Air Travel with Respiratory Disease

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Introduction
• Over 25 years ago it was already estimated that more than 5% of air travelers have a chronic respiratory disease
• Over one billion people travel by air each year
• No established method for quantifying risk
• 11% of in-flight emergencies are respiratory
• Greater awareness of risks will lead to greater safety

Challenges
• Aircraft crew subject to medical exams but passengers are not
• There are disparities between North American and European guidelines
• Uncertainty about assessment methods
• Misdiagnosis

Recommendations
• To enhance safety of passenger with lung disease and decrease in-flight medical incidents
• To increase recognition among health care professionals
• To provide up to date literature
• To provide consistent, practical and comprehensive advice

Recommendations
• To formulate key research questions to provoke further investigation
• To provide consistent, practical, and comprehensive advice for managing patients
• To promote the development of methods for monitoring the size of the problem.

The Flight Environment and Effects of Altitude
• Modern aircraft are pressurized to cabin altitudes of up to 8000 feet
• At 8000 feet, the partial pressure of oxygen drops to the equivalent of breathing 15.1% oxygen at sea level
• In a healthy patient, the PaO2 will fall to 53-64 mm Hg (Sp02 85-91%)
• If the patient already compromised, then???
Pre-flight assessment for adults

- The following groups should be assessed
- Severe COPD or asthma
- Severe restrictive disease, especially with hypoxemia or hypercapnia
- Cystic Fibrosis
- Travel intolerance with respiratory symptoms such as dyspnea, chest pain, confusion, syncope

Pre-flight Assessment

- Conditions worsened by hypoxemia such as cerebrovascular disease, coronary heart disease, heart failure
- Pulmonary TB
- Within 6 weeks of hospital discharge for acute respiratory illness
- Recent Pneumothorax

Preflight Assessment

- Risk of venous thromboembolism
- Pre-existing requirement for oxygen or ventilator support

Assessment

- History and examination with emphasis on cardiorespiratory disease, dyspnea and previous flying experience
- Spirometric tests
- Measurement of SpO2 by pulse oximetry
- ABG if hypercapnia is known or suspected
- If O2 sat is 92-95% hypoxic challenge test (in the pulm lab) is recommended. Or walk 50 meters or ascend a flight of stairs.

Who should not fly?

- Patients with active TB
- Need three negative smears or a negative culture result
- Those with a untreated pneumothorax. After weeks if treated.
- 6 weeks after major thoracic surgery with a medical assessment pre-flight
- Lung cancer itself not a contraindication but consider comorbid conditions.

Don’t fly until…..

- 10 days following abdominal surgery
- 24 hours after a colonoscopy
- 24 hours after a laparoscopy
- 7 days after neurosurgery
- 6 weeks after surgery for retinal detachment
- 1 week after other eye surgery
Additional Precautions for all passengers

- Avoid excessive alcohol especially if OSA or VTE risk
- Remain mobile
- If indicated, wear oxygen when walking
- Prophylactic measure if at risk of VTE
- Carry inhalers
- CPAP machine can be worn during the flight
- Vent dependent patients need a detailed physician letter and a medical attendant

Supplemental Oxygen

- Supplementary inflight oxygen usually give at a rate of 2l/min via nasal cannula
- Usually arranged by the airline
- Turned on when plane hits cruising altitude
- May be switched off upon descent
- If already on oxygen at home, should be increased on the plane at cruising altitude

Acute Illness in Aerospace: Sudden Decompression

- Can occur following a breach in the fuselage, failure of air compression systems, or unrestrained ascent
- Acute hypoxia due to reduced PaO2
- “Useful consciousness” a few second to minutes
- Remedy is breathing O2 from contained source and rapid descent to a lower altitude and higher pressure

Sudden Decompression

- Physiologic protection provided by aircraft pressurization
- This is accomplished by increasing the pressure above ambient pressure within the crew and passenger compartments
- In decompression, the effects on the body are primarily due to the rate of pressure loss and the pressure differential to which the body is exposed

Sudden Decompression

- Slow decompression >hypoxia symptoms
- Rapid decompression > physiological injury due to expansion of gas in the lungs and GI tract.
- After the decompression > physiological consequences such as acute hypoxia and decompression sickness

Gases in Acute Decompression: GI Tract

- Abdominal distress during rapid decompression usually no more severe than during lower
- Diaphragm is displaced downward by expansion of trapped gas in the stomach, reducing respiratory excursion
- Distention of abdominal organs may stimulate the abd branches of the Vagus nerve, resulting in cardiovascular depression and shock
Mechanical Expansion of Gases: The Lungs

- Most vulnerable organ system during a rapid decompression
- A transient positive pressure can build up in the lungs
- If the escape of air is blocked, can overdistend
- For example, forceful breathholding could result in tearing and rupture of lung tissues and capillaries leading to massive air bubbles traveling thru body and lodging in critical organs such as heart and brain

Altitude Decompression Sickness “DCS”

- Occurs during ascent
- Less common than in diving
- Risk increases with degree of hypobaric exposure, duration, and decreases with pre-exposure O2 treatment
- Threshold for development of DCS is 18,000 to 25,000 feet above sea level.
- 55% DCS incidence at 22,500 feet (Webb, et al)
- Over 35,000 feet can lead to severe DCS

DCS

- Exposures involving exercise result in higher incidence and earlier onset of symptoms
- Repeated simulated altitude exposures to 25,000 feet with 100% O2 significantly reduces DCS compared with a single exposure.

Pathophysiology of Pulmonary Barotrauma

- If the lung filter is overwhelmed, or the bubbles pass into the systemic arteries by an atrial septal defect, they may open the blood brain barrier, effecting brain and spinal cord function
- In untreated DCS, demyelination with preservation of axons may occur, like in MS
- Joint pain “the bends” is associated with gas in connective tissue
- This requires urgent compression in a hyperbaric chamber with high partial pressures of O2

Clinical Manifestations of Decompression Sickness

- Susceptibility related to age
- 300% increase in those over 42 years
- No gender difference, except for women on hormonal contraception
- Persons of either sex with higher BMI and lower physical fitness developed DCS more frequently
Clinical Manifestations of DCS

- Symptoms occur from 10 minutes to 12 hours after surfacing in 90% of cases
- Type 1 DCS includes mind manifestations such as joint pain (knees), muscle, skin and lymph nodes
- Type 2 DCS have pulmonary, cardiac, or CNS involvement
- Pulmonary symptoms from nitrogen bubbles include "the chokes," cough, dyspnea, chest pain, hemoptysis and/or severe hypoxemia

Clinical Manifestations of DCS

- DCS occurs during a flight because decompression occurs as the flight begins and symptoms typically improve with descent
- Delayed symptoms may occur
- Treatment with O2 and return to sea level succeeds in most patients
- Some patients require hyperbaric treatment
- In one study of initial complaints 82% of patients had musculoskeletal complaints, 2.7% chokes, 2.2% skin, 10.8% paresthesias, 0.5% neurologic

Type 2 DCS

- Wide spread of symptoms, can be difficult to diagnose
- Most common are joint pain (43%), headache (42%), visual disturbances (30%), and limb paresthesias (27.8%)
- Next most common symptoms are mental confusion (25%), limb numbness (16.5%), and extreme fatigue (10.5%)

Treatment for Altitude Decompression Sickness

- Doppler test can be used to detect microbubbles
- Treatment is 100% O2 and positioning in left decubitus and mild Trendelenburg position
- Patients with type II DCS should receive HBP as soon as possible
- Mainstay of treatment for gas bubble disease is therapeutic recompression while the patient is breathing supplemental O2
- This should be done as soon as possible but can be helpful after several days
- Should be repeated until patient stabilized

Treatment of DCS

- Adequate hydration essential
- HBO is safe, nontoxic and can be used even in neonates
- Pharmacologic agents are under study, including lidocaine, anticoagulants, steroids and calcium antagonists
- HBO treatment follows the diving protocol
- A newer treatment table, for higher altitudes, consists of 100% O2 delivered at 2 ATA for four 30-min periods with intervening 10-min air breaks (total treatment 2h). 91% successful.

Venous Gas Embolism

- Gas enters the systemic venous circulation due to the presence of sub-atmospheric pressure in these vessels
- The gas is transported to the lungs through the pulmonary arteries, causing interference with gas exchange, cardiac arrhythmias, pulmonary hypertension, right ventricular strain and eventually cardiac failure
### VGE
- “Millwheel murmur” is a splashing auscultatory sound due to the presence of gas in the cardiac chambers and great vessels.
- A decrease in end-tidal CO2 levels may result from the mismatch in ventilation and perfusion.
- Doppler ultrasound is sensitive and practical to detect intracardiac air.
- Transesophageal echo is more sensitive and definitive.

### VGE Treatment
- Placed in flat supine position.
- Supplemental O2 increases the gradient for nitrogen to egress from the bubbles.
- Volume expansion increases venous pressure, preventing the continued entry of gas into the venous circulation.
- HBO if neurologic changes.
- Some authors recommend a central line be placed to evacuate air from the right ventricle.

### Pulmonary Thromboembolism
- Long been recognized as a potential complication of travel.
- Virchow’s triad plus dehydration and reduced atmospheric pressure with reduced saturation.
- Other factors include age, obesity, previous venous disease, immobility, cardiac failure, chronic medical illness, cancer, thrombophilia, recent major surgery, contraceptives and hormone replacement.

### VTE: Incidence
- Incidence of VTE between 4.5% and 11% by ultrasound in passengers flying more than 8 hours.
- Lower risk in aisle seats.
- Scholl flight socks effective in reducing the risk.
- Compressive elastic stockings recommended in at risk passengers.
- Other prophylactic measures include active dorsiflexion of the foot, occasional walks around the cabin and physical activity at flight stops.

### VTE Prophylaxis
- Low dose aspirin for those with mild/moderate risk.
- 2 hours before takeoff and 24 hours after.
- Reduces the risk by about one third.
- For high risk, low molecular weight heparin 2 hours before take off.

### Systemic Arterial Gas Embolism
- Usually associated with underlying lung disease.
- Initial event is inflation of alveoli with leakage of air into the interstitial space.
- Embolism is caused by the entry of the gas into the pulmonary veins or directly into the arteries of the systemic circulation.
- May occur after the accumulation of of bubbles in the pulmonary artery raises right heart pressure enough to create right to left shunt through a previously closed patent foramen ovale.
- Obstruction can occur of coronary or cerebral arteries and may be fatal.
SAGE

- Entry of gas into the aorta causes distribution of gas bubbles into nearly all organs
- Small emboli in skeletal muscle well-tolerated
- Emboli into cardiac vessels induces EKG changes typical of ischemia; infarction and arrhythmias and infarction are possible, depending on the amount of gas embolized
- Emboli to cerebral vessels can cause pathologic changes by reduction of perfusion distal to the obstruction and an inflammatory response to the bubble

SAGE Symptoms

- Develop suddenly
- Clinical presentation varies based on the quantity of gas and the areas affected
- There may be minor motor weakness and headache or moderate confusion; complete disorientation, hemiparesis, convulsions, loss of consciousness and coma.
- Other well-known symptoms include asymmetry of the pupils, hemianopia, and impairment of the respiratory and circulatory centers, manifest as bradypnea, Cheyne-Stokes breathing, arrhythmias, or circulatory failure

SAGE Diagnosis

- In an aerospace flier who has an injury, evaluate with PFTs, CT scan of the lung, and bubble contrast echocardiography
- In cerebral injury, MRI may show increased volume of water in the injured tissue
- A nonspecific finding is an increased hematocrit, possibly due to extravascular shift of fluid into the injured tissues

SAGE Treatment

- Primary goal is the protection and maintenance of vital organs
- High FIO2 helps
- HBO in acute conditions
- No air travel until 14 days post-pneumothorax

References

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