Non-Cardiac Chest Pain

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Subjects
- Costochondritis
- Mediastinitis
- Pleural Effusion, Pleuritis
- Pneumothorax (simple and tension)
- Sarcoidosis
- Pulmonary Neoplasm
- NonCardiogenic Pulmonary Edema
- Inhalational Injury
- Environmental Lung Disease (Asbestosis, Silicosis, Pneumoconiosis)
- Tracheostomy Complications
- Oropharyngotomotomy (Indications and Complications)

Costochondritis, Musculoskeletal Chest Wall Pain
- Chest wall causes of chest pain are among the most common etiologies that are seen in the primary care setting accounting for 36% of episodes in one report.
- Given the amount to movement and work the chest wall does it is not surprising that MSK chest pain is common.
- Aside from the constant movement with breathing the muscles of the back, neck, abdomen, and shoulder all attach to the chest wall with frequent pulling and mechanical stress from just daily activities.
- Unfortunately a fair number of patients with myocardial ischemia not only have chest wall pain but palpation of the chest wall reproduces the patient’s ischemic pain.

A word of caution, it is not uncommon that pain from more serious entities, i.e. ACS/AMI, are attributed to MSK chest wall pain. It is common for patients with an AMI to have reproducible chest wall pain to palpation.

So, as Doctor and Philosopher Elmer Fudd would say, “When it concerns chest wall pain and AMI - Be verwy, verwy, careful!”

Musculoskeletal/Chest Wall Entities
- Demographics may differentiate acute coronary syndromes from those of the chest wall – age, family history, comorbidities.
- Musculoskeletal chest pain is often insidious, diffuse, associated with movement of the chest wall, and is more frequently seen in women.
- Most often there is some tenderness of the chest wall itself or the pain can be provoked by palpation, movement of the arms, deep breath, bending, or twisting the trunk.

- Costochondritis is the most common etiology of musculoskeletal chest wall pain with tenderness along the costosternal joints.
- Rheumatic disease can be a source of chest wall pain due to ankylosing spondylitis, psoriatic arthritis, rheumatoid arthritis, and fibromyalgia.
- Non-rheumatic diseases such as stress fractures, septic arthritis, sickle cell disease, and osteomyelitis are less common etiologies of chest pain.
- Finally, keep herpes zoster in mind with vague numbness, pain, and the characteristic vesicular rash.
**Musculoskeletal/Chest Wall Entities**

**Classic appearance of chest wall shingles, can often have a prodrome of pain, itching, burning before the vesicular rash appears. Early treatment with acyclovir/famcyclovir can shorten the time to healing with few risks or side-effects.**

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**Pleuritis/Pleurisy**

- Pleurisy is an inflammatory condition of the pleura of the chest with pain caused as the visceral and parietal pleura rubbing against each other during breathing.
- A viral etiology is most common, fever, cough, myalgias, and sore throat may accompany the typical pleuritic chest pain.
- Other etiologies for pleurisy can include autoimmune diseases (SLE, RA), bacterial infections, blunt chest trauma, pneumonia, lung cancer, lymphoma, sarcoidosis, pneumothorax, and smoking.

**Pleuritis/Pleurisy**

- Physical exam is often non-specific however the presence of a pleural friction rub is highly suggestive though rare finding.
- Tests are aimed at other potential etiologies as there is no specific test for pleurisy itself.
- CXR is typically done to look for pleural fluid and/or an infiltrate.
- Labs often include a CBC or inflammatory/rheumatoid markers and EKG for possible cardiac causes of pleuritic chest pain – CT or MRI are not infrequently ordered.
- D-dimer can also be helpful for patients who are PERC or Wells criteria positive.

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**Pleural Effusion**

- Pleural effusions are an accumulation of fluid between the parietal and visceral pleura.
- In many cases this problem is not one we treat in the emergency department.
- They can cause chest pain, typically pleuritic pain, SOB, DOE, up to respiratory failure.
- There are a number of conditions in which performance of an emergent thoracentesis might be necessary.

**Pleural Effusion**

- **Exudate etiologies:**
  - Abscess – lung or abdomen.
  - Aspergillus.
  - Connective tissue diseases – RA, Wegener’s.
  - Esophageal perforation, Boerhaave’s.
  - Inflammatory – SLE, ARDS, sarcoid, uremia.

- **Transudate etiologies:**
  - CHF by far the most common.
  - Hypoalbuminemia, nephrotic syndrome.
  - Peritoneal dialysis.
  - Misplaced central line catheter into the pleural space.

**A right pleural effusion that shows a meniscus with the remaining small amount of lung seen superiorly.**

An effusion this large can cause substantial respiratory distress that can be readily relieved with a thoracentesis that can bridge to more definitive operative drainage.
Pleural Effusion

- Analysis tries to differentiate exudates from transudates and there are several ways to do this – though in most cases this does not change treatment in the ED.

**Light's Criteria** – presence of any one indicates an exudate:
- Pleural fluid protein/serum protein > 0.5
- Pleural fluid LDH/serum LDH > 0.6
- Pleural fluid LDH > 2/3's upper limit of normal for serum LDH or > 200 U/L

*Most commonly see Light's Criteria is on an exam*

A second way to determine an exudate from a transudate using just tests on the fluid:

- **Two-Test Rule** –
  - Pleural fluid cholesterol > 45 mg/dl
  - Pleural fluid LDH > 0.45x upper limit of normal for serum LDH

- **Three-Test Rule** –
  - Pleural fluid protein > 2.9 g/dl
  - Pleural fluid cholesterol > 45 mg/dl
  - Pleural fluid LDH > 0.45x upper limit of normal for serum LDH

Other tests on the fluid may include:
- Amylase – elevated in pancreatitis effusion.
- Adenosine deaminase – elevated in Tb effusions.
- BNP – elevated in CHF.
- Tumor markers.
- Cell count – > 10,000 in infectious exudates.
- Differential – lymphocytes > 85% suggests lymphoma/Tb/sarcoid.
- Culture and sensitivity.

**Draining a Pleural Effusion**

- There are a number of ways to perform a thoracentesis but most commonly the use of one of the commercially available kits.

- **Cardinal Safe-T-Centesis kit is used in the Beaumont ED.**

- Typically the approach is at ~ 8th-9th rib space posteriorly with the patient sitting bolt upright to avoid hitting the lung or causing a pneumothorax.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Fluid Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Empyema</td>
<td>gross pus, C&amp;S, high WBC</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>+ chylous, + fetus membranes</td>
</tr>
<tr>
<td>Uremia</td>
<td>+ C&amp;S, UAC 7.7/7.9 + Al</td>
</tr>
<tr>
<td>Tb empyema</td>
<td>+ AFB stain, C&amp;S</td>
</tr>
<tr>
<td>Esophageal rupture</td>
<td>+ food, low fluid pH</td>
</tr>
<tr>
<td>Portal fluid</td>
<td>+ AFB stain, C&amp;S</td>
</tr>
<tr>
<td>Chylothorax</td>
<td>High triglyceride, chylomicrons</td>
</tr>
<tr>
<td>Noncaseous</td>
<td>Fluid/serum creatinine &gt; 10</td>
</tr>
<tr>
<td>Urothorax</td>
<td>Urothorax fluid/serum creatinine &gt; 10</td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
<td>Glc 300-400, protein &lt; 1 g/dl</td>
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</tbody>
</table>

**Peritoneal dialysis fluid**

- Glc 300-400 mg/dl
- Protein < 1 g/dl

Draining a Pleural Effusion
Pleural Effusion – Safe-T-Centesis Kit

Cardinal Kit containing:
- needle over catheter
- lidocaine + syringe + needles
- scalpel
- suture
- drainage tubing
- 1.5 liter plastic bag to hold effusion
- can be hooked up to evacuated bottle.

Mediastinitis

Mediastinitis is an inflammatory disease of the mediastinum.
- It is highly fatal, difficult to treat and esophageal rupture is one of the more common etiologies.
- It can also be the result of direct infection, extension of a chest infection (pneumonia, empyema, pericarditis), or even extending from the abdomen in rare cases.

Mediastinitis

Patients often present with symptoms of what is causing the mediastinitis such as esophageal rupture.
- Pleuritic chest pain is a hallmark in what is often a toxic looking febrile patient as the mediastinum suffers infection or inflammation rather poorly.
- A CXR may show fluid or even air dissecting along the numerous facial and tissue planes that make up the mediastinum, particularly around the heart.

Mediastinitis

Often a chest CT shows greater detail and is the diagnostic test of choice.
- Treatment is primarily surgical with drainage of the mediastinal space.
- In the ED treatment is directed at maintaining the patient’s respiratory and cardiac status, broad-spectrum antibiotics, and analgesics.

Mediastinitis

CXR showing air adjacent to the aorta and left pleural effusion in a patient with mediastinitis secondary to esophageal rupture and infection.

Esophageal Rupture

Herman Boerhaave, a Dutch physician, described the case of Baron Wassenaer:
- A patient with esophageal rupture. In 1724, Boerhaave was called to the bedside of the admiral, who complained of intense chest pain and that something had burst in his chest. He had just consumed a huge meal, had taken a self-prescribed emetic, and shortly afterwards he vomited, but only a little and this not easily.
- Over the next 16 hours, his condition progressively worsened until he died.
- Autopsy revealed a rent in an otherwise normal-looking esophagus, with food and medicine in the left chest cavity.
- Spontaneous esophageal rupture then became eponymously known as Boerhaave syndrome.

Roman "vomitorium", a required part of a bacchanal dinner.
Esophageal Rupture

Causes of esophageal rupture may include the following:

- Iatrogenic – NG tubes, EGD, misplaced ETT.
- Post-emetic – the most common cause.
- Esophageal disease that weakens the wall – ulcer, Zollinger–Ellison syndrome, cancer, hiatal hernia.
- Caustic ingestion – particularly lye products.
- Foreign body – erodes through the wall.

As the esophagus lacks a serosal layer it is prone to rupture, usually on the left, resulting in rather serious mediastinitis.

The usual history involves either vomiting or instrumentation of the esophagus followed by severe chest pain, radiating to the back, dysphagia, sometimes even melena.

Signs include subcutaneous emphysema, fever, shock, epigastric abdominal pain.

The classic Mackler triad: which includes vomiting, lower chest pain, and cervical subcutaneous emphysema, is present in approximately 50% of cases.

The Anderson hexad includes subcutaneous emphysema, rapid respirations, and abdominal rigidity to that seen in the Mackler triad.

Diagnostic approach to suspected esophageal rupture would include:

- CXR often shows a left sided effusion, subcutaneous air, and pneumothorax.
- CXR, Gastrographin swallow, or CT scanning are often obtained from the ED.
- Emergency EGD is often diagnostic as well.

CT scan showing large extravasation of contrast into the left pleural cavity due to esophageal perforation.

Endoscopy confirmed the diagnosis: 1 cm longitudinal left sided rupture was seen at the lower level of esophagus.

Esophageal Rupture

Treatment includes:

- Vigorous fluid resuscitation, placement of an NGT, chest tube if necessary, intubation for respiratory insufficiency.
- Empiric antibiotics for the mediastinitis to cover gram (+) and (-) organisms is recommended, but there are no good studies to back that up.
- Surgical repair for large tears, associated sepsis due to mediastinitis, and tears associated with malignancy may be necessary.
- Such repair can include diversion, stents, primary repair of several different types, even using fibrin glue.
Pneumothorax

Pneumothorax:
- As a non-cardiac chest emergency is often associated with chest pain and dyspnea is also a common symptom.
- Physical exam may elicit decreased breath sounds and a tympanic hemithorax.
- X-ray, tracheal deviation, and circulatory collapse can be seen with a tension PTX.
- A tension pneumothorax in which there is a ball valve effect where air comes out of the lung, gets trapped in the pleural space, and as pressure increases the heart gets pushed over cutting off venous return—urgent needle decompression followed by a chest tube can be life-saving.

Treatment of pneumothorax:
- Can depend on etiology: small, spontaneous pneumothoraces can be treated with oxygen and observation.
- Larger ones, large air leaks, or traumatic etiologies may need a chest tube with continuous Pleurovac drainage.
- Catheter aspiration of spontaneous pneumothorax (CASP) is also an option for a smaller pneumothorax and there are kits with Heimlich valves now available for this form of treatment.

Catheter-aspiration of a spontaneous PTX (CASP) can be done with a variety of small catheters either specialized for CASP or use a thoracentesis catheter. Seldinger wire technique can be helpful as is going in the usual chest tube site—4th to 5th ICS, mid-axillary line.
Interstitial Lung Disease

This category of pulmonary illness also encompasses the various "lungs" due to chronic inhalation of particulate matter:

- Silicosis from inhalation of silica in mining, industrial manufacturing.
- Asbestosis – in ship building, mining, milling, cement production.
- Contex or wool fibers – textile manufacturing.
- Smoke inhalation – fires, firefighters.
- Coal dust – mining, Black Lung – note the recent uptick in 1,000’s of cases in coal miners from quartz dust that presents much like black lung.

Example of an asbestos-wrapped heating pipe aboard a ship that was essentially just left uncovered exposing the crew and maintenance staff to high levels of inhaled asbestos.

Most of these syndromes result in a COPD picture of chronic cough, impaired lung function, and respiratory failure.

Malignancy rates in these patients is high particularly with asbestosis causing pulmonary mesothelioma.

Clinical presentation of ILD includes:

- Chest pain, dyspnea, DOE.
- Dry, non-productive cough.
- Abnormal CXR.
- Restrictive ventilatory pattern on PFT’s.

Work-up should include:

- Routine labs, serology, ABG’s.
- CXR, chest CT scan.
- PFT’s.

An example of sarcoid perihilar reticular pattern infiltrate characteristic of interstitial lung disease – bat wing pattern.
Interstitial Lung Disease

- Physical exam findings:
  - Crackles are common (sounds like Velcro) and inspiratory squeaks.
  - Cor pulmonale findings such as augmented P2, right sided lift, gallop are seen.
  - Cyanosis is uncommon but clubbing is very common.
- Treatment is symptomatic with oxygen supplementation and referral to a pulmonologist – emergent treatment is primarily in the last stages of disease as the dyspnea, hypoxia, and CO₂ retention become more problematic.

Lung Cancer

- Lung cancer is the most common cause of cancer mortality worldwide.
- Cigarette smoking is by far the most common inciting factor causing both non-small cell and small cell tumors (some 90% due to this single etiology).
- The most common emergency issue is respiratory distress due to a number of complications of the disease or treatment.
- Patients may also present to the ED with pain due to metastases or endocrine issues.

Intrathoracic effects causing respiratory distress are due to:
- Tumor invasion of the lung or pleura.
- Hemoptysis, cough.
- Effects of radiation/chemotherapy on lung function.
- Effusions compromising lung function.
- Metastases tend to go to liver, bone, and brain.
- Paraneoplastic problems like hypercalcemia, hypercoagulability, or Cushing's Syndrome are somewhat uncommon but do occur.

Lung Cancer

- Treatment in the ED is most often directed primarily towards maintaining or improving respiratory function.
- Intubation may be necessary through non-invasive ventilation (CPAP or BiPAP) can also be effective.
- Draining effusions can also improve respiratory status.

ARDS

- Acute Respiratory Distress Syndrome (ARDS) was known as a distinct form of acute lung injury (ALI) by the 1980s as mechanical ventilation became more widespread and patients developed this highly lethal form of respiratory failure.
- Mortality is 25-58% most often due to MODS (multi-organ dysfunction syndrome).
- Its pathophysiology and treatment was initially addressed by the military physicians in Viet Nam where it was called “Shock Lung” or “Da Nang Lung”.

ARDS

- ARDS or “shock lung” was seen in Viet Nam battle casualties particularly those who received massive IV fluids and transfusions (whole blood) leading to this classic CXR with bilateral infiltrates.
- ARDS is ARDS, note the patient is intubated and the huge amount of inflammatory and fluid overload changes.
- ARDS in a heavy smoker, note the spiculated appearance.
ARDS

It does not take a pathologist to see all the debris in these alveoli with almost complete destruction of the nice, lacey appearance of a normal lung – think of this picture as you are attempting to oxygenate your next ARDS patient.

ARDS

ARDS is a spectrum of acute lung injury and there is a somewhat arbitrary division between ARDS and ALI as noted below.

Both ARDS and ALI encompass:
- Acute to hyperacute onset.
- Bilateral infiltrates consistent with pulmonary edema.
- PaO2/FiO2 ratio < 200 regardless of PEEP level for ARDS (ALI has the same parameters save for a slightly better ratio of 200-300).

ARDS

The causes of ARDS are numerous with some 60 different etiologies including:
- Infection/sepsis, aspiration, pneumonia.
- Trauma, fat emboli, lung contusion.
- Multiple transfusions.
- Drug overdose, near drowning, inhalation injury, burns.

Easiest way to manage the ventilator in these patients is to "Google" ARDSnet and it will take you to the ARDSnet site that will walk you through the process. Early initiation of the protocol in the ED is important so YOU NEED TO KNOW THIS!

ARDS Net Ventilator Recommendations

INCLUSION CRITERIA for ARDS:
1. Acute onset \\
2. Bilateral infiltrates consistent with pulmonary edema \\
3. PaO2/FiO2 < 300

VENT SETUP & ADJUSTMENT:
1. Calculate predicted body weight: (males=50+2.3(ht. in inches)-60, females=45.5+2.3(ht. in inches)-60.
2. Select vent mode (AC, pressure, APEX).
3. Initial TV = 8 ml/kg PBW.
4. Reduce TV by 1 ml/kg at 2 hour intervals to achieve TV of 6 ml/kg.
5. Initial rate at baseline minute ventilation, not > 35/minute.
6. Adjust Vt and RR to achieve pH and plateau pressure goals (see next).

OXYGENATION GOALS:
PaO2 = 58-80 mmHg, consider FiO2/PEEP combinations as below:

PLATEAU PRESSURE GOALS:
1. Goal < 30 cmH2O.
2. If Pplat > 30 decrease Vt by 1 ml/kg increments of 2 ml/kg.
3. If Pplat > 35 and Vt < 6 ml/kg increase Vt by 1 ml/kg and then by 4 ml/kg every 2 minutes.
4. If Pplat < 25 and Vt > 6 ml/kg increase Vt by 1 ml/kg every 2 minutes.

ARDS

To a large extent the etiology is not as important in the ED as recognizing the syndrome and addressing it.

CXR is the main diagnostic test.

Blood gases that also allow one to calculate the PaO2/FiO2 ratio.

Elevated BNP is seen consistently in ARDS and can be helpful.

Placement of a Swan-Ganz catheter with measurement of the PA pressure is beyond what can be done in the ED in most cases.

Supine ventilation can also be quite helpful, again not something to risk in the ED.
ARDS
- In the ED the main concerns are airway and breathing for these patients.
- ARDS can start precipitously and patients can deteriorate rapidly—time is of the essence.
- Early intubation is strongly suggested in and as well as most cases of ALI.
- Sometimes you can support an ALI patient with NIV (CPAP or Bi-PAP) but most often these patients require intubation (NIV is a great way to preoxygenate ARDS patients prior to intubation).

This ARDS patient secondary to pneumonia initially did well on supplemental oxygen, progressed to BiPAP, and then was intubated; could not be weaned (advanced COPD), ended up trach'ed and PEG'ed.

Smoke Inhalation
- Smoke is a mixture of gases and aerosolized particles as the result of burning of some fuel.
- It is particularly irritating to the lungs and is often a mixture of highly toxic compounds that are readily absorbed through the lung aside from direct thermal and inflammatory injury to the lung.

Smoke Inhalation
- While smoke can have a high temperature it is often dry and does not conduct much thermal injury beyond the oropharynx and upper airway.
- However, smoke containing water particles carries much more thermal energy as does superheated gases particularly those under pressure.
- This can cause serious direct burns deep into the lung tissue.

Smoke Inhalation
- Fires in confined spaces use up the available O₂ causing hypoxia and suffocation.
- The plastics in buildings burn to toxic gases such as arsenic, cyanide, acrolein, formaldehyde, phosgene, SO₂, NO.
- All of which can also cause bronchospasm, dyspnea, wheezing, and respiratory failure up to 24-36 hours post-exposure if they do not kill the victim outright.

Smoke Inhalation
- CO is the most common lethal toxic gas produced in a fire.
- It tightly binds to hemoglobin preventing the release of oxygen at the tissue level.
- Clinically it can cause headache, nausea, confusion, fatigue, dyspnea, angina, CHF, seizures, coma, and death.
- Diagnosis is by measurement of carboxyhemoglobin either by photometric oximetry or venous and/or arterial measurement with co-oximetry.
- Levels over 20-25 are most often symptomatic with levels over 40 often fatal.
Smoke Inhalation

- Treatment of CO poisoning is primarily oxygen and in serious poisoning by hyperbaric chamber that can reduce the half-life to a mere 20 minutes—however hyperbaric therapy has not show any significant clinical or outcome improvement.
- Residual symptomatology and cognitive impairment is fairly common even with optimal treatment.
- Pregnant patients are at high risk for fetal demise as fetal hemoglobin binds CO even more efficiently than adult hemoglobin—in this case hyperbaric therapy may be helpful.

Smoke Inhalation

- Cyanide is a common by-product of burning plastics and synthetic fabrics.
- It binds to the mitochondrial cytochrome a3 decoupling electron transport and oxidative phosphorylation.
- It takes very little cyanide to produce cardiac dysfunction, apnea, coma, and death.
- Direct measurement of cyanide levels is not helpful and have a high index of suspicion in a smoke inhalation patient particularly one who remains in shock despite treatment and adequate oxygenation and fluids.

Smoke Inhalation

- The older cyanide antidote kit included administration of three medications: amyl nitrite pearls (inhalation) and sodium nitrate and sodium thiosulfate (infusion)—however methemoglobin was a by-product that could contribute to the oxygenation problems already at hand.
- The newer Cyanokit contains hydroxycobalamin that reacts with cyanide to form cyanocobalamin that can be eliminated by the kidneys—this is a simpler and more effective antidote that the older Lilly kit.
- One should have a low threshold for the administration of hydroxycobalamin in the appropriate clinical situation.

Tracheostomy Emergencies

- The idea and early performance of the tracheostomy dates back to Egyptian and Roman times.
- It was resurrected in the late 19th Century as a means to prevent suffocation in diphtheria but the initial patients all died of subsequent tracheal stenosis.
- It is a rarely performed procedure in the ED but complications of tracheostomy can present to the emergency department.
- The most common two complications that come to the ED are the tracheostomy tube falls out or plugs up, either way the tracheostomy may need changing.
- Often one can suction or clean the obstructed tube or replace the old tube with a new Shiley cuffed tracheostomy tube of appropriate size.
- However, should the stoma be compromised by granulation or scar tissue then it can be difficult to replace the tube, in that case surgical consultation might be necessary.
Tracheostomy Emergencies

- In the “obstructed” tracheostomy tube one must differentiate between the tube plugged with debris and tracheal stenosis distally – passage of a suction cannula should be able to differentiate the two.
- Infections around the stoma and/or skin breakdown can occur, most often the patient will need to be admitted for surgical consultation, antibiotics, and in more severe cases endotracheal intubation to allow the area to heal up.
- Aspiration pneumonia and nosocomial pneumonia can occur more often in these patients with the presentation often just a change in mental status; have a low threshold for obtaining a CXR.

- The most ominous tracheostomy emergency is bleeding from erosion of an arterial vessel (innominate) or a tracheoarterial fistula.
- The bleeding can be massive causing compromise swallowing the blood may be very difficult.
- The tracheostomy tube has to be removed however this raises the patient above which is already compromised, in such a case the may have to intubate the patient from above, sometimes this maneuver can tamponade the bleeding but not often.
- Occasionally one can see the bleeder and control it however in most cases these patients need to be taken to the OR to obtain control of the bleeding.
- Vigorous resuscitation with fluids and blood can be required in the ED.

Cricothyrotomy

- Like the tracheostomy this procedure came into practice with the diphtheria epidemics of the late 1800’s as an attempt to avoid the inevitable tracheal stenosis seen with tracheostomy.
- It was termed the “safe tracheostomy”.
- It was then forgotten and reintroduced to medical practice 3 more times with its establishment in the emergency physicians armamentarium as an airway rescue procedure.

- When to perform the procedure and how to perform it is a complex and difficult clinical decision on the part of the ED physician.
- It should be performed in any patient where there is access to the cricothyroid membrane (not obstructed by tumor/abscess/obesity), no expanding hematoma, and no blood clotting disorder in the patient where there is a failure to obtain an airway by orotracheal or other rescue airway means.

Appearance of diphtheria infections with impending airway obstruction.

Small amount of arterial bleeding seen above does not seem like much but shortly afterwards more notable bleeding started and kept increasing, the patient was taken urgently to the OR.
One of the most common complications is that the procedure is done in the wrong place. In the heat of the moment one mistakenly attempts the procedure above the thyroid cartilage. While infrequently performed this is a procedure the emergency physician has to have down in their mind. Periodically it bears one repeating the steps in ones and reviewing the kits to perform the procedure.

While there are many ways to do this procedure most commonly one used today is a percutaneous technique using a variation of the Seldinger wire technique. There are a number of kits available (Cook Medical Melker Kit in particular) that contain all the items necessary in one sealed, sterile package. One adjunctive assist device that I now teach is using an intubating bougie to first go through the cricothyroid membrane after it is incised and passing either a #6 trach tube or 6mm ETT tube over the bougie.

Complications include:
- Waiting too late to perform the procedure and the patient is not salvageable.
- Inability to successfully obtain access to the trachea and establish an airway.
- Placement of the cannula outside of the trachea.
- Bleeding, often from overlying thyroid tissue or vessels over the cricothyroid membrane.