



Hospital Peer Alert

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Hospital Peer Review is a monthly newsletter sponsored by the Rural Healthcare Quality Network to alert Critical Access Hospitals regarding findings from the Peer Review Program. Summarized are a few of the key findings and best practices that would be helpful for other critical access hospitals to be knowledgeable about. This newsletter is edited by Myron Bloom, Medical Director and he can be reached at drmbloom@msn.com.

Some Asthmatic and COPD patients try to Die after RSI, Why?

Immediate and severe life-threatening hypotension should be anticipated after endotracheal intubation for acute respiratory distress. When asthmatics and COPD patients are spontaneously breathing, airflow obstruction during exhalation delays lung emptying and the next breath may be initiated before exhalation is complete which is called dynamic hyperinflation. Besides increasing the work of breathing, it contributes to hypotension and risks alveolar over-distention and rupture. Hyperinflation also occurs as a result of excitement driven rapid vigorous ventilation during resuscitation after RSI.

Hyperinflation increases intrathoracic pressure and decreases venous return resulting in reduced cardiac output. Progressive cardiovascular collapse culminating in cardiac arrest due to pulseless electrical activity may follow. This hypotensive effect may be compounded by the use of sedatives and paralytics, which act as vasodilators and myocardial depressants. Successful rescue requires volume resuscitation and alleviation of the hyperinflation.

A rescue measure that should be considered if blood pressure fails to respond to volume resuscitation (and whenever ventilator alarms sound) is to temporarily disconnect the patient from the ventilator, manually exhale the patient (compress the chest), and then at first slowly (RR of 4-6) ventilate with 100% oxygen by ambu bag. This permits decompression of the lung and in turn a filling of the heart, while allowing the clinician to "feel" the mechanics of the respiratory system and still support oxygenation.

Hyperinflation can be minimized by decreasing the respiratory rate (functionally increasing expiratory time), increasing inspiratory flow rates (functionally decreasing the inspiratory time) and lowering the tidal volume. Expiratory time should be maximized to allow complete exhalation and prevent dynamic hyperinflation and intrinsic PEEP (elevation of alveolar pressure above atmospheric pressure at the end of

exhalation, also called auto-PEEP). Decreasing the respiratory rate and tidal volume may require the acceptance of elevated $p\text{CO}_2$, a strategy known as permissive hypercapnia.

[Respiratory drive is almost invariably increased in acute asthma, resulting in hyperventilation and a correspondingly decreased PaCO_2 . Thus, an elevated or even normal PaCO_2 indicates that airway narrowing is severe because in the absence of respiratory depressant medications such as narcotics or sedatives, hypercapnia occurs only when the peak expiratory flow falls below 25 percent of normal. Respiratory failure can then develop rapidly because of respiratory muscle fatigue or any further airway obstruction.]

Acute hypercapnia may produce a depressed level of consciousness, increases in cerebral blood flow and intracranial pressure, and depression of myocardial contractility. It shifts the oxyhemoglobin dissociation curve to the right, leading to an increased release of oxygen at the tissue level. Normal individuals do not exhibit a depressed level of consciousness until the PaCO_2 is greater than 60 to 70 mmHg. Patients with chronic hypercapnia may not develop symptoms until the PaCO_2 rises to greater than 90 or 100 mmHg as they have a compensatory increase in the plasma bicarbonate concentration and as a result, a larger elevation in PaCO_2 is required to produce the same degree of acidosis. Diminishing the risk of hyperinflation and barotrauma requires acceptance of PaCO_2 that is higher than normal and a pH that is lower than normal (but >7.25), a strategy that is called "permissive hypercapnia" or "controlled hypoventilation". Although hypercapnic acidosis can be associated with serious morbidity, a PaCO_2 near 70 mmHg and a pH near 7.25 can be safely tolerated as long as levels do not increase rapidly. After RSI, the clinician is controlling ventilation, so progressive sedation from CO_2 retention should not be a concern.

Upon intubation, FiO_2 should be set at 100% then decreased as tolerated to concentrations of 50% or lower to maintain oxygen saturation of 88-92%. The fraction of inspired oxygen (FiO_2) should be adjusted to achieve an arterial oxygen tension (PaO_2) just above 60 mmHg as higher levels increase the risk of oxygen toxicity without substantially increasing tissue oxygenation. With adequate circulation, ventilation and tissue perfusion can be monitored by pulse oximeter and venous blood gases which correlate well with arterial samples, with venous $p\text{CO}_2$ higher than arterial by 5-6 mmHg, venous pH lower by 0.03-0.06 and bicarb within 1 to 2 mEq/l). (Rang, L.C.F., et al, Can J Emerg Med 4(1):7, January 2002)

The ventilator inspiratory flow should be set at the highest rate the patient can tolerate without generating excessively high peak pressures; in adults, flow rates of 80 to 100 L/minute are recommended. The default inspiratory flow rate usually set at 60 L/minute is often inadequate. Large tidal volumes (eg, the usual 10 to 15 mL/kg) increase the risk of hyperinflation and barotrauma. Therefore, lower initial tidal volumes (eg, 5 to 7 mL/kg) with 100% FiO_2 are recommended during initial volume-assisted resuscitation. The respiratory rate should be set near or below physiologic rates (6 to 12 breaths per minute), keeping the minute ventilation under 115 mL/kg per min. The peak and plateau pressures should be noted and kept under 35 cm H_2O and 30 cm H_2O respectively to minimize hyperinflation and barotrauma.

Pneumothorax should be suspected if the patient abruptly becomes hypoxemic and/or hypotensive following intubation, especially if tracheal deviation, unilaterally decreased breath sounds, or high ventilatory resistance is found. Imaging should be performed expeditiously, or the patient may need emergent needle decompression or chest tube placement to relieve a presumptive pneumothorax.

Malpositioning of the endotracheal tube can be suspected from the depth of insertion. In the average adult, the incisors will be at the 18-24 cm mark when the tip of the tube is midway between the vocal cords and the carina. (Stone, DJ, Bogdonoff, DL, Anesth Analg 1992; 74:276) Unfortunately the tip of the tube follows the movement of the chin, moving up away from the carina with extension of the neck and deeper toward the carina with flexion of the neck.

If manual ventilations meet with minimal resistance, the clinician must verify that air is not escaping from the patient's nose or mouth because the ETT has migrated superiorly or that the cuff has deflated. If manual ventilations are difficult and do not improve the patient's condition, the patency of the ETT should be assessed using a suction catheter with failure to pass confirming obstruction of the ETT.

The cautious use of extrinsic PEEP in the ventilated asthmatic patient is controversial but it can be a helpful intervention particularly in patients with atelectasis by recruiting collapsed airways thereby reducing air trapping and improving ventilation. Some degree of PEEP (eg, 3 cm H₂O) may be necessary to compensate for the external resistance added to the respiratory tract by the endotracheal tube. PEEP may be increased in increments of 1-2 cm H₂O to determine whether additional PEEP is tolerated. Higher levels of PEEP (5 to 8 cm H₂O) may be beneficial for persistent and severe hypercarbia despite adequate tidal volumes (10 mL/kg) with expiratory times that permit complete exhalation. Most patients with asthma can tolerate and be successfully managed with PEEP in the range of 3 to 8 cm H₂O.

A Ventilator problem mnemonic -DOPE

- Dislodgment
- Obstruction
- Pneumothorax
- Equipment failure

How to make a simple IV Epinephrine drip:

- 2.5ml of 1:10,000 Epinephrine placed in 250cc NS = 1mcg/ml.
- 250cc given over 25 min is an infusion of 10mcg/ min.