# Two Cases of Bronchospasm during Operation in a Prone Position

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Althogh bronchospasm rarely occurs under general anesthesia it can cause serious ventilatory insufficiency that may not respond to treatment and can sometimes lead to critical conditions.

This report describes two patients with no history of asthma or any allergic diseases who developed bronchospasm during surgery in the prone position.

## Report of Cases

*Case 1*

The patient was a 54-year-old man who was 168 cm in height and weighed 68 kg.

He underwent general anesthesia for surgery via the dorsal approach (Love technique) for herniated lumbar disc. There was no past history or family history of allergic disorders or asthma. Hematological tests, electrocardiography, and lung function test performed on admission were within the normal range.

He was given atropine sulfate (0.5 mg) and hydroxyzine hydrochloride (50 mg) 45 min before anesthesia.

Anesthesia was induced with thiopental sodium (250 mg) and succinylcholine chloride (50 mg). After endotracheal intubation (8.0 mm i.d. tube), anesthesia was maintained by the inhalation of an $O_2/N_2O$ (2L/2L) mixture containing 1.5% halothane. The halothane concentration was later changed to 1.0%.

During this period, the clinical condition was normal. About 20 min after the induction of anesthesia, the patient was laid on a MacKay frame in the prone position.

About 10 min after starting the operation, the resistance of the bag used for controlled ventilation began to increase. Within a few minutes, spontaneous respiration with a long expiratory phase appeared and it became impossible to continue controlled ventilation via the breathing bag. Wheezing characteristic of bronchospasm was heard initially during expiration, and then developed during both expiration and inspiration. This soud became loud enough to be heard without a stethoscope.

As the wheezing indicated bronchospasm, several measures were taken, including adjustment of the cuff pressure of the endotracheal tube and relocation of the neck. Despite these measures, the bronchospasm became worse. As the operation had been started in the prone position, withdrawal and re-insertion of the endotracheal tube was not tried.

Inhalation of halothane was discontinued, and two intravenous doses of aminophylline (250 mg) and an

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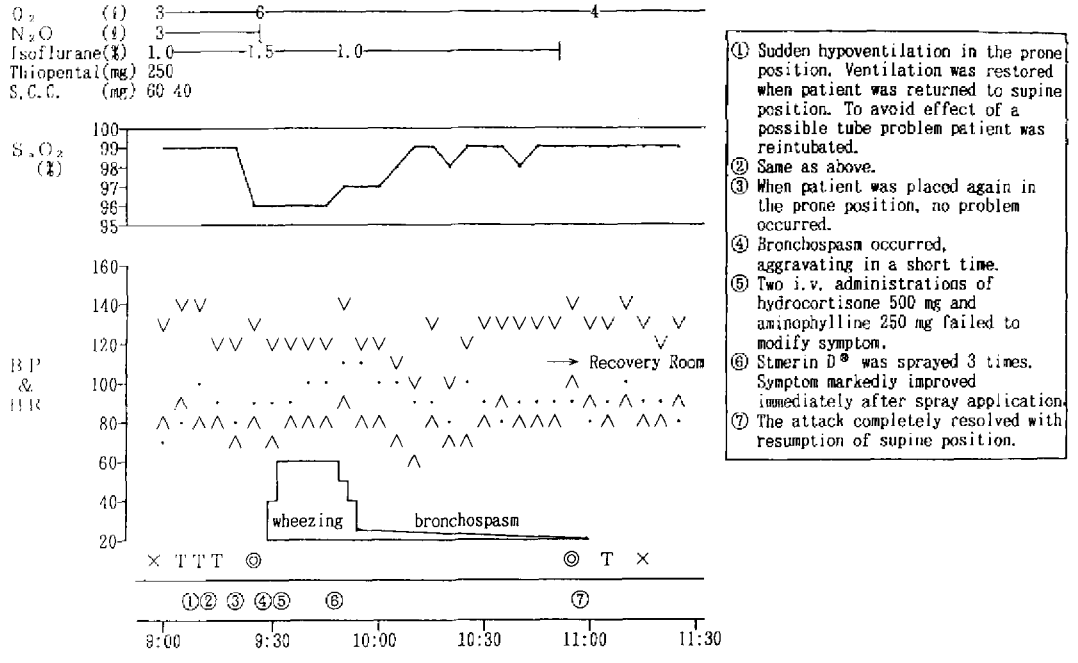


**Fig. 1.** Clinical course of Case 2

intravenous dose of hydrocortisone sodium succinate (1000 mg) were administered. Despite these measures, the bronchospasm showed no improvement. The $Pa_{CO_2}$ was high (62 mmHg), but the $Pa_{O_2}$ was normal (249 mmHg). While bronchospasm was present, no significant hemodynamic changes and only slight increases in blood pressure and pulse rate were noted.

A commercial aerosol preparation for asthma which contains isoproterenol and dexamethasone (Stmerin D®) was administered in two doses via the endotracheal tube. This treatment caused marked improvement of the bronchospasm and elimination of wheezing. Adequate ventilation using the breathing bag was again possible, and the operation was continued.

*Case 2 (fig.)*

The patient was a 37-year-old man who was 165 cm in height and weighed 73 kg. Both the past history and the family history were unremarkable. Preoperative examination revealed no abnormal findings and no allergic predisposition.

The patient underwent general anesthesia for lumbar disc herniation using the Love technique.

0.5 mg of atropine sulfate and 25 mg of hydroxyzine hydrochloride was administered 45 min prior to the induction of anesthesia.

Anesthesia was induced with thiopental sodium (250 mg) and succinylcholine chloride (60 mg). After endotracheal intubation (8.0 mm i.d. tube), anesthesia was maintained by the inhalation of an $O_2/N_2O$ (3L/3L) mixture containing 1.0% isoflurane.

After the hemodynamics were stabilized under controlled ventilation, the patient was laid on a MacKay frame in the prone position. Immediately, the resistance of the breathing bag increased so much that ventilation became impossible. The state was not improved by measures such as changing the angle of the neck or the depth and cuff pressure of the endotracheal tube.

When the patient was returned to the supine position, easy ventilation was restored without any specific treatment.

Since this finding suggested that there might have been something wrong with the endotracheal tube, re-intubation was performed with a new tube. As the patient's condition remained stable after re-intubation, he was again laid in the prone position. However, the same type of ventilatory insufficiency re-appeared. He was returned to the supine position, which again restored adequate ventilation without any specific treatment. The breath sounds were also normal. The endotracheal tube was again replaced with a new one and he was observed for a while. As no abnormal findings occurred, he was returned to the prone position and this time no respiratory disorders were observed. Throughout all these procedure, the $Sa_{O_2}$ remained constant at 99%. It was then decided to start the operation.

About 4 min after the start of surgery, the breathing bag used for ventilation became harder to squeeze and wheezing was heard.

The bronchospasm became rapidly worse, and both the expiratory and inspiratory phases were prolonged. The $Sa_{O_2}$ level decreased to 96%. Inhalation of $N_2O$ was discontinued, and the $O_2$ flow rate was increased to $6 L \cdot min^{-1}$. The concentration of isoflurane was increased to 1.5%. The patient was then given two intravenous bolus doses of hydrocortisone sodium chloride (500 mg) and aminophylline (250 mg), and the depth of the endotracheal tube and the cuff pressure were adjusted. However, all these measures failed to improve the bronchospasm.

Despite this, the $Sa_{O_2}$ ramained at 96% or more and no significant hemodynamic changes were observed.

About 20 min after the development of bronchospasm, an aerosol preparation for asthma (Stmerin D®) was administered in 3 doses via the endotracheal tube. This treatment produced marked improvement of the bronchospasm as in Case 1. Adequate ventilation was almost completey restored and the $Sa_{O_2}$ improved gradually althought mild to moderate wheezing remained.

Slight bronchospasm continued throughout the operation, but disappeared after the completion of surgery when the patient was returned to the supine position.

## Discussion

The incidence of bronchospasm under general anesthesia is higher in patients with a history of respiratory disease such as asthma or a predisposition to allergic disorders (i.e., individuals with increased parasymathetic tone). The incidence is also increased by medical or mechanical treatments that stimulate the parasympathetic nervous system and increase its tone.

Previous case reports on bronchospasm under general anesthesia have clearly noted that those patients had a history of asthma or other allergic disorders[1–5]. Bronchospasm has also been reported in patients with no allergic predisposition such as our cases, but the symptom was attributed to the use of protamine for extracorporeal circulation[6] and to histamine release induced by visceral traction[7].

The two patients reported here had no history of allergic disorders. They showed no abnormal findings in the preoperative lung function tests and they were not treated with any specific agent during anesthesia.

It is possible that the thoracic compliance and the pressures in the respiratory tract may change during general anesthesia in the prone position. However, no published reports have confirmed this possibility and the only

report we could find on respiratory dynamics during anesthesia suggested the absence of any obvious changes[8].

These two patients were unlikely to have high parasympathetic tone, because their hemodynamic and respiratory parameters (blood pressure, pulse rate, and $Sa_{O_2}$) as well as the amount of airway secretions, remained normal until bronchospasm developed.

In both patients, bronchospasm developed within 10 min of the start of operation, a time when dissection of muscles on both sides of the vertebral arch had been completed and a retractor for laminectomy had been inserted. At this time, intense force was suddenly applied to the muscles of the lower back. It has been reported that hypotension and bradycardia are sometimes provoked by stimulation of the $A\beta$ and $A\delta$ afferent fibers from the muscles, representing a noxious somatic sensation-autonomic reflex[9]. The bronchospasm reported here might have been related to such a reflex mechanism.

The standard measures for treating bronchospasm under anesthesia are adjustment of the depth of the endotracheal tube and cuff pressure, combined with medications including intravenous aminophylline, steroids, and lidocaine as well as continuous infusion of isoproterenol or epinephrine. The bronchospasm in our patients responded well to a commercial aerosol preparation for asthma that contained isoproterenol, atropine methyl bromide, and dexamethasone administered via the endotracheal tube.

This report deals with two patients who had no history of asthma and no allergic predisposition but developed a sudden onset of bronchospasm during operation in the prone position under general anesthesia. The endotracheal administration of a commercial aerosol preparation for asthma should probably be tried if other measures fail to improve bronchospasm under general anesthesia with endotracheal intubation.

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