Pulmonary Barotrauma & Pneumothorax During Anesthesia

Marlis Rezende, DVM, PhD, DACVAA
Colorado State University

In anesthesia, the term *barotrauma* is used to describe lung tissue trauma resulting from excessively high airway pressure associated with excessive inflation of the lungs and alveolar overdistension, which can result in alveolar and pleural rupture.[1,2] When alveolar rupture occurs, air leaks into the pleural space and creates a closed pneumothorax, which in turn can rapidly evolve into a tension pneumothorax, especially if mechanical ventilation is being used. The resulting excessively high intrathoracic pressure impairs venous return to the heart, severely compromising stroke volume and cardiac output. If not quickly recognized and corrected, life-threatening cardiovascular collapse can occur.[2]

Causes of Barotrauma

Although an inadvertently closed pop-off valve is regarded as the most common cause for pulmonary barotrauma and pneumothorax during anesthesia, clinicians should be aware of other potentially dangerous scenarios that could lead to alveolar overdistension and rupture. Equipment-related barotrauma is typically caused by either excessive gas inflow into the breathing circuit and airway or restriction/obstruction of the gas outflow pathway.[3,4]

Excessive Inflow of Gas

Excessive inflow can occur from improper use of the oxygen flush valve, aggressive ventilator settings (high airway pressures and tidal volumes), and/or inappropriate connection of oxygen tubing (meant for oxygen insufflation via open mask) to a cuffed endotracheal tube, laryngeal mask airway, or other airway device without the ability to allow excess gas to vent.

The oxygen flush valve allows oxygen at high pressure and volume into the breathing system (35-70 L/min with a pressure of 45-60 pounds per square inch gauge [PSIG], which becomes approximately 1 L/s into the breathing system).[3,4] A nonrebreathing system (eg, Bain breathing system) has a relatively small inner volume and little compliance. Therefore, use of the flush valve while a patient is connected to a nonrebreathing system transmits excess volume and pressure directly to the patient's airway and lungs. Similarly, if the oxygen flush valve is used during the inspiratory phase of mechanical ventilation, the patient's lungs may be exposed to excessive pressure and overdistension. During the inspiratory phase, the ventilator's driving pressure actively compresses the bellows to deliver a breath, and the ventilator's exhaust valve is closed. Because the ventilator's driving pressure (65-75 cm H_2O) prevents the expansion of the bellows until breathing system pressures overcome the driving pressure, activation of the flush valve at this time (eg, to reinflate the bellows after a brief disconnection) would direct all the volume and resulting pressure to the breathing circuit.[3]

Aggressive Ventilator Settings

Inappropriately performed or overly aggressive mechanical ventilation can also cause barotrauma. In contrast to spontaneous ventilation, in which inspiration relies on the negative intrathoracic pressure generated by chest expansion to passively inflate the lungs, mechanical ventilation actively inflates the lungs using positive pressure. If excessive high tidal volumes and/or peak inspiratory (and plateau) pressures are used, barotrauma may occur. Overzealous manual breaths can have similar effects, when high tidal volumes and/or airway pressures are generated.

Outflow Restriction or Obstruction

Any form of significant breathing circuit outflow restriction or occlusion can lead to excessive airway pressures and lung overinflation, as there is no path to release the excess gas from the breathing system. Common examples include pop-off valve obstruction, compression or kinking of the scavenger hose, obstruction of the F/AIR anesthesia gas filter canister vents, and kinking of the hose that connects the ventilator to the breathing circuit. Additional care should be taken when using nonrebreathing systems, as the combination of the required high fresh gas flow rates with the relatively small inner volume of the breathing circuit allows airway pressures to rise very quickly in the case of an obstruction.

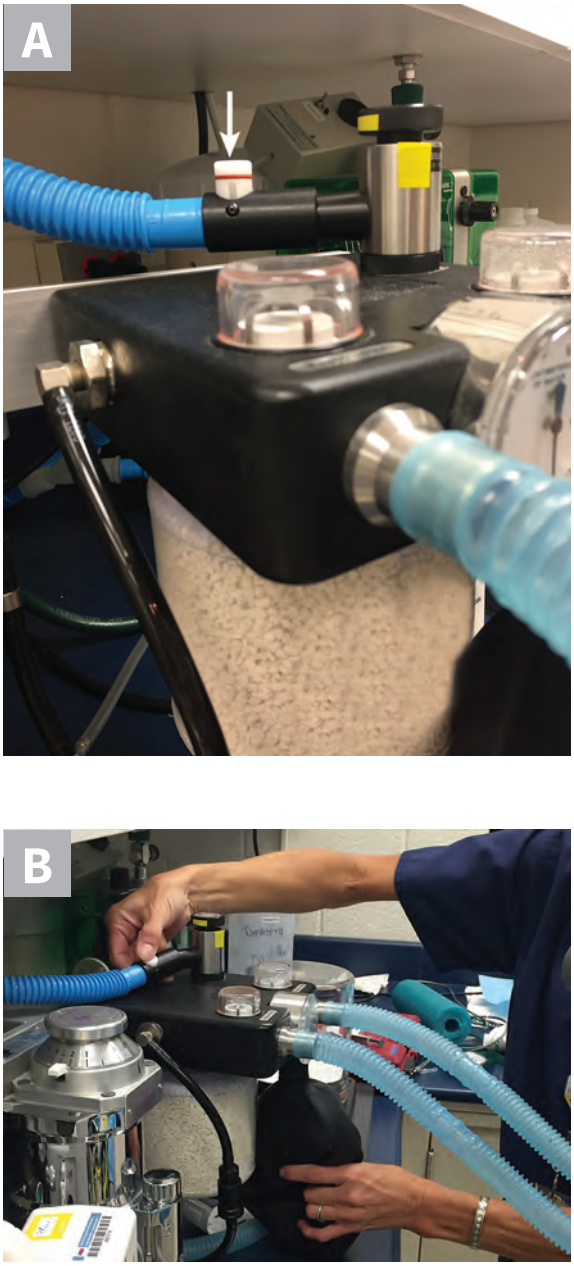
Patients at Increased Risk

Pre-existing lung disease (eg, pulmonary bullae, pneumonia, acute respiratory distress syndrome, feline asthma) may predispose animals to barotrauma and pneumothorax under anesthesia.[2] Other pre-existing conditions,

SAFETY DEVICES TO PREVENT BAROTRAUMA

Most anesthesia-associated barotrauma events (and resulting pneumothorax) can be avoided through both a functional understanding of the anesthesia machine and the presence of safety features designed to prevent harmful conditions or alert the team if such conditions arise, including:

- Pop-off occlusion valve (***Figure 1***, next page). This device can be attached to the outflow port of the anesthesia machine's original pop-off valve. When the top button of the valve is pushed, flow out of the pop-off valve is occluded and a manual breath can be administered. Once the button is released, flow through the valve is automatically re-established, minimizing the risk for forgetting to reopen the original pop-off valve, as it will always be left open. Although this can be useful during spontaneous ventilation, it does not completely eliminate the risk, as the original pop-off valve will still need to be closed (and opened afterward) if mechanical ventilation is to be used or during the process of pressure checking the anesthesia machine for leaks.
- High-pressure alarm (***Figure 2***, page 71). This device provides an audible alarm when the breathing circuit pressure reaches a set level (typically 20 cm H_2O). In addition, it can be used with both rebreathing and nonrebreathing systems with spontaneous or mechanical ventilation. The device will alarm when a dangerous breathing circuit pressure occurs (independent of cause), providing the clinician time to intervene.



▲ **FIGURE 1** Pop-off occlusion valve open (***A***; ***arrow***) and actively closed while giving a manual breath (***B***)

although not a consequence of true barotrauma, may increase the risk for a pneumothorax due to pulmonary tissue fragility or injury. This is especially notable in patients that have undergone trauma to the chest (eg, hit by a car, kicked by a horse), as the pulmonary contusions create areas of alveolar fragility.[5] Patients with pulmonary neoplastic masses, cysts, abscesses, or foreign body migration can be similarly predisposed to pneumothorax despite appropriate ventilation settings.

Pneumothorax

Pneumothorax is a life-threatening complication of barotrauma. Awareness and early recognition are key to a successful outcome. During anesthesia, a closed pneumothorax can rapidly evolve to a tension pneumothorax. In a *one-way valve* mechanism, air leaks out during lung inflation. As the lung tissue recoils during exhalation, air cannot escape via its entry path and becomes trapped outside the lungs in the thoracic cavity. The high-pressure intrathoracic environment that soon develops limits lung expansion and, most importantly, prevents venous return to the heart, leading to cardiovascular collapse.

As the lungs' ability to expand decreases and atelectasis increases, a change in breathing pattern typically occurs, followed by dyspnea and a decrease in oxygen saturation. As the pneumothorax evolves to a tension pneumothorax and venous return is compromised, severe hypotension and hypoxemia occurs. Reflex tachycardia may be present. The patient becomes more difficult to ventilate as lung compliance decreases and chest wall movement is diminished. On auscultation, lung sounds may be absent or significantly diminished.

Early recognition of clinical signs can be hindered in cases in which the patient's overall condition is already compromised (eg, patient in shock, with hypovolemia, and/or with significant intraoperative blood loss). Pneumotho-

rax may only become evident on cardiopulmonary collapse. It is therefore important that the clinician is aware of the potential risk for a pneumothorax based on the patient history and potential predisposing conditions.

If a pneumothorax is suspected, positive pressure ventilation should be immediately discontinued and thoracocentesis performed for emergency decompression of the chest. Once the intrathoracic pressure is relieved, arterial blood pressure and oxygen saturation will improve. A thoracostomy tube should then be placed to allow air to be continuously removed until the ruptured alveoli can seal to prevent the reoccurrence of a tension pneumothorax. (For a detailed description of how to perform a thoracocentesis or place a thoracostomy tube, see ***Suggested Reading***.)

Conclusion

Understanding and ensuring the proper functioning of the anesthesia machine before each use, adding safety features to help prevent and/or recognize mistakes, and having a dedicated individual to monitor anesthesia who is able to rapidly recognize equipment issues and adverse events are key to increasing patient safety.

▲ **FIGURE 2** High-pressure alarm and tubing (***arrow***) that is connected to the anesthesia machine's fresh gas outflow

References

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Suggested Reading

Hackett TB, Mazzaferro EM. Thoracocentesis and thoracostomy tube placement. In: Hackett TB, Mazzaferro EM. *Veterinary Emergency and Critical Care Procedures*. 2nd ed. Ames, IA: Wiley-Blackwell; 2012:101-132.