1 **Unexpected Emergencies in the PACU**

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2 **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.
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- ❑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 cathether 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.
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- ❑He starts having difficulty breathing, hypotensive, tachycardic, becomes very pale and almost collapses.
- ❑What do you suspect based on the signs and symptoms?

5 **Air Embolus**

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- ❑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.
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- ❑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
  - ❑
  - ❑

8 **Pathophysiology**

9 **Clinical Findings in Air Embolus**

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1 Symptoms

2 ❑Pulmonary

- ❑Dyspnea (100 percent incidence)
- ❑"Gasp reflex"-when a bolus of air enters the pulmonary circulation
- ❑"Sucking sound"-when air is sucked into intravascular space

❑Neurologic

- ❑Sense of doom

- ❑Dizziness/lightheadedness
- Physical Findings
- ❑Tachypnea
- ❑Wheeze
- ❑Rales/Crackles
- ❑Respiratory failure
- ❑
- ❑
- ❑
- ❑
- ❑Change in mental status
- ❑Focal neurological deficits

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- Symptoms
- ❑Cardiac
  - ❑Substernal chest pain
- ❑
- ❑
- ❑
- ❑
- ❑Skin
- ❑
- ❑
- ❑Ocular
- Physical Findings
- ▶
- ❑Hypotension
- ❑Tachycardia
- ❑Mill wheel murmur
- ❑Signs of right heart failure
  - ❑Elevated JVD
- ❑Shock
- ❑
- ❑Crepitus over superficial vessels (seen rarely in setting of massive air embolus)
- ❑Livedo reticularis
- ❑
- ❑
- ❑Bubbles within the retinal arteries

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**Air Entrainment**

- ❑Small < 0.5 ml/kg
  - ❑Decreased ETCO2, Oxygen desaturation, Altered mental status, Wheezing
- ❑Medium 0.5-2.0ml/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 > 2.0ml/kg
  - ❑Chest pain, Right heart failure, Cardiovascular collapse

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**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

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What are your immediate nursing interventions?

15 **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.

16 **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.
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17 **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).
- ❑

18 **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.

19 **A post-mortem chest radiograph demonstrating air filled heart chambers**

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

21 **<u>Case Study #2</u>**

- ❑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.
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- ❑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

23 **What do you suspect based on signs and symptoms?**
Is it ACS or a Pulmonary Embolus?

24 **Interventions**
- ❑ Increase Oxygen to 100% non- rebreather face mask.
- ❑12 Lead EKG and draw cardiac markers ( Troponin and cardiac enzymes) to R/O ACS
- ❑Draw ABG
- ❑Chest X-ray

25 **Troponin level and cardiac enzymes were negative.12 lead EKG nonspecific with T wave inversions.**

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- ❑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
  - ❑

27 **Pulmonary Embolus**

28 **Pulmonary Embolus**
- ❑It is a common and sometimes fatal disease.
- ❑It is due to obstruction of a pulmonary artery or one of its branches by material (e.g. thrombus, tumor, air, or fat) that originated elsewhere in the body. Most emboli arise from lower extremity proximal veins (iliac, femoral, and popliteal).
- ❑The overall incidence of PE is approximately 112 cases per 100,000. It is more likely in males than females and incidence increases with age.
- ❑Deaths from PE account for approximately 100,000 deaths per year in the United States.
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30 **Risk Factors – Virchow's Triad**
- ❑Hypercoagulability
  - ❑Advanced age
  - ❑Pregnancy/oral contraceptives
  - ❑Cancer
  - ❑Smoking
  - ❑Personal or family history of VTE
  - ❑Crohn's disease
  - ❑Nephritic syndrome
  - ❑Platelets abnormalities and inherited or acquired thrombotic disorders (antithrombin III, protein C or S deficiency, etc.)
  - ❑
  - ❑

31 **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

32 **Risk Factors – Virchow's Triad**

- ❑Injury of vessel wall
  - ❑Surgery
  - ❑Trauma
  - ❑Fractures

33 **Classification of Pulmonary Embolus**

- ❑Presence or absence of hemodynamic stability
  - ❑Hemodynamically unstable ( "massive" or "high-risk" PE.)
    - ❑SBP < 90 mmHg or a drop in SBP of ≥40 mmHg from baseline for >15 minutes. More likely to die from obstructive shock in the first two hours of presentation.
  - ❑Hemodynamically stable "submassive" or "intermediate-risk" if associated with right ventricular strain; or "low-risk" if there is no evidence of right ventricular strain.
- ❑The presence or absence of symptoms (symptomatic or asymptomatic).

34 **Classification of Pulmonary Embolus**

- ❑The temporal pattern of presentation:
  - ❑Acute- develop symptoms and signs immediately after obstruction of pulmonary vessels.
  - ❑Subacute-may also present within days or weeks following the initial event.
  - ❑Chronic- slowly develop symptoms of pulmonary hypertension over many years (i.e. chronic thromboembolic pulmonary hypertension (CTEPH).
- ❑The anatomic location:
  - ❑Saddle- lodges at the bifurcation of the main pulmonary artery, often extending into the right and left main pulmonary arteries. (3% to 6%)
  - ❑Most PE move beyond the bifurcation to lodge distally in the main lobar, segmental, or subsegmental branches of a pulmonary artery.

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- ❑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 at rest or with exertion (73%)
  - ❑Pleuritic chest pain (66%)
  - ❑Cough (37%)
  - ❑Tachypnea (70%)
  - ❑Orthopnea (28%)
  - ❑Calf or thigh pain and/or swelling (44%)
  - ❑Wheezing (21%)
  - ❑Hemoptysis (13%)

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- ❑These signs and symptoms 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 patients pretest probability is assessed.
- ❑The number of risk factors present will dictate the test needed to diagnose the condition.

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

❑Clinical Pre-test Probability
- ❑Wells Score
- ❑Geneva Score
  - ❑
  - ❑
  - ❑

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40 **Tests**

❑ABG – pH 7.50 PO 2- 70 PCO2 -30 HCO3-26
❑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

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41 **Pulmonary Embolus**

42 **Chest X-RAY**
**- Hampton's Hump, Westermark's Sign**

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46 **Treatment**

❑PE left untreated, has an overall mortality rate of up to 30%, which is significantly reduced with anticoagulation.
❑Initial resuscitative therapy for patients with suspected PE should focus upon oxygenating and stabilizing the patient.
❑Once the diagnosis is made, the mainstay of therapy is anticoagulation, depending upon the risk of bleeding.
❑Alternative treatments include:
- ❑Thrombolysis
- ❑Inferior vena cava filters
- ❑Embolectomy

47 **Risk factors for bleeding with anticoagulant therapy**

2 ❑Age >65 years
❑Age >75 years
❑Previous bleeding
❑Cancer
❑Metastatic cancer
❑Renal failure
❑Liver failure
❑Thrombocytopenia
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4 ❑Previous stroke
❑Diabetes
❑Anemia
❑Antiplatelet therapy
❑Poor anticoagulant control
❑Comorbidity and reduced functional capacity
❑Recent surgery
❑Frequent falls
❑Alcohol abuse
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48 **Continuous Monitoring**

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

49 **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. After the procedure there was still no response to the TOF.
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50 **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.

51 **What do you suspect based on this information?**

52 **Pseudocholinesterase Deficiency or Delayed Awakening From Anesthesia**

53 **Pseudocholinesterase Deficiency**

❑Inherited enzyme abnormality which results in a slow metabolism of exogenous choline ester muscle relaxants (succinylcholine & mivacurium)

- Affects 1 in 3,200 to 1 in 5,000
- Individuals of Persian-Jewish decent and Alaskan Native American are at high risk
- Silent condition
- Only manifests itself when patient receives muscle relaxants succinylcholine or mivacurium during surgery
- Un-reversible muscle weakness

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54 **Pseudocholinesterase Deficiency**

- Can be occur in three ways:
  - Decrease in amount: most commonly associated with hepatic dysfunction
  - Decreased activity: associated with late stages of pregnancy, estrogen therapy, and pesticide poisoning
  - Genetic variant: inherited absence

55 **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.

56 **Delayed Awakening from Anesthesia**

57 **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

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59 **Patient Factors**

- Gender:
  - Apfelbaum *et al.* reported that men are 1.4 times more likely to have delayed recovery than women.
  - Lower sensitivity to the hypnotic effect of anesthetics in women may account for their faster recovery.
  - The female sex hormone was postulated to play a role in the gender differences in recovery time.
- Genetic factors:
  - It is becoming increasingly apparent that genetically controlled variations in drug disposition and response are important determinants of adverse effects of drug therapy.
  - Unexpected responses and prolonged somnolence after specific anesthetic are commonly associated with a genetic defect of the metabolic pathway of the agent or its receptor.

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60 **Patient Factors**

- Geriatric patients
  - Elderly patients have increased sensitivity toward general anesthetics, opioids and benzodiazepines, and slow return of consciousness due to progressive decline in central

nervous system (CNS) function.
- ❑Studies have shown that demand for opioids is reduced by almost 50% in geriatric patients. The decrease in volume of distribution, clearance rate, and plasma protein binding results in high free plasma concentration of drugs.
- ❑Pediatric patients
  - ❑Because of larger body surface area, heat loss is greater in children resulting in hypothermia, slow drug metabolism, and delayed return of consciousness.

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61 **Management**
- ❑Airway, Breathing, Circulation
- ❑Temperature-correct hypothermia or hyperthermia
- ❑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

62 **Management**
- ❑Check blood glucose and treat accordingly
- ❑Correct acid-base imbalance if indicated
- ❑Correct electrolyte abnormalities
- ❑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.

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