Occult Hypercarbia*

An Unrecognized Phenomenon During Percutaneous Endoscopic Tracheostomy

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Bronchoscopy has been incorporated as a useful adjunct to increase the safety and effectiveness of percutaneous endoscopic tracheostomy (PET). Insertion of the bronchoscope, along with the intraluminal dilators of the PET set, into the airway potentially leads to hypoventilation and hypercarbia during the procedure. Using continuous in-line arterial blood gas monitoring, we documented profound hypercarbia in two patients undergoing PET in the surgical ICU. In a third patient, the rise in PaCO$_2$ was accompanied by a marked rise in intracranial pressure (ICP), and a corresponding fall in cerebral perfusion pressure. While transient hypercarbia seems well tolerated by most patients, this phenomenon and its effect on cerebral blood flow should be strongly considered before performing PET on the critically ill patient with evidence of elevated ICP.

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CPP=cerebral perfusion pressure; ICP=intracranial pressure; MAP=mean arterial blood pressure; PET=percutaneous endoscopic tracheostomy

Key words: hypercarbia; percutaneous tracheostomy; tracheostomy

Tracheostomy is one of the most commonly performed surgical procedures in the critical care setting. The early use of tracheostomy as a method of primary airway management has been proposed as a means to decrease pulmonary morbidity and to shorten ventilator, ICU, and hospital days.1 Percutaneous dilatational tracheostomy has been introduced as an alternative to standard operative tracheostomy.2,5 Percutaneous tracheostomy has been shown to be safe, with intraprocedural complication rates similar to those seen with standard tracheostomy.6,7 The addition of endoscopic guidance has further increased the safety of this procedure and may prevent such complications as pneumothorax, subcutaneous emphysema, and paratracheal false passage previously reported with percutaneous tracheostomy when performed without endoscopic guidance.8-11 The use of bronchoscopic guidance to confirm guidewire placement, coupled with the use of intraluminal dilators to enlarge the tracheostomy, potentially leads to hypercarbia due to hypoventilation, and subsequent respiratory acidosis. We hypothesize that this phenomenon, not described in previous studies incorporating this procedure, occurs unrecognized during percutaneous endoscopic tracheostomy (PET).

METHODS AND MATERIALS

Percutaneous endoscopic tracheostomy was performed at the bedside in two patients requiring mechanical support for respiratory failure. In both patients, radial artery catheters with continuous in-line arterial blood gas monitoring capabilities (prototype manufactured by Abbott Critical Care Systems, Mountain View, Calif) were placed.12 Continuous arterial pH, PaCO$_2$, and PaO$_2$ tracings were obtained on each patient before, during, and after the procedure. A third patient with a traumatic head injury was similarly studied using standard radial artery pressure monitoring as well as a Camino intracranial pressure monitor (Camino Labs, San Diego). Arterial blood gas values were determined before the start of the tracheostomy and intermittently throughout the procedure. Simultaneous measurements of mean arterial blood pressure (MAP) and intracranial pressure (ICP) were recorded. Cerebral perfusion pressure (CPP) was calculated by the formula:

$$\text{CPP} = \text{MAP} - \text{ICP}$$

Percutaneous endoscopic tracheostomy with bronchoscopy was performed as previously described.8 In all cases, this ICU bedside procedure was performed by surgeons experienced with the procedure. Briefly, patients were maintained on volume-cycled ventilation with an inspired oxygen fraction of 1.0 throughout the procedure. Other ventilator settings were not altered for the procedure. Respiratory care personnel stood by to monitor the ventilator and any ventilator alarms which occurred during the procedure. Intravenously administered narcotics and benzodiazepines, as well as paralytics, were used for sedation as needed. The patients were positioned with the neck extended. A flexible fiberoptic adult bronchoscope was introduced via a side arm adaptor of the endotracheal tube. The endotracheal tube was then withdrawn such that the tip remained below the level of the vocal cords. After transillumination and palpation of the cricothyroid membrane and trachea, a small incision was made in the skin over the second tracheal ring. The platysma muscle was bluntly divided and a needle catheter (Ciaglia Percutaneous Introducer Set, Cook Critical Care, Bloomington, Ind) was inserted below the second tracheal ring into the tracheal lumen. Placement was confirmed both endoscopically as well as by the aspiration of air through the needle catheter. A guidewire was then passed through the catheter into the tracheal lumen. A Teflon guiding catheter was placed over the guidewire, and successive dilators were then used to progressively dilate the tracheostomy up to 32F. Each dilator was passed three times prior to successive progression in caliber. The 32F size allowed placement of a No. 8 tracheostomy tube.
tube in the trachea. Throughout the procedure, the bronchoscope was intermittently used to confirm guidewire, dilator, and ultimately tracheostomy position. After successful placement of the tracheostomy, the guidewire, Teflon guiding catheter, and dilator were removed, and placement was confirmed by visualization of the tracheal carina during bronchoscopy via the new tracheostomy. The tracheostomy was then sewn into place. A chest radiograph was then performed to confirm tracheostomy tube placement and to rule out pneumothorax.

RESULTS

All procedures were completed in approximately 30 min. There were no intraprocedural complications (eg, significant hypoxia, bleeding, or hypotension) in these three patients. In addition, no postprocedural complications (eg, subcutaneous emphysema or pneumothorax) were evident on subsequent chest x-ray films.

Continuous tracings of PaCO₂, PaO₂, and arterial pH of patient 1 are presented in Figure 1. The PaCO₂ level rose promptly at the beginning of the procedure from near normal baselines to a maximum of 78 mm Hg. The PaCO₂ level remained elevated throughout the entire procedure, with minimal decreases occurring during periods between separate passage of each dilator and repeated visualization using the bronchoscope. The PaCO₂ returned to baseline only as ventilation was normalized after the completion of the percutaneous tracheostomy. The PaO₂ was maintained at satisfactory levels throughout the procedure. Neither bronchoscopy nor tracheal dilatation resulted in significant hypoxia. Arterial pH dropped from 7.36 to a nadir of 7.10 during the percutaneous tracheostomy. This respiratory acidosis was progressive and sustained throughout the procedure. Variation in pH during alternate passages of dilators and the bronchoscope was not seen. The acidosis improved with normalization of ventilation at the completion of the procedure. Tracings of PaCO₂, PaO₂, and arterial pH for patient 2 were similar to those seen in patient 1. The PaCO₂ rose from 42 to 72 mm Hg, and arterial pH fell from 7.42 to 7.20 during percutaneous tracheostomy. The PaO₂ remained relatively constant throughout the procedure.

The results of the intermittent monitoring of PaCO₂ in patient 3 are presented in Figure 2. The PaCO₂ rose from a baseline of 27 to 43 mm Hg during the procedure, which lasted only 8 min. Despite this brief procedure time, ICP rose from a baseline of 10 to 48 mm Hg, with CPP falling from 65 to 38 mm Hg (Fig 2).

DISCUSSION

The preferred artificial airway for initial use in the mechanically ventilated patient is the translaryngeal endotracheal tube. Replacement of the translaryngeal endotracheal tube with a tracheostomy provides a more secure airway for long-term care, facilitates tracheobronchial suctioning, and improves patient comfort. In addition, the early use of tracheostomy also may decrease pulmonary morbidity and shorten ventilator, ICU, and hospital days. As a result, tracheostomy has become a commonly performed procedure for the management of critically ill or injured patients with respiratory insufficiency, prolonged coma, or neuromuscular paralysis because of spinal cord injury.

Standard operative tracheostomy is the gold standard by which other methods of obtaining a surgical airway must be compared. Recent data suggest that the mortality and morbidity rates associated with operative tracheostomy are
as low as 0 and 4%, respectively. Still, this procedure usually requires between 20 and 40 min of operating room time to perform with subsequent hospital charges of nearly $5000.10

Percutaneous dilatational tracheostomy has been introduced as an alternative to standard operative tracheostomy.2-5 This procedure has been found to have a safety profile comparable to that of operative tracheostomy.4,6,10 Long-term follow-up of patients undergoing this procedure has failed to reveal any significant sequelae.14 In addition, percutaneous dilatational tracheostomy generally is recognized as a faster procedure.2-5 The procedure can be safely and rapidly performed at the bedside in the ICU.5,10 As a result, the charges for percutaneous dilatational tracheostomy may be as much as $3,400 less per patient when compared with standard operative tracheostomy in the operating room.10

The addition of endoscopic guidance to percutaneous tracheostomy has further increased the safety of this procedure.5-11 Complications such as pneumothorax, paratracheal false passage of dilators or the tracheostomy tube, and perforation of the posterior wall of the trachea, all previously reported with blind percutaneous methods, are largely prevented with bronchoscopic visualization of the tracheal cannulation.5,8-11 However, the insertion of a bronchoscope into an airway already compromised by intraluminal dilators results in further obstruction of the ventilatory path, worsening the hypventilation. This iatrogenic hypventilation results in occult hypercarbia.

The patients described in this report underwent uneventful PET. No intra-procedural complications occurred. Posttracheostomy chest x-ray films demonstrated proper placement of each tracheostomy tube. However, all three patients became profoundly hypercarbic during the procedure. As a result, the patients also developed a significant respiratory acidosis. This occult hypercarbia and respiratory acidosis persisted throughout the duration of the procedure and only slowly resolved with the resumption of normal ventilation at the conclusion of the procedure.

The specific etiology of hypercarbia during percutaneous endoscopic tracheostomy cannot be determined from this report. Ventilator/patient dysynchrony may result in hypventilation. However, dysynchrony did not appear to occur during the performance of these procedures. The addition of paralysis, which would eliminate ventilator/patient dysynchrony, has been noted to have no effect on the profound hypercarbia which develops during PET. While the introduction of a bronchoscope into the airway may well limit ventilation, this complication of bronchoscopy in the ICU is not well-recognized.15 Still, hypercarbia during bronchoscopy has been reported.16 The presence of dilators in the airway, and the presence of an uncontrolled tracheostomy during the procedure, also may play a role in the development of hypercarbia. Since significant hypercarbia developed, even during a procedure lasting only 10 min, the length of time needed to safely perform PET does not appear to be responsible for the hypercarbia which was noted. Still, minimizing the length of the procedure may be beneficial to these critically ill patients. To this end, we recommend that experienced personnel perform this procedure in the ICU, with bronchoscopic time minimized when possible. The physiologic effects of hypercarbia are complex.17 While hypercarbia may cause extreme hypoxia when breathing room air, the effects of increased PaCO2 on PaO2 are insignificant when breathing a higher inspired fraction of oxygen.18,19 A greater effect on oxygenation may result from the rightward shift of the oxyhemoglobin dissociation curve caused by respiratory acidosis. This effect may facilitate oxygen unloading to the tissues and subsequently improve oxygen delivery at the cellular level.

Acute hypercarbia also affects the central nervous system in a number of ways. Acute hypercarbia (PaCO2, 90 to 100 mm Hg) may lead to narcosis. Higher PaCO2 levels may result in convulsion. These central nervous system findings may correlate better with cerebrospinal fluid pH rather than arterial pH or PaCO2.20 Perhaps more importantly, cerebrospinal fluid pH correlates with cerebral blood flow. Acutely elevated levels of PaCO2 may lead to markedly increased cerebral blood flow and a corresponding increase in ICP, as demonstrated in patient 3. In the patient with head injury, therapy is often directed at minimizing ICP by hyperventilation to a PaCO2 of 25 to 30 mm Hg. In this subgroup of patients, the occult hypercarbia which occurs during PET may have deleterious effects. Acute hypercarbia also results in increased norepinephrine release from sympathetic nerve endings and increased norepinephrine and epinephrine release from the adrenal medulla.21 Dysrhythmias are common during hypercarbia, probably as a result of this increase in circulating catecholamine concentrations.17 At the cardiovascular level, this increased sympathetic activity is counterbalanced by a direct myocardial depressant effect of acidosis. Pulmonary vascular resistance also is increased as a result of acidosis.22,23

Iatrogenic acute hypercarbia during percutaneous endoscopic tracheostomy may result in many complex physiologic derangements, some of which may be detrimental. The CO2 diffuses rapidly into cells, and most of the effects of acute hypercarbia appear to be due to the resulting intracellular acidosis. Occult hypercarbia during PET may therefore result in unsuspected intracellular acidosis. Surgeons performing this procedure should be aware of this unrecognized phenomenon. Monitoring end-tidal CO2 may be warranted during the procedure, but the loss of exhaled gases through the tracheostomy or via suctioning through the bronchoscope may invalidate this technique. Steps to minimize occult hypercarbia, such as using the smallest bronchoscope available, minimizing suctioning during bronchoscopy, and minimizing the length of time the bronchoscope is present in the endotracheal tube should all be taken during PET.

SUMMARY

Marked elevations in PaCO2 were demonstrated in three patients undergoing uneventful percutaneous tracheostomy utilizing bronchoscopy. Although all three patients tolerated the procedure without incident, a potential exists for significant morbidity secondary to the detrimental physiologic effects of intracellular acidosis. Surgeons per-
References


Fluoxetine Hydrochloride (Prozac)-Induced Pulmonary Disease*

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We describe a patient who developed progressive dyspnea, lung infiltrates, and restrictive lung disease in association with the antidepressant fluoxetine hydrochloride (Prozac). The pathologic findings were consistent with hypersensitivity pneumonitis. An associated pulmonary phospholipidosis was also noted.

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Key words: drug-induced lung disease; fluoxetine hydrochloride (Prozac); hypersensitivity pneumonitis; phospholipidosis

Fluoxetine hydrochloride (Prozac; Distal Products; Indianapolis) is a widely used antidepressant. Preclinical studies have shown that this amphiphilic cationic drug can induce pulmonary and/or systemic phospholipidosis in animals, but to our knowledge, this phenomenon has not been reported in humans. Other amphiphilic cationic drugs, such as amiodarone, can induce pulmonary alveolitis and phospholipidosis in humans and in animals. We describe the development of hypersensitivity pneumonitis in a patient receiving fluoxetine hydrochloride and demonstrate evidence for phospholipidosis.

Case Report

A 62-year-old woman with manic-depressive illness presented to our outpatient center with dyspnea and nonproductive cough. One and a half years earlier, she had been hospitalized for subacute onset of cough, dyspnea, and fevers. She had been taking fluoxetine hydrochloride (20 to 60 mg daily) for 4 months. On the presumption she had acute bronchitis, she was treated with bronchodilators and antibiotics, with slow symptom resolution. While in the hospital, the fluoxetine hydrochloride therapy was discontinued. In the interim 10 months, after other antidepressants failed to control her depression, treatment with these was discontinued and fluoxetine hydrochloride therapy was eventually restarted at 20 mg daily. Within 5 days of restarting the fluoxetine hydrochloride therapy, she developed progressive dyspnea. She denied chest pain, orthopnea, peripheral edema, or fever. Suspecting that the fluoxetine hydrochloride may have triggered her condition, she discontinued treatment with it.

Four weeks later, she presented to us with unrelenting dry cough, but subjectively less dyspnea. A nonsmoker, she denied any other respiratory or cardiac symptoms, fever, arthralgias, skin tightness or eruptions, Raynaud’s phenomenon, or dysphagia. She

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