

Continuous Monitoring of Arterial Blood Gas during Bronchopulmonary Lavage for Pulmonary Alveolar Proteinosis

Hiroshi Sakamoto*, Yoshitada Ito**, Hiroshi Kawahigashi***
 Hideyuki Mashio***, Yukiko Goda***
 Takahisa Mayumi**, Osamu Kemmotsu**

Abstract

Pulmonary proteinosis is a rare lung disorder in which an amorphous proteinaceous material is deposited in alveoli and bronchioli. Bronchopulmonary lavage (BPL) is the only successful treatment. We had a case with pulmonary proteinosis scheduled for BPL under general anesthesia.

Anesthesia was induced with fentanyl and thiamylal. The trachea was intubated with a double lumen tube following vecuronium. Anesthesia was maintained with sevoflurane in oxygen. An arterial cannula with a sensor tip, a pulmonary artery catheter and a pulse oximeter were placed to monitor arterial blood pH, PaO₂, PaCO₂, mixed venous oxygen saturation (S \bar{V} O₂), pulse oxymetry (SPO₂), arterial blood pressure, pulmonary and central venous pressure and cardiac index continuously. Physiological saline was infused into the washing lung by a pressure of 30 cmH₂O and drained by -60 cmH₂O. After BPL, mechanical ventilation was continued for 18 hours. Right and left ventricular stroke work indices (RVSWI and LVSWI), systemic and pulmonary vascular resistance indices (SVRI, PVRI), oxygen delivery index (DO₂I), oxygen

consumption (\dot{V} O₂), alveolar to arterial oxygen tension difference (DAaO₂), alveolar to arterial blood oxygen tension difference (Cavo₂) and intrapulmonary shunt (Qs/Qt) were calculated.

PAP, CVP, PaO₂, SPO₂, and S \bar{V} O₂ increased by the instillation of fluid while they were vice versa by the drainage. During the instillation, the increased intra-alveolar pressure caused the diversion of pulmonary blood flow to the ventilated lung. Drainage reversed these changes. PaO₂, SPO₂ and S \bar{V} O₂ indicate blood oxygenation. The change of PaO₂ is the quickest and most among them. Because arterial oxygenation changes quickly during BPL, the placement of an arterial cannula with a continuous blood gas analysis sensor is useful.

Key words : Continuous arterial blood gas analysis, Bronchopulmonary lavage, Unilateral whole lung lavage, Pulmonary proteinosis.

Introduction

Pulmonary alveolar proteinosis, which is characterized by the progressive accumulation of lipid, protein, and cellular debris in alveolar spaces, could show the symptom of impaired gas exchange¹⁾. This is dependent on the deficiency in cleaning of cellular debris and the byproducts of alveolar metabolism and secondary infection²⁻⁴⁾. If the patients with pulmonary alveolar proteinosis failed to respond to inhalational aerosol such as trypsin, bronchopulmonary lavage

*Department of Anesthesia, Cardiovascular Center Hokkaido Ohno hospital, Sapporo, Japan

**Department of Anesthesiology, Hokkaido University School of Medicine, Sapporo, Japan

***Department of Anesthesia, Sapporo City General Hospital

(BPL) that could remove alveolar materials in a part or entire lung is the choice of treatment^(2,5-10).

BPL procedure requires general anesthesia and one lung ventilation to separate the washed and non-washed lung. The infusion of large amount of saline can be associated with significant changes of hemodynamics and arterial oxygenation. During BPL, hemodynamics has been assessed by continuous monitoring of central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and cardiac output (CO) but arterial oxygenation has been evaluated by intermittent analysis of arterial blood gas and continuous noninvasive measurement of arterial oxygen saturation using pulse oxymetry. Hypoxia is the most serious complication associated with BPL⁽¹¹⁾.

Continuous intra-arterial blood gas monitoring could have potential advantages in clinical situation over intermittent measurements during anesthesia and in the Intensive Care Unit (ICU)⁽¹²⁻¹⁵⁾. Availability of continuous data could offer useful information for an immediate therapeutic decision that is necessary in critical situation such as BPL procedure in the patients with pulmonary alveolar proteinosis⁽¹⁵⁾. In this study, we evaluated the usefulness of continuous intra-arterial blood gas monitoring compared to arterial oxygen saturation using pulse oxymetry in BPL procedure for pulmonary alveolar proteinosis.

Case Report

A 40-year old male chiefly complained of dry cough and became progressively short of breath for these two months. He has been medicated chlorpromazine 500 mg, haloperidol 20 mg, fluphenazine 20 mg, perphenazine 15 mg, sulpiride 1200 mg, sultopride 1800 mg, timiperone 12 mg, biperiden 6 mg, trihexyphenidyl 6 mg, levomepromazine 50 mg, nitrazepam 10 mg, estazolam 5 mg, sennosides 24 mg and pantethine 600 mg for schiz-ophrenia for recent 12 years. After establishing the diagnosis of pulmonary alveolar proteinosis by transpulmonary lung biopsy, four unilateral whole-lung lavages under general anesthesia at a week interval were scheduled. On the preoperative evaluation, he clearly recognized his present condition and agreed to undergo BPL pro-

cedure to improve his symptoms. Chest examination revealed normal diaphragmatic excursion but slight fine rales at basilar and axillary region. Reduction of % vital capacity (75%) and the first second forced expiratory volume (77.2%) might reflect the decrease of PaO₂ (61.9 mmHg) at room air. There was a remarkable reduction in the diffusing capacity.

Anesthesia course

Premedication consisted of brotizolam 0.25 mg and ranitidine 150 mg orally 90 min before induction of anesthesia. General anesthesia was induced with intravenous (i.v.) thiamylal 4 mg·kg⁻¹ and i.v. fentanyl 100 μg. After i.v. vecuronium 1.2 mg·kg⁻¹, his trachea was intubated with a 39F left-sided double lumen endobronchial tube (Broncho-Cath[®], Mallinckrodt Anesthesia Products, St. Louis, MO). The correct position of the tube was confirmed by fiberoptic bronchoscopy. Anesthesia was maintained with isoflurane 1 MAC combined with supplementary i.v. fentanyl. We placed a thin fiberoptic sensor tip (FOxS[®], Puritan-Bennett Corp., Carlsbad, CA, USA) through the radial artery for continuous monitoring of arterial blood pressure and arterial gas (PB3300 IABG monitor[®], Puritan-Bennett Corp., Carlsbad, CA, USA). Then a pulmonary artery catheter (Edwards Swan-Ganz CCO/SvO₂ thermodilution catheter[®], Baxter Health Care Corp., Irvine, CA, USA) was placed through the right internal jugular vein to monitor central venous pressure (CVP), pulmonary artery pressure (PAP), mixed venous oxygen saturation (SvO₂) and cardiac output (CO) continuously (Vigilance[®], Baxter Health Care Corp., Irvine, CA, USA)⁽¹⁶⁾. Cardiac index (CI), right and left ventricular stroke work indices (RVSWI and LVSWI), systemic and pulmonary vascular resistance indices (SVRI, PVRI), oxygen delivery index (DO₂I), oxygen consumption ($\dot{V}O_2$), alveolar to arterial oxygen tension difference (DAAO₂), alveolar to arterial blood oxygen tension difference (Cavo₂) and intrapulmonary shunt (Qs/Qt) were calculated by a built-in computer.

After mechanical ventilation under FiO₂ 1.0 and a fresh gas flow of 4 l·min⁻¹ for 15 min to wash out nitrogen in lungs, the orifice of the double lumen tube

to the lavage-side was clamped^{6,17)}. During one lung ventilation, we kept the airway pressure less than 30 mmHg adjusting tidal volume, ventilation frequency and inspiratory-expiratory phase time ratio to avoid barotrauma. After confirming the lavage-side lung to be collapsed by chest X-ray, one lung was filled up with maximum of 1.8 liters of normal saline, which was infused by gravity from the position 30 cm above the patient's midaxillary line. When no fluid flowed into the lung, the lung was drained by gravity to 60cm below the midaxillary line^{5-7,17)}. Mechanical percussion was applied during filling and drainage phases. This process was repeated 7 to 18 times in the right and 5 to 7 times in the left lung, respectively. About 94% of the total infused fluid was drained.

After completion of the procedure, both lungs were re-expanded^{5-7,17)}. The patient was transferred to the ICU with a single lumen endotracheal tube in place and ventilated with PEEP⁵⁾. The patient's trachea was extubated after 18 hours in the ICU. Continuous arterial blood gas monitoring was continued till extubation.

The changes of PaO₂, PaCO₂, SpO₂, SvO₂, Cavo₂, $\dot{V}O_2$ and DAaO₂ during BPL were shown in table 1. The changes of HR, mAP, mPAP, CVP, CI, LVSWI, RVSWI, SVRI, and PVRI during BPL were shown in

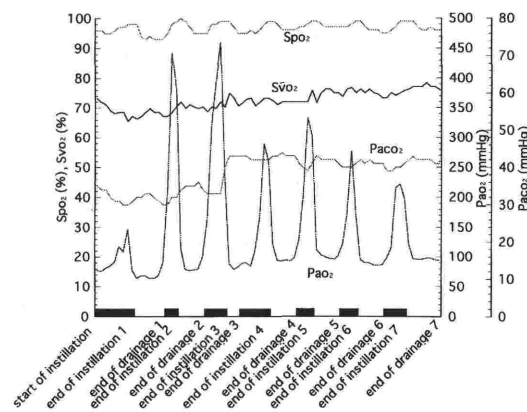


Fig. 1 Typical changes of blood gas analysis during bronchopulmonary lavage.
 PaO₂=arterial oxygen tension, PaCO₂=arterial carbon dioxide tension, SpO₂=pulse oxymetry, SvO₂=mixed venous oxygen saturation
 ■= instillation phase.

table 2. Typical respiratory and hemodynamical changes during the left lung lavage were shown in figures 1-4.

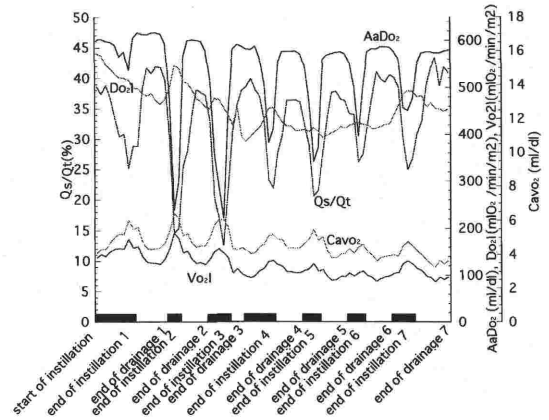


Fig. 2 Typical changes of respiratory function during bronchopulmonary lavage.
 Qs/Qt = intrapulmonary shunt, DAaO₂ = alveolar to arterial oxygen tension difference, DQI = oxygen delivery index, VoI = oxygen consumption index, Cavo₂ = arterial to mixed venous oxygen content difference
 ■= instillation phase.

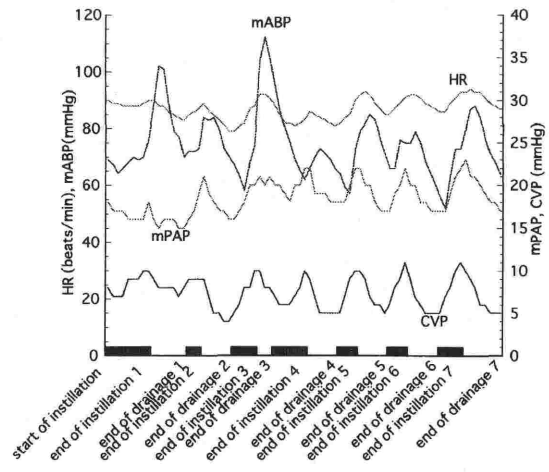


Fig. 3 Typical changes of hemodynamics during bronchopulmonary lavage.
 HR = heart rate, mABP = mean arterial blood pressure, mPAP = mean pulmonary arterial blood pressure, CVP = central venous pressure
 ■= instillation phase.

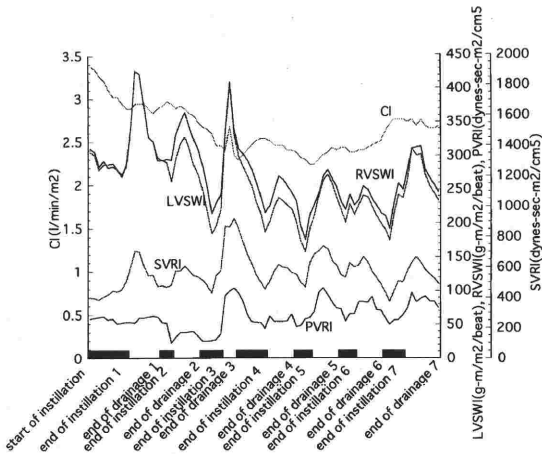


Fig. 4 Typical changes of cardiac function during bronchopulmonary lavage.

CI = cardiac index, LVSWI = left ventricular stroke work index, RVSWI = right ventricular stroke work index, PVRI = pulmonary vascular resistance index, SVRI = systemic vascular resistance index
 ■ = instillation phase.

Discussion

Intermittent cardiovascular and respiratory monitoring associated with BPL were well documented^{5,8,18-19}. The repeated changes in BPL are that PaO₂ increases during the infusion phase and abruptly decreases during the drainage phase. These changes were clearly and continuously observed in our case.

These changes are resulted from the change of intrathoracic pressure. The fluid instillation results in not only the alveolar expansion but also the collapse of pulmonary arteries and capillaries in the washing lung. The collapse of pulmonary arteries and capillaries causes the increase of PAP. The alveolar expansion induces the mediastinal shift from fluid-filled lung^{6,7}. The squeezed blood from the washing lung initially increases venous return. Then PAP increases as the instillation fluid fills the washing lung. Finally, the squeezed vessels and mediastinal shift provoke the reduction of RVSWI and LVSWI^{5-7,20}. This situation frequently accompanied with the reduction of mABP and HR. More blood flows into the ventilated lung because of the collapsed pulmonary blood vessels in the washing lungs. This moves the ventilation-perfu-

sion ratio toward 1.0 and results in reduction of Qs/Qt and increases in PaO₂ and SPO₂^{5,7}. The elevation of PaO₂ may increase Cavo₂ followed by increased $\dot{V}O_2$ because the patient is metabolically stable under general anesthesia⁵. The drainage phase shows a reverse effect. As fluid drained, mPAP and CVP decrease. The blood perfuses dilated capillary bed in the washing lung⁷. This could decrease venous return, PAP, CVP, RVSWI, and LVSWI and Qs/Qt increases⁶. The reduction of CI and the ventilation-perfusion ratio results in decreases of PaO₂, SPO₂, Cavo₂ and $\dot{V}O_2$. The changes of mPAP and CVP produce the same directional changes of SVRI and PVRI. But these changes of mPAP and CVP are mainly affected by the intrathoracic pressure with filling and emptying lungs with fluid instead of air. They are not reflecting ventricular volume and these SVRI and PVRI may not indicate vascular resistance²¹⁻²³.

All the changes in respiration and hemodynamics depend on how much instilled saline is in the washing lung and it means they depend on the intra-alveolar pressure. One liter of instilled fluid is almost the half of full dose of it and the values of mABP, mPAP, CVP, CI, PaO₂, S $\dot{V}O_2$ and Qs/Qt at 1 liter instillation are about the median of the values between at the end of drainage and at the end of instillation. This may suggest that the relationship between PaO₂, S $\dot{V}O_2$, Qs/Qt and intra-alveolar pressure is linear.

S $\dot{V}O_2$ monitoring requires the placement of a pulmonary artery catheter. The pulmonary artery catheter gives pulmonary hemodynamic data other than S $\dot{V}O_2$ but they are not essential during BPL. Although SPO₂ is non-invasive, SPO₂ can not detect a sudden PaO₂ decrease accurately and promptly because of the sigmoid shape of oxygen-dissociation curve. Our results indicate that PaO₂ changed quicker and more than SPO₂ and S $\dot{V}O_2$. An early detection of hypoxemia and its treatment are important for prevention of hypoxemic damages to vital organs during BPL. Although continuous monitoring of PaO₂ is invasive, the catheter is worth placing in the radial artery during BPL.

In conclusion, we evaluated the usefulness of continuous arterial blood gas monitoring during bron-

hopulmonary lavage in a patient with pulmonary alveolar proteinosis. We also examined cardiovascular changes associated with this procedure. Respiratory and hemodynamic changes depend on the quantity of lavage fluid in the washing lung. And continuous blood gas monitoring is the quick and accurate way to know arterial oxygenation of the patient during BPL.

References

- 1) Rosen SH, Castleman B, Liebow AA, et al : Pulmonary alveolar proteinosis. *N Eng J Med* 258 : 1123-1142, 1958
- 2) Wasserman K, Blank N, Fletcher G : Lung lavage (alveolar washing) in alveolar proteinosis. *Am J Med* 44 : 611-617, 1968
- 3) Ramirez RJ : Bronchopulmonary lavage, new techniques and observations. *Dis Chest* 50 : 581-588, 1966
- 4) Kawakami M : Pulmonary alveolar proteinosis, clinical features and some findings relating to sex differences. *Hai to Shin* 39 : 68-74 (abstract in English)
- 5) Blenkarn GD, Lanning CF, Kylstra JA : Anesthetic management of volume controlled unilateral lung lavage. *Canad Anaesth Soc J* 22 : 154-163, 1975
- 6) Lippmann M, Mok MS : Anesthetic Management of pulmonary lavage in adults. *Anesth Analg* 56 : 661-668, 1977
- 7) Smith JD, Millen JE, Safar P, et al : Intrathoracic pressure, pulmonary vascular pressures and gas exchange during pulmonary lavage. *Anesthesiology* 33 : 401-405, 1970
- 8) Rogers RM, Levin DC, Gray BA, et al : Physiologic effects of bronchopulmonary lavage in alveolar proteinosis. *Am Rev Respir Dis* 118 : 255-264, 1978
- 9) Selecky PA, Wasserman K, Benfield JR, et al : The clinical physiological effect of whole-lung lavage in pulmonary alveolar proteinosis: a ten-year experience. *Ann Thorac Surg* 24 : 451-461, 1977
- 10) Kao D, Wasserman K, Costley D, et al : Advances in the treatment of pulmonary alveolar proteinosis. *Am Rev Respir Dis* 111 : 361-363, 1975
- 11) Benumof JL, Alfery DD : Anesthesia for thoracic surgery, anesthesia 4th edition. edited by Miller RD, New York, Churchill Livingstone, 1986, pp.1380-1419
- 12) Wahr JA, Tremper KK : Continuous intravascular blood gas monitoring. *J Cardiothorac Vasc Anesth* : 342-353, 1994
- 13) Venkatesh B, Hendry SP : Continuous intra-arterial blood gas monitoring. *Intensive Care Med* 22 : 818-828, 1996
- 14) Roupie EE, Brochard L, Lemaire FJ : Clinical evaluation of a continuous intra-arterial blood gas system in critically ill patients. *Intensive Care Med* 22 : 1162-1168, 1996
- 15) Bohrer H, Schmidt H, Amann R, et al : Superiority of continuous intra-arterial blood gas monitoring over pulse oxymetry with acute respiratory failure. *Anaesthesia* 496 : 551, 1994
- 16) Okada K, Kinoshita Y, Iwasaki H, et al : Evaluation of monitoring during lung lavage. *Jpn J Anesthesiol* 37 : 727-730, 1988 (abstract in English)
- 17) Ramirez RJ: Alveolar Proteinosis : Importance of pulmonary lavage. *Amer Rev Resp Dis* 103 : 666-678, 1971
- 18) Kobayashi T, Nakanishi T, Kishizuchi S, Murakami S : A case of alveolar proteinosis change in blood gases by pulmonary lavage. *Jpn J Anesthesiol* 27 : 43-47, 1978 (abstract in English)
- 19) Goto Y, Akashi M, Ishikawa K, et al : Clinical studies on the classification of techniques and efficiency of whole-lung lavage. *Jpn J Anesthesiol* 31 : 19-28, 1982 (abstract in English)
- 20) Busque : Pulmonary lavage in the treatment of alveolar proteinosis. *Can Anaesth Soc J* 42 : 380-389, 1977
- 21) Swenson JD, Astle KL, Bailey PL : Reduction in left ventricular filling during bronchopulmonary lavage demonstrated by transesophageal echocardiography. *Anesth Analg* 81 : 634-637, 1995
- 22) Hansen RM, Viquerat CE, Mathay MA, et al : Poor correlation between pulmonary arterial wedge pressure and left ventricular end-diastolic volume after coronary artery bypass graft surgery. *Anesthesiology* 64 : 764-770, 1986
- 23) O'Quin R, Marini JJ : Pulmonary artery occlusion pressure:clinical physiology, measurement and interpretation. *Am Rev Respir Dis* 128 : 319-326, 1983

(Circ Cont 19 : 274~278, 1998)