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**Atrial Fibrillation Post-Lung Lobectomy**

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**Introduction**

According to the American Heart Association, atrial fibrillation affected 3.6 million individuals in the United States in 2010 and is expected to double by year 2050. Atrial fibrillation (AF), a common arrhythmia, can occur after thoracic surgery and is an ongoing concern for postoperative morbidity and mortality (Imператор et al., 2012). There is a 20-25% occurrence rate of atrial fibrillation post noncardiac surgery, such as lung lobectomy, and a 40% incidence rate after aortic surgery (D’Amore & Kamara, 2012, p. 429). Cardiovascular events such as death, nonfatal myocardial infarction, readmission, and failure, and infection are significant complications following thoracic surgery and can result in increased length of hospitalization and care, and increased nursing support (Ladder & Rutherford, 2014, p. 274). The impact of POAF on patient outcomes has prompted nurses to seek to better understand the most significant symptoms of POAF, including diaphoresis, and possibly reducing the financial burden to patients experiencing POAF by reducing treatment costs and length of hospital stay (Imperial Laidler, 2014, p. 274).

**Legend**

Cancer in the liver of lung

**POAF Pathophysiology**

Atrial fibrillation is defined as a tachycardia characterized by predominantly uncoordinated atrial activation with consequent deterioration of atrial function (Conti, 2012, p. 18). AF occurs when the electrical impulse is not initiated in the sinus (SA) node and instead is initiated by atrial cells over multiple re-entry circuits in the atria (Lader & Rutherford, 2014, p. 274). This fibrillation may occur at baseline in those with a normal heart rate or may cause AF in those with a baseline heart rate slower than 40 beats per minute. AF has been said to be capable of causing coronary artery ischemia, and potentially infarction if the myocardium cannot get blood flow at normal rates through the ventricles. The incomplete emptying of the heart is associated with a decrease in diastolic filling and a possible clot formation, which is a major complication of AF. The presence of a clot in the left atrium has been associated with an increased risk of cardioembolic stroke (Kanmura, Omae, & Rutherford, 2014, p. 210, 2018, 2031). According to Chelazzi, Villa, and De Gaudio (2011), the electrophysiological mechanisms involved in POAF are not fully understood; however, the mechanisms are multifactorial (Lader & Rutherford, 2014, p. 273). Researchers believe that patients who develop POAF may already have had an electrophysiological substrate for this arrhythmia before thoracic surgery, which may have been present but undetected before thoracic surgery. Mechanical manipulation of the heart during thoracic surgery leads to local tissue trauma and an inflammatory response. Increased sympathetic activity from this inflammatory response is in addition to the surgical trauma and pain. Circulating catecholamines may act on sensitized myocardial cells causing a shortened refractory period and an increased heart rate (Chelazzi, Villa, & De Gaudio, 2011). This local inflammation, and elevation in arterial catecholamines within the operative atrium, may result in atrial strain and atrial fibrillation. POAF associated factors in eliciting new atrial fibrillation include associated factors in which mechanism is causing the POAF such as surgical complications or acute surgical precipitates, the patient’s medical history, and the best intervention to correct this cardiac arrhythmia. By learning more about POAF, it may be possible to prevent POAF. In addition to hypothesis, intraoperative hypoxia and anemia are known to contribute to POAF by sensitizing myocardial tissue and conduction tissue cells and atroventricular cells. This alteration can modify the cell’s electrical activity and cause spontaneous arrhythmias (Chelazzi, Villa, & De Gaudio, 2011).

**Common Symptoms of POAF**

- **Severe Signs and Symptoms of POAF**

  - Hypotropy
  - Hypoxia
  - Hypoventilation
  - Dyspnea
  - Fatigue

- **Common Symptoms of POAF**

  - Palpitations
  - Fatigue, generalized weakness
  - Dizziness
  - Shortness of breath

**Pathophysiology of POAF. (Chelazzi, 2010)**

The peak of sympathetic activation occurs within 24 hours post-operatively, thus the onset of POAF usually develops between 48 to 72 hours after thoracic surgery. (Maesen, Nij, Maesen, Alles, & Schotten, 2012, p. 18). Pain alone, during the postoperative period, can trigger a sympathetic response which can result in increased sympathetic output and an imbalance between sympathetic and parasympathetic activity causes atrial ectopic beats and may cause POAF. In addition, maladaptive indicators such as inadequate post-operative pain-control is associated with a decreased occurrence of POAF. Thoracic epidural anesthesia (TEA) may reduce POAF because this produces a negative chronotropic effect on the heart. Increased sympathetic activity can cause POAF, which is associated with increased sympatho-adrenergic activity. POAF morbidity and mortality can occur from POAF. (Chelazzi, Villa, & De Gaudio, 2011). The low magnitude of potassium and magnesium preoperative changes in arterial potentials and the pharmacological activity of the heart rate and the maximum degree of the heart rate are preoperative changes which may result in POAF. The peak of sympathetic activation occurs within 24 hours post-operatively, thus the onset of POAF usually develops between 48 to 72 hours after thoracic surgery. (Mansin Nij, Maesen, Alles, & Schotten, 2012, p. 188). POAF is a result of sympatho-adrenergic activity. POAF can be used as an index of the sympatho-adrenergic activity, and the sympatho-adrenergic activity is related to POAF. POAF is associated with a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF is associated with a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF. POAF may be induced by a rise in plasma catecholamines, which is a major cause of POAF.