CHAPTER 235

Hypoxia: Hypoventilation

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CASE SYNOPSIS

A 63-year-old man with emphysema has a decrease in oxygen saturation to 90% in the postanesthesia care unit (PACU) after general anesthesia for exploratory laparotomy. He takes ipratropium and uses albuterol inhalers at home. A central line was placed in the right internal jugular vein prior to induction. The intraoperative course was unremarkable, and the patient was extubated in the operating room. Vital signs in the PACU are as follows: heart rate, 105 beats/minute; blood pressure, 105/63 mm Hg; and respiratory rate, 32. Approximately 30 minutes later, the patient complains of difficulty breathing.

PROBLEM ANALYSIS

Definition

"Hypoxia" is differentiated from "hypoxemia" in *Stedman's Medical Dictionary*. Hypoxia exists when a subnormal level of oxygen is present in the inspired gas, arterial blood, or tissue. Hypoxia is further divided into four categories: (1) Anemic hypoxia occurs from a decrease in functional hemoglobin content. (2) Hypoxemic hypoxia occurs from inadequate oxygenation of the lungs. (3) Circulatory hypoxia occurs from inadequate perfusion. (4) Histotoxic hypoxia occurs from inadequate utilization of oxygen at the cellular level.

Hypoxemia exists when subnormal oxygenation of arterial blood is present, resulting in an arterial oxygen tension (Pa_{O_2}) of less than 60 mm Hg. The primary causes of hypoxemia are low fraction of inspired oxygen (F_{IO_2}), hypoventilation, ventilation/perfusion mismatch, true shunt, and diffusion impairment. Normal Pa_{O_2} can be calculated by the following equation: Pa_{O_2} = [illegible]/3. Note that Pa_{O_2} decreases with aging, whereas Pa_{CO_2} remains relatively constant.

Recognition

Hypoxemia can be detected easily and rapidly in the PACU by routine use of pulse oximetry and confirmed with arterial blood gas measurements (Pa_{O_2} less than 60 mm Hg). Early recognition can facilitate therapeutic intervention before life-threatening hypoxemia develops. A Pa_{O_2} of less than 60 mm Hg corresponds to the steep portion of the oxyhemoglobin dissociation curve, with the oxygen content of the blood decreasing by incrementally greater amounts for the same decrease in Pa_{O_2}.

Clinical features of hypoxia may overlap with those of hypercapnia. The early clinical signs of hypoxemia are tachycardia, tachypnea, agitation, and altered mental status, whereas hypotension, bradycardia, obtundation, and cardiac arrest are late signs.

The alveolar-arterial oxygen difference [$P(A - a)O_2$] is normally less than 20 mm Hg and can be calculated when hypoxemia occurs. When hypoxemia is secondary to hypoventilation or decreased F_{IO_2}, the $P(A - a)O_2$ gradient is normal. When hypoxemia is secondary to ventilation/perfusion mismatch, true

shunting, or diffusion impairment, the $P(A - a)O_2$ gradient is increased.

The hallmark of hypoventilation is increased $PaCO_2$, and with supplemental oxygen, the PaO_2 increases. Ventilation/perfusion mismatch and diffusion impairment are characterized by their partial response to oxygen administration. True shunting is diagnosed when supplemental oxygen fails to substantially increase PaO_2.

Risk Assessment

The etiology of pulmonary dysfunction in the postoperative period is multifactorial. The preoperative pulmonary risk factors for postoperative hypoxemia are preexisting lung disease, thoracic or upper abdominal surgery, obesity, advanced age, smoking, and prolonged surgery.

Anesthesia has profound effects on pulmonary mechanics. A decrease in functional residual capacity (FRC) occurs with general anesthesia. Weakness of respiratory muscles can occur with regional anesthesia, primarily affecting patients with obstructive lung disease, since they are dependent on accessory muscles for exhalation. Lung volumes and compliance are decreased with the supine position and procedures near the diaphragm, since the abdominal contents push up against the diaphragm. Rapid shallow breathing, ineffective cough, and retained secretions lead to decreased lung volumes, contributing to atelectasis and postoperative pneumonia.

The principal causes of hypoxemia in the PACU are hypoventilation and ventilation/perfusion mismatch, with increased right-to-left intrapulmonary shunt. Hypoventilation can be caused by opioids and benzodiazepines, depending on total dosage given and timing of administration, and owing to residual effects of the inhalational agents. Respiratory muscle weakness, evidenced by inadequate muscle relaxant antagonism, regional anesthesia, and diseases such as Guillain-Barré, myasthenia gravis, amyotrophic lateral sclerosis, and muscular dystrophy, can produce hypoventilation.

Increased right-to-left intrapulmonary shunting can be caused by atelectasis, pulmonary edema, aspiration, or pneumothorax and can be confirmed with chest radiographs. Pulmonary embolism (air, fat, foreign body, amniotic fluid) and myocardial infarction also are causes for postoperative hypoxemia. Diffusion impairment, seen with interstitial fibrosis, asbestosis, and sarcoidosis, is an uncommon cause of hypoxemia.

Implications

The ventilatory and circulatory responses to hypoxemia are summarized in Table 235–1. Severity of any metabolic acidosis correlates with the prolongation of hypoxemia. With severe hypoxemia, cardiovascular and respiratory depression occur, furthering hypotension, bradycardia, and decreased minute ventilation.

Table 235–1
Ventilatory and Circulatory Responses to Hypoxemia

Increased minute ventilation
Hypoxic pulmonary vasoconstriction
Pulmonary blood flow redistribution
Increased cardiac output
Increased systemic arterial pressure
Increased heart rate
Decreased systemic vascular resistance
Decreased tissue oxygen (O_2) delivery
Increased hemoglobin (Hb) concentration
Right shift Hb-O_2 dissociation curve

MANAGEMENT

Treatment of hypoxemia with oxygen usually improves PaO_2 when it is due to hypoventilation, ventilation/perfusion mismatch, or diffusion impairment. Maintenance of adequate PaO_2 can be provided by mask continuous positive airway pressure (CPAP) delivered via mask in the postoperative period, since CPAP restores lung volumes. However, if hyoxemia persists (PaO_2 less than 60 mm Hg) despite maximal oxygen therapy ($FIO_2 = 1.0$) or if there is poor compliance with mask CPAP, endotracheal intubation (possibly with positive end-expiratory pressure) is required. Mechanical ventilation with positive end-expiratory pressure increases FRC, permits a decrease in FIO_2, and improves arterial oxygenation.

PREVENTION

Supplemental oxygen should be administered to all patients recovering from regional or general anesthesia during transport to the PACU, as well as in the PACU. Postoperative hypoxemia secondary to diffusion impairment or hypoventilation can be prevented with supplemental oxygen. In addition, patients with ventilation/perfusion mismatch will respond at least partially to oxygen administration.

Because the supine position is associated with a decrease in FRC, patients—especially obese patients—should recover in the semi-sitting (Fowler's) position to increase FRC and decrease the pressure of abdominal contents on the diaphragm. Lung volumes also can be increased by chest physiotherapy to

assist in clearance of pulmonary secretions. Postoperative pain relief is essential to ensure maintenance of FRC and reduce the severity of hypoxemia, especially with upper abdominal or thoracic surgeries. Pain is associated with rapid shallow breathing. Adequate analgesia may be provided by parenteral opioids, injection of local anesthetic around the incision by the surgeon or continuous local anesthetic or opioid epidural techniques, or with other selected regional anesthetic techniques, depending on the surgical site. Last, use of incentive spirometry can help restore and maintain FRC.

Hypoxemia can be difficult to diagnose clinically. However, with the routine use of pulse oximetry in the PACU, hypoxemia can be detected early and confirmed with arterial blood gas measurements, with more prompt therapeutic and preventive intervention.

Further Reading

1. Barash P, Cullen B, Stoelting R: Clinical Anesthesia, ed 2. Philadelphia, JB Lippincott, 1992, pp 1529–1532.
2. Bennett J, Plum F (eds): Cecil Textbook of Medicine, ed 20. Philadelphia, WB Saunders Company, 1996.
3. Faust R: Anesthesiology Review, ed 2. New York, Churchill Livingstone, 1994, pp 24, 549–550.
4. Gravenstein N, Kirby R: Complications in Anesthesiology, ed 2. Philadelphia, Lippincott-Raven, 1996, pp 251–270.
5. Miller R: Anesthesia, ed 3. New York, Churchill Livingstone, 1990, pp 2118–2122.
6. Stedman's Medical Dictionary, 26th ed. Baltimore, Williams & Wilkins, 1995, pp 841–842.
7. Shapiro B, Peruzzi W, Templin R: Clinical Application of Blood Gases, ed 5. St Louis, Mosby–Year Book, 1994.
8. Yao F, Artusio J: Anesthesiology: Problem-Oriented Patient Management, ed 3. Philadelphia, JB Lippincott, 1993, pp 724–733.