Auto-PEEP-like condition recognized by a sudden decrease in airway pressure during pressure controlled ventilation and low-flow anesthesia

—A case report—

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During mechanical ventilation in the intensive care unit, auto-positive end-expiratory pressure (auto-PEEP) has been reported to occur in obstructive airway conditions aggravated by inappropriate ventilator settings. In this paper, we report a case of auto-PEEP-like problem during anesthesia, mainly caused by excessive sputum. After being positioned prone for spine surgery, the patient received pressure controlled ventilation at a low fresh gas flow rate. One hour after the start of surgery, sudden decreases in pressure and flow occurred. The typical maneuvers which could be performed by the anesthesiologists in the situations suggesting leakage within the breathing circuit consist of pressing the oxygen flush valve and manual hyperventilation for the initial evaluation. But from our experience in this case, we have learned that such maneuvers could cause unacceptable aggravation in the event of auto-PEEP. Also in this report, we discuss the difficulties in prediction based on the present knowledge of preoperative evaluation and the presumably best management policy regarding this type of auto-PEEP. (Anesth Pain Med 2015; 10: 223-226)

Key Words: Airway pressure, Anesthesia, Auto-PEEP, Low fresh gas flow rate, Pressure controlled ventilation.

CASE REPORT

A previously healthy woman (46-year-old, 155 cm, 49 kg) sitting in the passenger seat was involved in a car accident. In the emergency room, she was found to have a complex
fracture and epidural hematoma in the area of the frontal bone, T3 and T4 spine fractures, multiple rib and scapula fractures, and bilateral hemothorax or pleural effusion accompanied by a decrease in consciousness. After emergency craniotomy, she showed improvement in consciousness but mechanical ventilation was required due to excessive production of sputum. Two days later, she suffered self-extubation and received physical therapy and oxygen. One week after the trauma, posterior fixation of the thoracic spine was planned. An AP chest X-ray obtained on the day before the operation showed persistent hemothorax or pleural effusion (Fig. 1), but chest tube insertion was not performed because she did not show any signs of significant respiratory distress.

In the operating room before induction of anesthesia, the patient had an SpO2 of 98% under the inhalation of oxygen (5 L/min) via nasal prongs. After prone positioning, the operation was begun. Anesthesia ventilator used was Dräger primus™ (Dräger AG & Co, Germany). At the start of surgery, we used volume controlled ventilation (tidal volume 440 ml, respiratory rate 14 /min, and I : E ratio 1 : 2) and the resulting peak airway pressure was 23 cmH2O. At an FiO2 of 60%, SpO2 was 94%, and ABGA showed a pH 7.5, a PaCO2 37 mmHg, a PaO2 78 mmHg, and an HCO3- 29 mM. After that, the ventilation mode was changed into pressure control mode. Under the set pressure of 22 cmH2O, respiratory rate of 14 /min and I : E ratio of 1 : 2, tidal volume was around 460 ml and the resulting blood gas values showed little improvement; SpO2 was 98% and ABGA showed a pH 7.5, a PaCO2 33 mmHg, a PaO2 93 mmHg, and an HCO3- 29 mM. The fresh gas flow rate was 1.1 L/min, which was within the range for low fresh gas flow [10].

One hour after the operation, there were acute changes in respiratory monitoring parameters. Airway pressure, ventilatory flow rate, and end-tidal CO2 waveforms showed acutely decreasing patterns with an accompanying decrease in SpO2 to 95%. The acute changes occurred within 2 minutes and the lastly recognized peak airway pressure level was below 10 cmH2O. After observing these changes, manual ventilation was begun by pressing the oxygen flush valve. After inflation of the reservoir bag, only a single manual pressing of the bag was performed. But, after the initial feeling of increased resistance, there was a feeling of progressive deflation of the reservoir bag, suggesting leakage within the breathing circuit. Therefore, we disconnected the breathing circuit from the endotracheal tube and then tried to identify the leakage point. At that time, we could hear a loud sound suggesting the release of accumulated pressure, also mixed with a coarse vibrating sound related to the presence of sputum. We suspected that auto-PEEP had occurred. After several sessions of suctioning of the thick yellowish sputum, arterial blood gas values returned to the previous level (a pH of 7.5, a PaCO2 of 40 mmHg, a PaO2 of 95 mmHg, an HCO3- of 31 mM under pressure controlled ventilation with an inspiratory pressure of 22 cmH2O and an FiO2 of 60%). At that time, hemodynamic signs were stable with blood pressure of 100-110/55-60 mmHg and heart rate of 95-100 beats/min.

Fig. 1. Preoperative chest AP view shows diffuse increased opacities in both lungs, suggesting hemothorax or pleural effusion.

Fig. 2. Postoperative chest AP view shows reduction in previous opacities in both lungs secondary to the insertion of both chest tubes. Also, it shows instrumentation of T1-7.
After the operation, the patient received bilateral chest tube insertion under mechanical ventilation (Fig. 2). The amount of drainage of blood-tinged effusion from the right and left sides of the chest was 800 ml and 550 ml, respectively. Two days later, she was successfully liberated from mechanical ventilation.

DISCUSSION

The pressurized and hyperinflated condition of the lungs in this patient was caused by sputum accumulation in the prone position. The stenosed airway condition induced by sputum accumulation caused entrapment of most of the mechanical breaths. Pepe and Marinii [11] observed similar problems in patients receiving mechanical ventilation under the diagnosis of exacerbated chronic pulmonary disease or respiratory complications of burn injuries and they named them auto-PEEP. They also reported the clinically significant physiological changes such as increased work of breathing and decreased cardiac output. Importantly, they recognized the difficulty in detecting the presence of auto-PEEP by observing the pressure gauge within the breathing circuit, and suggested a special measurement method: expiratory port occlusion at the end of the set exhalation period. After the initial reports in patients with chronic obstructive pulmonary disease, auto-PEEP has also been reported in many other diseases (asthma, burns, acute respiratory distress syndrome), pathophysiological conditions (excessive airway secretions, airway edema), and inadequate ventilator settings (high minute ventilation, decreased expiratory time, or increased I : E ratio), which could be regarded as the direct causes of auto-PEEP [1-6].

Almost all cases reported in the usual literature occurred in the ICU patients. The breathing circuits of mechanical ventilators in the ICU are frequently categorized as the circle type only from the external appearance, but more accurately, they are semi-open systems which expel all gas received during the previous inspiratory phase into the air [9]. In comparison, anesthesia ventilators operate in a closed or semi-closed fashion according to the fresh gas flow rate, causing the whole or a certain amount of the previously inspired volume to be rebreathed. We think this difference can also lead to changes in clinical manifestations associated with auto-PEEP occurring under anesthesia. For example in the ICU ventilator setting, the patients developing auto-PEEP in the volume control mode show increased peak and plateau pressures although the presence of auto-PEEP itself is not detected in the pressure monitoring system [11-13]. If the patient is under ventilation in the pressure control mode, even the inspiratory pressure levels do not show any changes.

However, if auto-PEEP develops during the use of anesthesia ventilators, varying manifestations may be observed depending on the balance between the fresh gas flow rate and the entrapment volume. In our case, the fresh gas flow rate was not high enough to compensate for the entrapment volume, resulting in acute decreases in airway pressure and flow rate following underfilling of the bellows chamber over time. In the pressure control mode, simultaneous development of increased peak and plateau airway pressures could not be observed [11-13]. Ultimately, acute decreases in respiratory parameters alerted us to take immediate action. On the contrary, if the fresh gas flow rate had been high enough, early changes in pressure and flow waves might not have been detected and prolonged accumulation of entrapped volume could have caused severe cardiopulmonary complications.

Regarding diagnostic tests, although the expiratory port occlusion would be the most valuable test, there are some other indicators that help in recognizing the development of auto-PEEP. From simultaneous observations of pressure and flow waves, the continuing expiratory flow which has not yet reached zero at the starting point of inspiratory airway pressure can be a clue. Also, after giving a long enough expiratory time transiently and thereby decreasing the level of supposedly existing auto-PEEP, the peak and plateau airway pressures during the next mechanical breath can be lowered if auto-PEEP has really developed [1,3,14]. However in our case, the feature of acutely decreasing pressure and flow did not permit detailed observations possibly related to auto-PEEP. Therefore, in the kind of situation, as in our case, obscuring the presence of auto-PEEP, the typical immediate action that should be taken by the anesthesiologists would be to identify the leakage point in the breathing circuit. For this purpose, anesthesiologists usually switch to manual ventilation with or without pressing the oxygen flush valve and also frequently disconnect the breathing circuit from the endotracheal tube in order to identify the leakage points. During this process, we confirmed the presence of auto-PEEP in the patient. We consider that repetitive flushing and manual ventilation maneuvers could have caused a major problem in this kind of situation.

Were there any exact predictive factors for the occurrence of this problem? Although data on symptoms such as significant coughing or sputum production were not available from the medical records, there is a possibility that the patient had
significant secretions but did not show respiratory symptoms due to multiple rib fractures. Also, the management for pleural effusion during the preoperative phase was very conservative because the amount of pleural effusion around both lungs was rather high (right side 800 ml, left side 550 ml), suggesting the existence of acute unresolved conditions. Most importantly, many essential questions remained unanswered. Many anesthesiologists would probably think that most patients who have excessive sputum rarely develop auto-PEEP. From this point of view, it turns out that we do not have exact knowledge about the process of formation of auto-PEEP related to the role of the amount of sputum or the predisposing conditions. Therefore, we need to systematically perform more clinical studies for assessing the exact predisposing conditions or mechanistic studies for determining the exact physiological processes involved in the development of the problem.

Although we cannot accurately state the exact underlying predisposing factors for this problem, there are reasonable ways of preventing this problem. If the patient had received more aggressive lung care, it could have prevented the development of auto-PEEP. Also, if the anesthesiologists had checked the breath sounds frequently in such a risky patient, it could have prevented the occurrence of this problem.

Among the established treatment methods of auto-PEEP, the ventilation strategy is comprised of decreasing the tidal volume and increasing the expiratory time with some allowance for increased PaCO₂ resulting from decreased minute ventilation. The other methods include the suctioning of secretions, use of an appropriate size endotracheal tube and bronchodilators. Also, there is a general agreement on the use of extrinsic PEEP, which is a pressure of about 80% of the level of auto-PEEP, and it facilitates the exhalation of the entrapped volume without aggravating the auto-PEEP [1,3,4,7].

In conclusion, the anesthesiologists should know about the possibility of development of auto-PEEP resulting from excessive sputum according to the pathological condition of the patients’ lungs, although it is not yet understood in detail. If the anesthesiologists observe acute decreases in respiratory monitoring parameters under low flow rate conditions, they should also consider the possibility of auto-PEEP and take some measures including avoidance of manual hyperventilation and disconnection of the breathing circuit from the endotracheal tube.

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