Restrict IV fluids unless hypovolemic
- d. Analgesics as indicated
- J. Tracheobronchial injuries
 - 1. Epidemiology
 - a. Incidence: Rare: less than 3% of chest trauma
 - b. Tracheal injuries are usually caused by penetrating trauma
 - c. Bronchial injuries are usually caused by deceleration (blunt) mechanisms
 - d. **Morbidity/mortality**: High mortality rate: greater than 30% due to associated airway obstruction. Majority of the patients die at the scene secondary to asphyxia.

2. Pathophysiology

- a. Site of injury
 - (1) Tear can occur anywhere along tracheal/bronchial tree
 - (2) Transverse rupture is the most common type of tracheal injury
 - (3) > 80% occur within 2.5 cm of the carina (point of fixation)
 - b. Rapid movement of air into pleural space
 - c. Tension pneumothorax refractory to needle decompression
 - d. Continuous flow of air from needle decompression site
 - e. Severe hypoxia
 - f. **Associated injuries**: Esophageal, great vessels, lung, cardiac, and cervical spine injuries are noted because of anatomical location and mechanism. Mechanism can also account for associated head injuries and facial injuries.

3. Clinical presentation

- a. Variability in presentation: ranging from asymptomatic to severe dyspnea/cyanosis
- b. **Inspection:** Hoarseness, dyspnea, tachypnea, ventilatory distress, hemoptysis
- c. **Palpation:** Massive subcutaneous emphysema
- d. Auscultation
 - (1) Decreased or absent breath sounds associated with pneumothorax
 - (2) Hamman's sign may be present (crunching sound noted with heart auscultation)
- e. S&S tension pneumothorax

4. Emergency intervention

- a. Gain airway control
- b. 15 L oxygen; do not use high pressures without preparing for needle decompression
- c. Vascular access, prepare resuscitation equipment
 - (1) Tracheobronchial rupture/tear via blunt trauma occurs secondary to crushing or compressive injury that causes a sudden decrease in

anterior/posterior diameter and increase in lateral diameter (i.e., lap belt across the throat in MVC). Since the lungs may move laterally in response to the force, the trachea/bronchi may transect secondary to exceeded stretch.

- (2) The cricoid ring and carina serve as fixed points for the trachea. When there is an acute acceleration/deceleration force, a shearing force is produced at these fixed points and creates a tracheal injury.
- (3) Direct insult to a hyperextended cervical trachea may cause rupture, e.g. striking the dashboard, steering wheel.
- (4) Rapid increase in intrathoracic pressures concomitant with a closed glottis may produce a "blowout" injury to the trachea with a linear rupture. (Similar to mechanism precipitating closed pneumothorax.)

K. Sternal fracture

1. Epidemiology

- a. Incidence: 5-8% of all thoracic injuries
- b. **Mechanism of injury** is anterior blunt chest trauma at or below the manubriosternal junction. Classic example is when the chest hits the steering wheel or dashboard in an MVC. Other injury mechanisms include direct blow to the sternum or compression of the sternum associated with hyperflexion sternal injury
- c. Potential life threatening injuries include myocardial contusion, cardiac rupture, cardiac tamponade, or severe pulmonary insult

d. Morbidity/mortality

- (1) 25-45% mortality
- (2) High association with myocardial or lung injury
 - (a) Myocardial contusion
 - (b) Myocardial rupture
 - (C) Pulmonary contusion

2. Pathophysiology

- a. Severe impact as area is well supported by the ribs and clavicles
 - b. Associated injuries cause morbidity and mortality
 - (1) Pulmonary and myocardial contusion
 - (2) Flail chest if rib attachments are disrupted on both sides
 - (3) Vascular disruption of thoracic vessels
 - (4) Intraabdominal injuries
 - (5) Head trauma
 - c. Rarely is fracture displaced posteriorly to directly impinge on heart or vessels, but it does occur

3. Clinical findings

- a. Inspect for anterior chest pain, sternal deformity, contusion, localized pain, tachypnea, ECG changes associated with myocardial contusion
- b. Palpate for tenderness, instability, crepitus over sternum
- c. Unstable fractures may result in a flail chest
- d. ECG changes

4. Emergency interventions

- a. Gain airway control
- b. Oxygen based on clinical presentation
- c. Restrict fluids if pulmonary contusion is present
- d. Provide analgesics per local protocols
- e. Allow chest wall self-splinting

VIII. Other chest injuries

A. Simple pneumothorax

1. Epidemiology

- a. Incidence
 - (1) 10-40% of all blunt chest trauma (check current data)
 - (2) Almost 100% in penetrating chest trauma
- b. Morbidity/mortality: Depends on extent of atelectasis and associated injuries

- c. **Classification** of pneumothorax is determined by the degree of collapse:
 - (1) Small: 15% or less occupation of the pleural cavity
 - (2) **Moderate**: 15%-60% occupation of the pleural cavity
 - (3) Large: 60% or greater occupation of the pleural cavity

2. Pathophysiology

- a. Lung is 1-3 cm away from the chest wall. Pneumothorax results from a one-time leak of a stable amount of air into the pleural space.
- b. Disruption of pleural integrity may result from a laceration caused by a fractured rib, penetrating missile, or barotrauma as seen in the "paper bag syndrome" or may occur spontaneously following strenuous exercise, severe coughing episodes (particularly in patients with COPD), after air travel, or in patients with Marfan's Syndrome.
- c. Loss of intrapulmonary/intrapleural subatmospheric pressure
- d. Elastic recoil of lung tissue allows it to collapse when pleural integrity is disrupted.
- e. Pulmonary function may be good in a healthy person
- f. Small tears may self-seal; larger ones may progress
- g. Respiratory compromise depends on the degree of collapse and the amount of pulmonary reserve. Larger pneumothoraces produce a decreased area for V_A/Q matching resulting in a physiologic shunt in the lungs and hypoxemia

3. Clinical presentation

- a. Inspect for dyspnea, tachypnea, ventilatory distress; sudden, pleuritic chest pain that may refer to shoulder or arm on affected side; may have altered chest wall movement and/or ventilation perfusion mismatch and signs of hypoxia
- b. Percuss: Hyperresonance on affected side
- c. Palpate: Tachycardia; trachea may tug towards affected side; difficult to appreciate with a clinical exam
- d. Auscultate: Decreased or absent breath sounds on affected side. If upright, air will accumulate in the apices. Assess there first for altered breath sounds. If supine, air accumulates in the anterior chest.

4. Emergency interventions

- a. Optimize ventilations and gas exchange with 12-15 L O₂; sit up
- b. Monitor for signs of tension pneumothorax
- c. Peripheral venous access and administer NS or LR

B. Rib fractures

1. Epidemiology

a. Incidence and significance

- (1) Most common thoracic injury; seen in more than 50% of cases with significant blunt chest trauma. Significant chiefly because the pain involved inhibits the patient from taking adequate breaths.
- (2) Pediatric patients have cartilaginous ribs that bend easily. They experience decreased incidence of fractures, but increased incidence of pulmonary injury.
- (3) Geriatric patients have calcified ribs that are less flexible and more easily fractured. Also tend to have increased morbidity due to co-morbid conditions and decreased pulmonary reserves.

b. Etiology

- (1) Caused by direct blows to the chest: Mechanism usually associated with age-related injuries:
 - (a) Sports/recreational injuries in adolescents
 - (b) MVC/assaults in adults
 - (c) Falls in the geriatric population
- (2) Occurs at the point of impact or along the border of the object that impacts the chest. May also occur at a weakened point where the ribs flex (posterior angle).
- (3) Ribs 4 through 9 are most commonly fractured as they are the least protected. The first three ribs are relatively protected by the shoulder

girdle and the lowest ribs (the "floating ribs") are less exposed and more mobile, so are also relatively protected.

(4) Morbidity increases with the number of fractures, extremes of age, and associated chronic respiratory or cardiac conditions - especially in the elderly. Two or more rib fractures are associated with an increased incidence of internal injury

2. Pathophysiology

- a. Because the ribs are part of a ring, when a rib breaks...
- b. Decreased minute ventilation due to splinting from pain
- c. Whenever a person breathes shallowly, for any reason, the alveoli do not get fully inflated; little by little, they begin to collapse and the progressive atelectasis makes the lungs more vulnerable to pneumonia. Additionally, there may be decreased surfactant production that normally lowers surface tension and facilitates alveolar opening. This results in shunting of blood to nonventilated alveoli that can lead to arterial hypoxemia, plugging of proximal airways, segmental and lobar collapse, pneumonia, and bronchiectasis.

3. **Concomitant injuries**

- a. 1st through 3rd ribs take great force to fracture. Assoc. w/ fractures of neck, clavicle, scapula, and great vessel injury (subclavian artery/vein, aortic injury) severe intrathoracic injury (tracheobronchial injury, aortic rupture, and other vascular injuries, especially if multiple ribs are involved.
- b. Fractures of the ninth, tenth, and eleventh ribs are associated with intraabdominal injury: lower left rib fractures: splenic injury; lower right rib fractures: liver injury.
- c. Sternal fractures are associated with pulmonary contusion and/or blunt cardiac injury
- 4. Cartilaginous injuries are similar, but often more painful and take longer to heal

5. Clinical presentation

- a. Inspection
 - (1) Chest wall pain localized to the site of injury aggravated by breathing, coughing, or movement
 - (2) May have overlying chest wall contusion or deformity
 - (3) Subcutaneous emphysema implies...
 - (4) Shallow ventilatory effort and reduced chest wall excursion
 - (a) Hypoxia
 - (b) Hypoventilation atelectasis
 - (C) Muscle spasm at fracture site
 - (5) Splinting: Patient will often lean toward the fracture to relieve muscular tension on the site
 - (6) Inadequate cough
- b. Palpation
 - (1) Point tenderness to palpation; may be unstable
 - (2) Deformity, crepitus (grating sensation) at fracture site
- 6. Complications
 - a. Pneumo/hemothorax due to laceration of bronchi or intercostal arteries
 - b. Pneumomediastinum
 - c. Tension pneumothorax
 - d. Non-union of fracture; Costochondral separation
 - e. Pneumonia (large problem in elderly)
 - f. Neurologic deficits if laceration of intercostal nerves
 - g. **Pitfall**: Do not underestimate the severe pathophysiology of rib fractures, especially in elderly patients.
- 7. Emergency interventions
 - a. Optimize ventilations to prevent atelectasis and pneumonia. Encourage patient to cough and deep breathe. Splint chest with pillow.
 - b. Administer analgesics to facilitate chest wall motion.

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