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Hypercarbia During Tracheostomy in the Head-Injured Patient: Compariscon of Percutaneous Endoscopic, Percutaneous Doppler and Standard Tracheostomy

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Citation

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Abstract

Purpose: Bronchoscopy during percutaneous tracheostomy may cause a significant increase in pCO2 due to hypoventilation. The clinical relevance of this hypercarbia is unclear. We examined the effects of procedure-induced hypercarbia on cerebral perfusion during percutaneous endoscopic (PET), percutaneous doppler (PDT), and standard surgical tracheostomy (ST).

Methods: Three patients with indwelling radial artery catheters and intracranial pressure monitors underwent PET, PDT, or ST in the Intensive Care Unit. Intermittent arterial blood gases were obtained throughout each procedure. Simultaneous measurements of mean arterial pressure (MAP), intracranial pressure (ICP), and cerebral perfusion pressure (CPP=MAP-ICP) were recorded.

Results: All tracheostomies were successfully performed with no technical complications. No episodes of hypoxia occurred during the procedures.

Conclusions: Bronchoscopy during percutaneous endoscopic tracheostomy leads to hypoventilation, hypercarbia, and respiratory acidosis. In the head-injured patient, this hypercarbia does result in a marked increase in ICP and a related decrease in CPP to ischemic ranges. Tracheostomy (either standard surgical or percutaneous using the doppler ultrasound to position the endotracheal tube) can be safely performed in the head injured patient without adversely affecting cerebral perfusion. The potential for hypoventilation should be considered when choosing the method of tracheostomy in the head-injured patient where hypercarbia may be detrimental to cerebral hemodynamics.

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INTRODUCTION

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, intensive care unit, and hospital days.[1] The use of tracheostomy in the head-injured patient is controversial. While a tracheostomy, once in place, may minimize agitation (and associated increases in intracranial pressure) in the head-injured patient, the procedure itself may result in the very alterations in cerebral hemodynamics one is trying to avoid.

Percutaneous dilatational tracheostomy, with the positioning of the endotracheal tube performed blindly or by the Doppler method, has been introduced as an alternative to standard operative tracheostomy.[2,3,4,5,6] The percutaneous technique has been shown to be safe, with intraprocedural complication rates similar to those seen with standard surgical tracheostomy.[7,8] The addition of endoscopic guidance (fiberoptic bronchoscopy) 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.[9,10,11,12] However, the use of bronchoscopic guidance to confirm guidewire placement, coupled with the use of intraluminal dilators to enlarge the tracheotomy, may lead to hypercarbia due to hypoventilation, and subsequent respiratory acidosis. In an earlier report, we demonstrated significant hypercarbia does occur during percutaneous endoscopic tracheostomy.[13] This hypercarbia is minimized with either percutaneous doppler tracheostomy or standard surgical tracheostomy. The purpose of this report is to examine the potential consequences of this hypercarbia in relation to cerebral hemodynamics and perfusion in the head-injured patient.

MATERIALS AND METHODS

This study was approved by the University of Pennsylvania Institutional Review Board Committee on Studies Involving Human Beings. Informed consent was obtained from patients and/or family members before each procedure.

Three patients in the Surgical Intensive Care Unit (SICU) requiring tracheostomy underwent percutaneous endoscopic tracheostomy, percutaneous Doppler tracheostomy, or surgical tracheostomy. The method of tracheostomy performed was left to individual surgeon preference (nonrandomized). All procedures were performed in the SICU, except in cases where additional procedures (e.g. jejunostomy tube placement) were simultaneously being performed. All patients had in place an indwelling radial artery catheters and intracranial pressure monitors. Arterial blood gases were drawn before the start of the tracheostomy, and serially (approximately every four minutes) throughout the procedure. In addition, simultaneous measurements of mean arterial pressure (MAP), intracranial pressure (ICP) and cerebral perfusion pressure (CPP=MAP-ICP) were recorded. The person obtaining the blood gas specimen and recording cerebral hemodynamics was blinded to the procedure as it progressed. Specimens were drawn and pressures and times recorded without directly observation of the procedure. In all three cases, the procedure (tracheostomy and/or bronchoscopy) was performed by surgeons or surgical residents experienced with the procedure.

Percutaneous endoscopic tracheostomy with bronchoscopy was performed as previously described.9 The patient had in place an 8.0 endotracheal tube. Briefly, the patient was maintained on volume-cycled ventilation with an inspired oxygen fraction of 1.0 throughout the procedure. Minute volume was set at previous ventilator settings using an assist-control mode of ventilation and maintained throughout the procedure. Ventilator alarms and settings were not adjusted during the procedure. Intravenous narcotics (morphine) and benzodiazepines (midazolam) were used for sedation and confirmed neuromuscular blockade (vecuronium) achieved before initiation of the procedure. The patient was positioned with the neck extended. A flexible fiberoptic adult bronchoscope (5.8mm OD) was introduced via a side arm adapter 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, IN) 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 tracheotomy up to 36 Fr. Each dilator was passed three times prior to progression to the next larger size. The 36 Fr. size allowed placement of a No. 8 tracheostomy 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 sutures into place. A chest x-ray was then performed to confirm tracheostomy tube placement and to exclude pneumothorax.

Percutaneous doppler tracheostomy was performed in a manner similar to that described above. However, the bronchoscope was not used. Rather, the endotracheal tube was positioned just below the level of the vocal cords with the aid of a sterile doppler device.6 Briefly, a small incision was made in the skin over the second tracheal ring, and the doppler probe placed over the trachea. The endotracheal tube was then slowly withdrawn. As the tip of the tube reached the second tracheal ring, the intensity of the doppler signal increased greatly due to an increased signal from unencumbered, turbulent air. The endotracheal tube was then resecured, and the trachea entered with the needle catheter, as confirmed by the aspiration of air through the needle. The percutaneous tracheostomy was then performed as described above.

Standard surgical tracheostomy was performed as previously described.[14] Intravenous sedation, narcotics, and neuromuscular blockade were used for standard surgical tracheostomy in a fashion identical to the anesthesia provided to those patients undergoing percutaneous tracheostomy. Of note, ventilation proceeded throughout the procedure until the endotracheal tube was withdrawn under direct vision through the tracheotomy, and the tracheostomy tube was placed.

The results of the blood gas measurements are presented as changes in PaCO2 and arterial pH from the baseline measurements recorded before the procedure began. Maximal change in PaCO2 and arterial pH each represent the single value of maximal change from the baseline for each individual procedure. Both maximal absolute value and change from baseline were recorded for intracranial pressure. Minimal absolute value, and maximal change from baseline were recorded for cerebral perfusion pressure.

RESULTS

No significant intraprocedural complications (e.g., hypoxia, bleeding, or hypotension) were noted. The length of each procedure is listed in Table 1, and appeared similar. No postprocedural complications (e.g., subcutaneous emphysema or pneumothorax) were evident on subsequent chest x-ray.

The baseline PaCO2 did not significantly differ among the three groups. No patient was hyperventilated as a baseline. The maximum changes in PaCO2 and arterial pH are presented in Table 1. The maximum increase in PaCO2 during percutaneous endoscopic tracheostomy was markedly greater than that increase seen during either percutaneous doppler or standard surgical tracheostomy. The maximum increase in PaCO2 during percutaneous doppler tracheostomy and that increase seen during standard tracheostomy appeared similar. The maximal decrease in arterial pH during percutaneous endoscopic tracheostomy mirrored the reciprocal increase in PaCO2 during the procedure. As a result, the maximal respiratory acidosis noted was markedly greater during percutaneous endoscopic tracheostomy than the maximal acidosis seen during either percutaneous doppler or standard surgical tracheostomy.

Both maximal absolute value and change from baseline for intracranial pressure are presented in Table 2. The maximal change in intracranial pressure was much greater during percutaneous endoscopic tracheostomy when compared to the minimal change seen during standard surgical tracheostomy and the modest change seen during percutaneous doppler tracheostomy. In addition, the maximal intracranial cranial pressure was markedly elevated during percutaneous endoscopic tracheostomy to a level considered unacceptable. The maximal intracranial pressure seen during standard tracheostomy and percutaneous doppler tracheostomy fell well within acceptable limits.

Minimal absolute value, and maximal (negative) change from baseline for cerebral perfusion pressure are presented in Table 3. The marked increase in intracranial pressure noted during percutaneous endoscopic tracheostomy resulted in significant decrease of cerebral perfusion pressure into ischemic ranges. In contrast, cerebral perfusion pressure was maintained during both standard durgical tracheostomy and percutaneous doppler tracheostomy, despite the increases seen in intracranial pressure during these procedures.

DISCUSSION

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.4,7,11 The addition of endoscopic guidance to percutaneous tracheostomy has further increased the safety of this procedure.9-12 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.9-12 However, the insertion of a bronchoscope into an airway already compromised by intraluminal dilators results in further obstruction of the ventilatory path, worsening the hypoventilation. This iatrogenic hypoventilation results in occult hypercarbia.13

The use of a doppler with the percutaneous tracheostomy is another option to facilitate the percutaneous tracheostomy.6 The doppler probe is used for the guidance of endotracheal tube positioning to decrease the incidence of inadvertent extubation during the procedure and also to document that the endotracheal tube has been sufficiently withdrawn prior to dilator placement. To date, no studies evaluating the safety of percutaneous doppler tracheostomy in comparison to that of standard percutaneous tracheostomy or percutaneous endoscopic tracheostomy have been reported.

The patients described in this report all underwent uneventful tracheostomy. No intraprocedural complications occurred. Procedure times for all three techniques were not significantly different. Post-tracheostomy chest x-rays demonstrated proper placement of each tracheostomy tube. However, the patient undergoing percutaneous endoscopic tracheostomy became profoundly hypercarbic during the procedure. As a result, the patient also developed a respiratory acidosis. The impact of this hypercarbia on cerebral hemodynamics was profound, but not unexpected. Intracranial pressure markedly rose. Despite a modest increase in mean arterial pressure during the procedure, cerebral perfusion pressure fell dramatically into ischemic ranges unacceptable to our Surgical Critical Care and Neurosurgical services.

PaCO2 also rose during percutaneous doppler tracheostomy and standard surgical tracheostomy. However, both the maximum change in PaCO2 and accompanying fall in arterial pH were markedly less than that seen during percutaneous endoscopic tracheostomy. As a result, the rise in intracranial pressure, and subsequent change in cerebral perfusion pressure was mitigated during these two procedures. In both instances, cerebral perfusion pressure was maintained well within acceptable ranges.

The specific etiology of hypercarbia during percutaneous tracheostomy may be multifactorial. Ventilator/patient dyssynchrony may result in hypoventilation. However, the use of neuromuscular blockade eliminates ventilator/patient dyssynchrony. The presence of dilators in the tracheal lumen may also contribute to the development of hypercarbia. The temporary occlusion (partial or complete) by the dilators (often manifest by the pressure-limit alarm on the ventilator) may explain the small increase in PaCO2 noted during percutaneous doppler tracheostomy.

The presence of an open, uncontrolled tracheotomy during the procedure may also contribute to hypoventilation and hypercarbia. Air may escape through this stoma while dilators are exchanged between successive passages. This potentially may result in the loss of tidal volumes, essentially a tracheo-cutaneous fistula. In addition, the loss of exhaled volumes through this stoma may make the analysis and tracking of minute ventilation meaningless.

Although the exact length of “bronchoscopic time” for each procedure was not recorded, the use of bronchoscopic guidance during percutaneous endoscopic tracheostomy appears to be the single most important factor responsible for the hypercarbia which develops during the procedure. When the bronchoscope is omitted from the procedure (i.e. percutaneous doppler tracheostomy), hypercarbia is minimized and the resultant respiratory acidosis significantly attenuated. While the introduction of a bronchoscope into the airway may well limit ventilation, again often manifest by the pressure-limit alarm on the ventilator, this complication of bronchoscopy in the intensive care unit is not well recognized15 , though hypercarbia during bronchoscopy has been reported.[16] The potential benefits of endoscopic guidance during percutaneous tracheostomy (increased safety of the procedure) must be weighed against the hypercarbia and subsequent respiratory acidosis which develop. We currently minimize bronchoscopic time (when possible) during the performance of this procedure. Furthermore, with increased experience and confidence with this procedure we feel this technique can be safely performed without bronchoscopic assistance.

Monitoring end-tidal CO2 or expired minute volumes may be warranted during the procedure, but the loss of exhaled gases through the tracheotomy or via suctioning through the bronchoscope may invalidate this technique.[17] Continuous in-line arterial blood gas monitoring would be optimal, but is not routinely available and may also not be cost effective.

A limitation of this report is the small number of patients enrolled. We previously have documented, in a much larger series, the development of significant hypercarbia during percutaneous endoscopic tracheostomy.13 However, we had failed to demonstrate the presumed implication of this work - that the hypercarbia which develops during percutaneous endoscopic tracheostomy may lead to adverse cerebral hemodynamics in the head-injured patients. The findings of this current report did not surprise us. However, seeing the dramatic rise in intracranial pressure, and consequent fall in cerebral perfusion pressure to ischemic ranges in a single patient undergoing percutaneous endoscopic tracheostomy has persuaded us to discontinue this study. We do not feel it is ethical to have additional head-injured patients undergo percutaneous endoscopic tracheostomy when we have previously demonstrated the hypercarbia which develops, and know its adverse effects on cerebral hemodynamics.

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 when performing percutaneous endoscopic tracheostomy. Other techniques of tracheostomy are more appropriate in the head-injured patient where hypercarbia and respiratory acidosis may be deleterious to cerebral hemodynamics.

{image:1}

{image:2}

{image:3}

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