Case Study #1

- Mr. Jones is a 70 year old male (75 kg) who has undergone a small bowel resection for CA.
- **Previous surgery**: Prostatectomy (Prostate CA)
- **Medical history**: Hypertension, Hyperlipidemia, IDDM, Non-smoker
- WWII Veteran. Married and has two daughters who live out of state.
OR course was uneventful. During the 2nd hour of the postanesthetic period he suddenly sits up starts screaming, fighting, biting and jumps out of the stretcher. He is now standing at the foot of the stretcher.

He pulls at his triple-lumen central catheter via the subclavian vein and it gets caught on the rail. The dressing is not intact, tubing and fluid are on the floor.

The system is opened through one port of the catheter hub.
He starts having difficulty breathing, hypotensive, tachycardiac, becomes very pale and almost collapses.

What do you suspect based on the signs and symptoms?
Air Embolus
The insertion site for his central vascular catheter is above the level of the heart. Mr. Jones was standing when the tubing separated from the catheter hub.

The venous pressure at the catheter tip is lower than the atmospheric pressure. When Mr. Jones took a breath, air was sucked into the right side of this heart through the open catheter lumen.
Air will enter the venous system when there is an open communication between atmospheric air and the central veins, and there is a simultaneous decrease in intravenous pressure, as occurs during inspiration.

Negative intrathoracic pressure can occur with:
- Deep inspiration
- Dyspnea
- Hypovolemia
- Upright position
Pathophysiology

Opening into a Central Vein and Subatmospheric Central Venous Pressure

Air Entry into a Central Vein

Mechanical Obstruction in the Pulmonary Outflow Tract

Ischemic Pulmonary Vessel Constriction (Increased Afterload)

Decreased Ejection of Blood Out of the Right Ventricle

Reduced Left Cardiac Chamber Filling (Decreased Preload)

Decreased Cardiac Output

Decreased Systemic Tissue Perfusion

Generalized Tissue Hypoxia

Shock

Death

### Symptoms of Air Emboli


<table>
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<tr>
<th>Cardiovascular</th>
<th>Pulmonary</th>
<th>Neurologic</th>
<th>Non-specific</th>
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<tr>
<td>Mill-wheel murmur</td>
<td>Cyanosis</td>
<td>Confusion</td>
<td>Diaphoresis</td>
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<tr>
<td>Tachycardia, Weak, thready pulse</td>
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<td>Substernal chest pain</td>
<td>Tachypnea</td>
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<tr>
<td>Hypotension</td>
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<td>Pallor, Cyanosis</td>
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<tr>
<td>Jugular venous distention</td>
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<td>Seizures</td>
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<td>Myocardial ischemia</td>
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Air Entrainment

- **Small < 0.5 ml/kg**
  - Decreased ETCO₂, Oxygen desaturation, Altered mental status, Wheezing

- **Medium 0.5-2.0 ml/kg**
  - Wheezing, Hypotension, Breathlessness, Right heart strain, ST changes, peaked P waves, Jugular venous distention, Myocardial ischemia, Altered mental status, Cerebral ischemia, Pulmonary vasoconstriction, Bronchoconstriction

- **Large > 2.0 ml/kg**
  - Chest pain, Right heart failure, Cardiovascular collapse

Air Entrainment

The patient’s response is dependent on variables, including the:

- Volume of air
- Rapidness with which it enters the venous circulation
- Cardiovascular status of the patient
What are your immediate nursing interventions?
Nursing Interventions

- Clamp the open port. The patient may then breathe.
- If there was bleeding through the port, intrathoracic pressure may have been high enough to prevent the air embolism.
- If little or no blood is evident, the chance of a larger air embolism is greater.
Nursing Interventions

- Immediately place the patient in left lateral Trendelenburg position
  - Left lateral Trendelenburg elevates intrathoracic pressure, terminating air entry.
  - It also causes the occlusive air pocket to move away from the pulmonic valve, thereby allowing blood to flow into the pulmonary vessels, and subsequently into the left cardiac chambers.
Nursing Interventions

- **Apply 100% Oxygen by using a non-rebreather face mask.**
  - It enhances oxygen delivery to poorly perfused tissues.
  - Intubation possibility depending on how well he does with the non-rebreather face mask.
  - The embolus is comprised of room air, which is 79% nitrogen. It hastens the absorption of the air embolus by washing out alveolar nitrogen, thus increasing diffusion of nitrogen in the air embolus across the alveoli (into the lungs) where it is rapidly exhaled (blown-off).
Other Possible Interventions

- Aspiration of the air bolus through an intact central line.
- Intracardiac aspiration with the assistance of cardiac or color flow Doppler ultrasonography
- Closed chest massage to displace the air away from the pulmonary outflow.
- Hyperbaric therapy
- Left lateral Trendelenburg is easily and quickly accomplished with few potential detrimental effects; it is suggested as the initial intervention.
A post-mortem chest radiograph demonstrating air filled heart chambers

Release of air (bubbles) from the right atrium when opened during autopsy

Case Study #2

- Mr. Smith is a 75 year old male (100 kg) who has undergone a liver resection for liver CA.
- Previous surgeries: Mohs for basal cell CA, cholecystectomy for gall stones a month ago
- Medical history: Congestive heart failure, Hypertension, Hyperlipidemia, IDDM, Smokes 1-2 packs/day, Crohn’s disease
- Married with three children- two sons and one daughter. The daughter is an Emergency Room Nurse.
- He is retired. His past occupation was as a truck driver.
OR course uneventful. During the 3rd hour of the postanesthetic period he suddenly drops his oxygenation saturation to 88% on 40% face mask

- Respiratory Rate is 45 labored using accessory muscles
- Heart Rate is 135- Normal Sinus Tachycardia
- No change in BP
- c/o pleuritic chest pain- sharp and stabbing, becomes worse when he breathes deeply
- Breath sounds- crackles in lower bases which were not present before
What do you suspect based on signs and symptoms?

Is it an Myocardial Infarction or a Pulmonary Embolus?
Interventions

- Increase Oxygen to 100% non-rebreather face mask.
- 12 Lead EKG and draw cardiac markers (Troponin and cardiac enzymes) to R/O MI.
- Draw ABG
- Chest X-ray
Troponin level and cardiac enzymes were negative
12 lead EKG nonspecific with T wave inversions
68% of PE patients have T-wave inversions

Poor prognosis with previously normal EKG who develop

- Atrial arrhythmias
- RBBB
- ST elevation changes or depression over the precordial leads
Pulmonary Embolus
Risk Factors – Virchow’s Triad

- Hypercoagulability
  - Advanced age
  - Pregnancy/oral contraceptives
  - Cancer
  - Smoking
- Personal or family history of VTE
- Crohn’s disease
- Nephrotic syndrome
- Platelet abnormalities and inherited or acquired thrombotic disorders (antithrombin III, protein C or S deficiency, etc.)
Risk Factors – Virchow’s Triad

- Blood stasis
  - Advanced age
  - Bedrest/hospitalization/institutional care
  - Obesity
  - Heart failure
  - Stroke
  - Cancer
- Chronic obstructive pulmonary disease
- Long distance travel
- Chronic venous insufficiency/varicose veins
- Hyperviscosity syndromes
Risk Factors – Virchow’s Triad

- Injury of vessel wall
  - Surgery
  - Trauma
  - Fractures

LIP has a high clinical suspicion based on:
- History
- Analysis of risk factors
- Signs and Symptoms
- First level examinations
  - EKG
  - Chest X-ray
  - ABG

Clinical Pre-test Probability
- Wells Score
- Geneva Score
No single exam finding can diagnose a PE because the most common signs and symptoms for PE are not very specific.

The most common signs and symptoms, dyspnea, pleuritic pain, cough, tachypnea, and crackles, can be seen in number of other disease processes including congestive heart failure, pneumonia, etc.

However, one element that is consistent in all the studies is that a patient’s pretest probability is assessed.

The number of risk factors present will dictate the tests needed to diagnose the condition.
## Tests

- **ABG** – pH 7.50  PO$_2$ - 70  PCO$_2$ - 30 HCO$_3$ - 26
- Chest X-ray shows atelectasis
- Echocardiogram
- Pulmonary helical computer tomography (CT)
- Angiography
- Ventilation Perfusion (V/Q) Scan
- Venous Compression or color ultrasonography of the lower extremities
- Draw Elisa D-Dimer Assay

  - D-dimers are released as a result of fibrinolysis, suggesting the presence of intravascular thrombosis

Pulmonary Embolism

- Bronchial Vein
- Embolus
- Right Lung
- Pulmonary Artery
- Left Lung
Treatment

- In unstable patients affected by massive PTE, the treatment of choice is:
  - Thrombolysis followed by IV unfractionated heparin (UFH) and oral anticoagulant therapy (Vitamin K antagonists such as warfarin)

- In hemodynamically stable patients affected by sub-massive PTE, the treatment of choice is:
  - IV intravenous UFH, followed by oral anticoagulants

- In stable patients with non-massive PTE the choice treatment is:
  - Low molecular weight heparin (LMWH) twice a day followed by oral anticoagulants

Continuous Monitoring

- Airway Management
- Check ABGs
- Does he need to be intubated?
- If he drops his blood pressure?
- Vasopressor support - inotrope - Dopamine
- Check labs - coagulation, PT/PTT, platelet count, INR
Case Study # 3

- The anesthesia provider brings out to your PACU Ms. Soon who is a 47 year old female who has undergone a lumpectomy.

- **Medical history:** hypertension, IDDM, No previous surgeries.

- She is still intubated and is placed on a ventilator.

- During handoff, the anesthesia provider states the OR course was uneventful except after the rapid induction of propofol and succinylcholine. He noticed that when he assessed her using a train of four (TOF) there were no twitches which indicates 100% block. After the procedure there was still no response to the TOF.
Train of Four (TOF)-Peripheral Nerve Stimulator

- The expected response is twitches of the thumb toward the hand. The number of twitches corresponds to the level of paralysis.
  - 4 indicates 75%
  - 3 indicates 80%
  - 2 indicates 85%
  - O twitches indicates 100% blockage.
What do you suspect based on this information?
Pseudochocholinesterase Deficiency or Delayed Awakening From Anesthesia
Pseudocholinesterase Deficiency

- An **inherited enzyme abnormality** that results in abnormally slow metabolism degradation of exogenous choline ester drugs such as succinylcholine and mivacurium (muscle relaxants).

- Affects 1 in 2,500 to 1 in 2,800 individuals

- It is identified when an anesthetized patient has prolonged paralysis after receiving neuromuscular blocking agents dependent on pseudocholinesterase enzymes for hydrolysis.
Pseudocholinesterase Deficiency - Pathophysiology

- The effectiveness of pseudocholinesterase can be hampered in three (3) ways:
  - **Decrease in total amount** of pseudocholinesterase – most commonly associated with hepatic dysfunction (hepatitis, cirrhosis, and liver metastases).
  - **Decrease activity** of pseudocholinesterase – associated with late stages of pregnancy, estrogen therapy, and pesticide poisoning
  - **Unusual genetic variants** of pseudocholinesterase (genetic absence)
Pseudocholinesterase Deficiency

- This is a silent condition that only manifests itself when people who have the deficiency receive the muscle relaxants succinylcholine or mivacurium during a surgery.
- Succinylcholine and mivacurium are metabolized by plasma cholinesterase.
- Unreversible muscle weakness ("floppy") and apnea.
Pseudocholinesterase Deficiency - Management

- Requires **mechanical ventilation to support respiration**. Necessary until muscle strength gradually returns.
- Psychological support, sedation.
- Constant vigilance: **patient alert**, fearful, feel pain.
- May order **dibucaine levels**. The dibucaine number indicates the percent inhibition of enzyme activity by this agent.
Delayed Awakening From Anesthesia
Causes of Delayed Awakening

- Residual drug effect
- Duration and the type of anesthetic given
- Potentiation by other drugs
- Prolonged Neuromuscular blockade
- Metabolic and endocrinal disorders
- Acid-base and electrolyte imbalance
- Hypothermia
- Neurological complication
Management

- Airway, Breathing, Circulation
- Temperature
- Intensive Monitoring
- Review the history, investigate peri-operative management including the anesthesia record and timings of drug administration
- Assess for persisting NM blockade using the nerve stimulator (TOF). Repeat reversal if needed.
- Give naloxone- for suspected opioid narcosis
- Give Flumazenil- for Benzodiazepine overdosage
Management

- Check blood glucose and treat accordingly
- Correct acid-base imbalance if indicated
- If no other cause found, an intracerebral event may be suspected and a full neurological evaluation should be performed. Radiological Imaging (CT or MRI) is often required.
