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Bronchoscopy in ICU

A Practical Guide



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CHAPTER 3

Pathophysiological Changes during Bronchoscopy in ICU

Manoj K Goel, Jagdish C Suri

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INTRODUCTION

Patients in the intensive care unit (ICU) are critically ill. Their respiratory insufficiency or failure is often accompanied by dysfunction or failure of other organ systems, e.g. cardiogenic or neurogenic pulmonary edema, liver failure, major surgery, or trauma. The vast majority of the ICU patients receive some form of mechanical ventilation either by noninvasive bilevel positive airway pressure (NIV) or via an endotracheal tube (ETT) or tracheostomy tube (T tube). Bronchoscopy is an invasive procedure that may cause or trigger a series of pathophysiologic consequences on the respiratory mechanics, gas exchange, and hemodynamics. Knowledge of these undesirable responses summarized in this article is essential to keep the complication rate of bronchoscopy low in critically ill patients.

SIZE OF BRONCHOSCOPE AND AIRWAY

In a nonintubated patient, a 5.7 mm outside diameter bronchoscope occupies only 10% of the total cross-sectional area of the trachea,[1-3] as a result, endotracheal pressures generated are similar to those in patients without bronchoscopy which are –5 cm H_2O during inspiration and +3.5 cm H_2O during expiration.[4] On the other hand, bronchoscope through an ETT or tracheostomy tube generates pressures of –10 to +9 cm H_2O during inspiration and expiration, respectively. Therefore, the bronchoscope diameter and its relation with the airway diameter do play an important role in causing airflow obstruction in intubated patients.[1] The increase in airway pressures during bronchoscopy is related to the internal diameter of the trachea and outer diameter (OD) of bronchoscope.[4] A bronchoscope with OD of 5.7 mm occupies 40% of cross-sectional area of 9 mm ETT whereas 51% and 66% of cross-sectional area of 8 mm and 7 mm ETT, respectively,[5] signifying that smaller the diameter of ETT, more is obstruction to airflow during bronchoscopy. This not only compromises the ability to ventilate the patient, but it makes the procedure considerably more difficult for the bronchoscopist, because the narrow artificial airway decreases the maneuverability of the bronchoscope. In practice, one needs a substantially larger ETT in order to be able to advance the bronchoscope without difficulty while maintaining adequate ventilation.[6]

If a standard flexible fiberoptic bronchoscopy (FFB) of 5-mm OD is used and if 40 mm^2 is the minimum acceptable cross-sectional airway area that should remain free,[2] the ETT size should be at least 8 mm to maintain an adequate minute ventilation as well as to minimize risk of barotrauma.[1-3] It is recommended to use an ETT of an internal diameter at least 2 mm larger than the OD of the bronchoscope.[7] The choice of using a narrow bronchoscope has a potential disadvantage of lesser aspiration capacity as compared with those of a larger size.[1]

EFFECTS ON LUNG MECHANICS

Klein et al.[8] studied the changes produced after bronchoalveolar lavage (BAL) in 18 mechanically ventilated patients. It was observed that there was a transitory increase in pulmonary resistance and a decrease of more than 20% of pulmonary compliance 8 minutes after the procedure. 3 hours later, the compliance continued to be lower than the values at the beginning, but there was a trend toward reaching the initial values. Similar findings were reported in another study,[5] even though the compliance changes and pulmonary resistance values showed a tendency of reaching pre-BAL values 90 minutes after the procedure.

AIRWAY RESISTANCE

Since the resistance to airflow is inversely proportional to the fourth power of the radius of the tube, the obstruction of the airway by the bronchoscope causes a marked increase in the Raw. In an intubated and mechanically ventilated patient, the increase in Raw will affect both the peak inspiratory pressure (PIP) and the positive end-expiratory pressure (PEEP) as well as the tidal volume (Vt) that is delivered to the patient.

Peak Inspiratory Pressure and Positive End-expiratory Pressure

When the patient is ventilated in a volume control (VC) mode, the increase in Raw during bronchoscopy will result in increases in the PIP and the PEEP. These pressure changes increase the risk for barotrauma, auto-PEEP and significant hemodynamic instability. On the other hand, pressure control (PC) ventilation has little effect on pressures, but it may severely limit the delivered Vt. Lindholm et al.[3] described the cardiorespiratory effects of fiberoptic bronchoscopy (FOB) in 38 patients. During the procedure, peak tracheal pressures varied between 18 cm H_2O and 60 cm H_2O, while PIP varied between 28 cm H_2O and 80 cm H_2O. Even though PEEP was discontinued during the procedure, auto-PEEP averaging 10.4 cm H_2O was present in all but five patients immediately on insertion of the bronchoscope. The auto-PEEP occurs during bronchoscopy due to impedance to expiratory flow and insufficient time for complete expiration.[1-3] Usually PEEP remains below 20 cm H_2O in an 8 mm ETT. A PEEP of up to 35 cm H_2O has been recorded with a 7 mm ETT.[1,3] Decreasing ETT size is associated with increasing levels of auto-PEEP.[7]

Tidal Volume

The reduction in delivered Vt is a direct result of obstruction of the airway by the bronchoscope and, if the high-pressure limit setting is reached, the ventilator prematurely cycles from the inspiratory to the expiratory phase, resulting in a further decrease in delivered Vt.[7] Suction applied through the bronchoscope also decreases the delivered Vt.[2,3] As much as 200–300 mL of the Vt can be removed during each suction period.[9] In addition, some of the delivered volume can be lost through the swivel adaptor of the ETT. Although Vt falls dramatically in all cases during bronchoscopy, but returns to prebronchoscopic values immediately on its removal, especially in VC mode.[3]

In the PC mode, the presence of the bronchoscope in the airway results in a reduction in delivered Vt similar to that observed in the VC mode when the high-pressure limit is not readjusted. A common practice among bronchoscopists is to transiently withdraw the bronchoscope for brief periods to allow the patient unobstructed ventilation. This would expose the lung to risk of very high Vts, and thus the potential for barotrauma. It is, therefore, important that the inspiratory pressure level be reduced to prebronchoscopic values before the bronchoscope is removed from the ETT.

Plateau Pressure

An experimental study on lung models showed that, despite PIPs that averaged 82 cm H_2O at the proximal airway in the conventional mechanical ventilation (CMV) mode, the plateau pressures remain low. Measured within the lung model, Pulmonary alveolar pressure (Palv) averaged 11 cm H_2O at a compliance of 100 mL/cm H_2O, and 17 cm H_2O at a compliance of 50 cm H_2O,

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compared to an average Palv of 8 cm H_2O prior to insertion of the bronchoscope.[7] The authors concluded that the increased resistance offered by the presence of the bronchoscope in the airway protected the alveolar space from the excessively high PIPs.

LUNG COMPLIANCE

Changes in lung compliance occur primarily because of atelectasis that may develop as a result of suctioning that may lead to alveolar collapse; and/or because of the instillation of normal saline during BAL that washes out surfactant. Application of endotracheal suctioning may significantly decrease the dynamic lung compliance, whereas the instillation of normal saline during BAL will decrease the static lung compliance.[6] A study[5] showed a significant drop in pulmonary compliance in 54% of the patients. This decrease in compliance could be more than 20%.[5,8] Considering that patients in the ICU tend to have conditions associated with very low lung compliance, e.g. acute respiratory distress syndrome, pneumonia, atelectasis, an additional decrease may have disproportionately severe effects.

FUNCTIONAL RESIDUAL CAPACITY

Insertion of a bronchoscope in intubated patients induces a 30% increase in the functional residual capacity (FRC) due to air trapping as well as a 40% decrease in forced expiratory volume (FEV_1) due to decrease in Vt.[1] Another study reported reduction in FRC due to decrease in Vt and suctioning.

EFFECTS ON GAS EXCHANGE

Transient deterioration of gas exchange due to partial obstruction of the airway is one of the most common problems encountered during bronchoscopy. This is largely due to reductions in lung volumes and FRC, which may produce alveolar closure during FFB. Another mechanism is reflex bronchospasm induced by stimulation of subepithelial vagal receptors in the upper airway.[2,9] Utilization of short-suction periods of no more than 3 seconds and adequate topical anesthesia should minimize the hypoxic response to these two stimuli.[9] Postprocedure, these gas exchange abnormalities return to baseline values, from about 15 minutes to several hours depending upon the severity of parenchymal disease.[3]

HYPOXEMIA

Hypoxemia is probably the single most common gas exchange abnormality.[5,10,11] Trouillet et al.[12] observed in 107 patients a PaO_2 decline of 26% after the procedure. A study[3] reported that bronchoscopy causes moderate decrease in PaO_2 averaging 1.1 to 2.5 kPa. In this study, the authors observed that PaO_2 decreases by about 40% during suctioning.[3] In another study,[13] the mean change of PaO_2 from baseline during bronchoscopy was 26% by the end of the procedure; however, large inter-individual variations in PaO_2 changes were noticed. Oxygen saturations can remain lower than preprocedure levels 3 hours after bronchoscopy in 30% of patients.[14] The degree of hypoxemia induced by FFB was linked to the severity of pulmonary dysfunction and the decrease in alveolar ventilation.[12] The mechanisms that cause hypoxemia are multiple, and they may differ from patient to patient. In critically ill patients, hypoxemia can be caused by atelectasis due to the decrease in Vt, from the depletion of intra-alveolar oxygen due to frequent suctioning during the procedure and by the instillation of normal saline used for the BAL that washes out surfactant and causes localized "flooding" of the alveoli. Sedation used for the procedure and discoordination between the patient and the ventilator also contribute to development of hypoxemia.[5]

Trouillet JL et al.[12] reported that hypoxia is consistently associated when midazolam is used as the sedative drug in ventilated patients during bronchoscopy; and the degree of hypoxemia is linked to premedication and to the severity of illness.

HYPERCAPNIA

The presence of a bronchoscope in the airways causes a slight increase in the $PaCO_2$ averaging 1.1 kPa.[3] The rise in the $PaCO_2$ was observed by about 30%.[3] Hypercapnia is primarily the result

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of the hypoventilation that is caused by the airway obstruction and cannot be compensated by increasing the frequency of respiration and/or the Vt on the ventilator. The hypercapnia is usually well tolerated by the majority of the patients, and it does not require any special interventions. However, in patients with pulmonary hypertension and cerebral edema, caution needs to be exercised. Hypercapnia may cause acute and severe pulmonary vasoconstriction, thus raising the already increased pulmonary vascular resistance and pulmonary hypertension.[6] On the other hand, hypercapnia may cause cerebral vasodilatation worsening the cerebral edema.[6]

CARDIOVASCULAR EFFECTS

Although bronchoscope does not directly affect the cardiovascular system, it may cause significant cardiac as well as hemodynamic changes. Lindholm et al.[3] reported an increase in cardiac output reaching 50% during the procedure, and returning to baseline in 15 minutes after its completion. Schiffman et al.[15] demonstrated sinus tachycardia is the most common hemodynamic consequence during bronchoscopy observed in 55–58% patients followed by sinus bradycardia in 5–8%, premature ventricular contraction in 8%, and atrial premature contraction in 3–5% patients. Katz et al.[14] documented that at least an occasional atrial or ventricular ectopic beat occurred in 80% of patients during or after bronchoscopy. A significant rise of 10% above the control values was observed in arterial pressures, associated with an increase in pulse rate of 5%.[12]

These hemodynamics effects during bronchoscopy are attributed to multiple factors. There is reflex sympathetic discharge caused by mechanical irritation of the airways.[3,4,16] Hypoxemia and/or elevation of $PaCO_2$ lead to increase in vascular tone.[3] Increase in PIP, development of auto-PEEP and rise in intrathoracic pressure may affect the venous return and/or the afterload of the left ventricle. Increases in heart rate and in systemic blood pressure may also be caused by patient apprehension, anxiety, and discomfort. The development of auto-PEEP is of special importance because it may cause decrease in cardiac output. However, use of external PEEP or the requirement for inotropic agents was not found to be a significant risk factor.[12] All these effects are usually well tolerated by most patients but they can be very deleterious in patients with compromised cardiac function, increased systemic blood pressure, or increased intracranial pressure.

CONCLUSION

In principle, FFB in the ICU is not different from a bronchoscopy performed in a stable noncritically ill patient. The equipment as well as the basic technique is the same. Yet, differences in the patient population and in the conditions under which the procedure is performed in the ICU demand very different considerations and precautions. Bronchoscopy in critically ill patients can lead to a significant, although transitory, deterioration in pulmonary mechanics, hemodynamics status, and gas exchange parameters. A careful methodic attention to the anesthetic protocol, ventilator settings, monitoring of vital parameters during the peribronchoscopy period must be ensured for the maintenance of adequate ventilation and oxygenation while minimizing the risk of untoward physiologic effects.

From a practical point of view, in patients with compromised pulmonary functions, low O_2 saturation and hemodynamic instability, nonbronchoscopic techniques may be considered.

REFERENCES

1. Jolliet P, Chevrolet JC. Bronchoscopy in the intensive care unit. Intensive Care Med. 1992;18:160-9.
2. deCastro FR, Violan JS. Flexible bronchoscopy in mechanically ventilated patients. J Bronchol. 1996;3:64-8.
3. Lindholm C, Ollmann B, Snyder J, Millen E, Grenvik A. Cardiorespiratory effects of flexible fiberoptic bronchoscopy in critically ill patients. Chest. 1978;74:362-7.
4. Shennib H, Baslaim G. Bronchoscopy in the intensive care unit. Chest Clin North Am. 1996;6:349-61.
5. Estella A. Effects on respiratory mechanics of bronchoalveolar lavage in mechanically ventilated patients. J Bronchol Intervent Pulmonol. 2010;17:228-31.
6. Priftis KN, Anthracopoulos MB, Eber E, Koumbourlis AC, Wood RE. Paediatric Bronchoscopy. Progress in Respiratory Research. Basel: Karger; 2010. pp. 54-63.

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7. Lawson RW, Peters JI, Shelledy DC. Effects of fiberoptic bronchoscopy during mechanical ventilation in a lung model. Chest. 2000;118:824-31.
8. Klein U, Karzai W, Zimmermann P, Hannemann U, Koschel U, Brunner JX, et al. Changes in pulmonary mechanics after fiberoptic bronchoalveolar lavage in mechanically ventilated patients. Intensive Care Med. 1998;24: 1289-93.
9. Snow N, Lucas AE. Bronchoscopy in the critically ill surgical patient. Am Surg. 1984;50:441-5.
10. Steinberg KP, Mitchell DR, Maunder RJ, Milberg JA, Whitcomb ME, Hudson LD. Safety of bronchoalveolar lavage in patients with adult respiratory distress syndrome. Am Rev Respir Dis. 1993;148:556-61.
11. Meduri GU, Chastre J. The standardization of bronchoscopic techniques for ventilator-associated pneumonia. Chest. 1992;102:557S-64S.
12. Trouillet JL, Guiguet M, Gibert C, Fagon JY, Dreyfuss D, Blanchet F, et al. Fiberoptic bronchoscopy in ventilated patients: evaluation of cardiopulmonary risk under midazolam sedation. Chest. 1990;97:927-33.
13. Shrader DL, Lakshminarayan S. The effect of fiberoptic bronchoscopy on cardiac rhythm. Chest. 1978;73: 821-4.
14. Katz AS, Michelson EL, Stawicki J, Holford FD. Cardiac arrhythmias. Frequency during fiberoptic bronchoscopy and correlation with hypoxemia. Arch Intern Med. 1981;141:603-6.
15. Schiffman PL, Westlake RE, Fourre JA, Leonard ET. Arterial oxygen saturation and cardiac rhythm during transoral fiberoptic bronchoscopy. J Med Soc N J. 1982;79: 723-6.
16. Dellinger RP, Bandi V. Fiberoptic bronchoscopy in the intensive care unit. Crit Care Clin. 1992;8:755-2.

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Bronchoscopy in ICU

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Key Features

- The book `Bronchoscopy in ICU' provides a state of the art detailed descriptions of bronchoscopic procedures depicting a variety of bronchoscopy findings
- Introductory chapters provide idea of anatomy of tracheobronchial tree and bronchoscope accessories
- Subsequent chapters provide detailed instructions for performing procedures such as bronchial washing and lavage, handing of bronchoscope, bronchoscopy assisted by noninvasive ventilation, and bronchoscopy during mechanical ventilation
- Pediatric bronchoscopy in ICU is a highlighting feature of the book
- The book is a ready reckoner as it provides step-by-step descriptions and abundant illustrations
- The book provides a comprehensive and up-to-date content for intensivists, pulmonologists, anesthesiologists, surgeons and postgraduate students.

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