# Tension Pneumothorax Resulting in Cardiac Arrest during Emergency Tracheotomy under Transtracheal Jet Ventilation

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Transtracheal jet ventilation (TTJV) has been popularized as a useful technique in acute upper airway obstruction. But its complications in emergency settings could be sometimes fatal. This report summarizes the course of a patient who suffered bilateral tension pneumothorax resulting in cardiac arrest during emergency tracheotomy under high frequency jet ventilation.

## Case Report

A 48-year-old man was admitted because of severe dyspnea. The patient was in a healthy condition until one week before, when he noticed dyspnea on exertion. He had a history of thyroid papillary adenocarcinoma for which he had undergone right thyroid lobectomy ago. After that, tumorectomy for local recurrence has been performed for three times. On admission, he was severely dyspneic with peripheral cyanosis and remarkable stridor. Arterial blood gas analysis in room air showed compensated respiratory acidosis with pH of 7.41, Pa<sub>O2</sub> of 54 mmHg, and Pa<sub>CO2</sub> of 57 mmHg. His hemoglobin was  $14.9 \text{ g} \cdot \text{dl}^{-1}$ . Chest revealed severe tracheal stenosis

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distal to cricoid cartilage. Because of this stenosis, intubation was considered to be difficult in case of emergency. The patient was scheduled for tracheostomy under local analgesia. No premedication was given. Electrocardiogram (EKG), blood pressure and pulse oximetry were monitored. Preoperative blood pressure was 132/93 mmHg and heart rate was 93 beats·min<sup>-1</sup>. Surgery started under local infiltration with 1%lidocaine. The identification of trachea was difficult due to tumor invasion. Blood pressure and heart rate rose gradually to 234/148 mmHg and 133 beats·min<sup>-1</sup> respectively until 45 min after the start of operation. Oxygen saturation (Sa<sub>O2</sub>) was stable at 98-100% during the first 45 min. When the surgeons began to manipulate around the trachea, Sa<sub>O2</sub> suddenly started to decline and the patient's ventilatory effort was severely disturbed. When Sa<sub>O<sub>2</sub></sub> fell to 35%, 14G iv teflon catheter (Angiocath, Deseret Medical Inc., Sandy, Utah, USA) was inserted into the trachea at approximately 4 cm above the carina, and MERA HFO jet ventilator (AE-20, Senko Ika Kogyo, Inc., Tokyo, Japan) was connected to the catheter and ventilation was maintained to deliver 100 b.p.m. at a driving pressure of 15 psi. After initiating TTJV, oxygen saturation rapidly returned to 100%, but the patient was deeply comatose. Tracheostomy was performed under TTJV and Sa<sub>O<sub>2</sub></sub> remained at above 90%.

His blood pressure started to fall 60 min after the start of operation, and at 78 min heart rate declined to 50 beats·min<sup>-1</sup>. EKG showed multiple ventricular premature contractions. The patient's face became severely swollen and carotid and femoral arteries were not palpable. Tension pneumothorax was suspected by sudden cardiovascular collapse and impaired venous return. Bilateral pleural cavities were immediately decompressed with 14G iv catheter, and 2 mg of epinephrine was administered intravenously. Pulse became palpable and EKG revealed sinus tachycardia at a rate of 165 beats·min<sup>-1</sup>, but facial swelling did not improve. After placement of chest tubes, a 4.0 mm endotracheal tube (ETT) was inserted into the trachea from the tracheostomy site and the patient was manually ventilated using 100% oxygen. Arterial blood gas data after resuscitation showed respiratory acidosis with pH of 7.18, PaO<sub>2</sub> of 85 mmHg and Pa<sub>CO<sub>2</sub></sub> of 120 mmHg. In order to replace the small-bore ETT, angiographic guidewire was inserted through the 4.0 mm tube, then the tube was removed and a 4.5 mm ETT was threaded over the guidewire. In spite of vigorous manual ventilation, Pa<sub>CO2</sub> remained at 90-100 mmHg until a 7.5 mm ETT was finally placed in a similar fashion. The patient's blood pressure fell to 100/50 mmHg after manual ventilation was begun. As he became excited, 80 mg of ketamine was given intravenously for sedation. After a 7.5 mm ETT was inserted into the trachea, manual ventilation became easier and arterial blood gas remarkably improved (pH 7.41, Pa<sub>O2</sub> 380 mmHg and Pa<sub>CO<sub>2</sub></sub> 46 mmHg). He recovered from unconsciousness and showed no neurological deficit. Facial swelling improved but postoperative chest X-ray showed mediastinal emphysema. He was admitted to intensive care unit and was mechanically ventilated. He was weaned from artificial ventilation on the following day. The patient suffered from right lower lobe pneumonia postoperatively. Three weeks later, the patient underwent laryngotomy and T-tube insertion under general anesthesia. Subsequent recovery was



**Fig. 1.** Preoperative chest X-ray film showing severe tracheal stenosis due to compression of thyroid tumor. (Arrow)

uneventful.

### Discussion

Transtracheal jet ventilation (TTJV) is accepted as an alternative method when conventional airway/ventilation management is unsuccessful<sup>1</sup>. There are many case reports in which TTJV was effective in the anesthetic management for difficult intubation<sup>2,3</sup>, and for acute upper airway obstruction<sup>4</sup>.

Cricothyroid membrane is generally suitable for insertion of large-bore iv catheter because (1) its anatomical position is relatively easy to identify, (2) it has poor vascular supply with low risk of hemorrhage, (3) tracheal lumen is well maintained by the ring-shaped cricoid cartilage. Because this patient's stenotic lesion was located far beyond the cricoid cartilage as can be seen in the chest X-ray film (fig. 1), it was impossible to insert iv catheter and to initiate elective TTJV preoperatively.

This patient could maintain ventilation on arrival in the operating room, and preoperative oxygen saturation was 100%. He was completely obstructed due to operative manipulation and bleeding into tracheal lumen. When oxygen saturation rapidly fell, oxygenation immediately improved by application of TTJV. But bilateral tension pneumothorax resulted in circulatory collapse.

The incidence of complications of TTJV in acute respiratory distress is much higher than in elective condition<sup>5</sup>. These complications include hemorrhage, esophageal perforation, air embolism, and barotrauma. There are mainly two mechanisms of barotrauma due to TTJV, which are (1) direct airway mucosal damage from high pressure airflow, (2) inadequate orifice and time for exhalation which causes an increase in airway pressure, resulting in alveolar rupture. Although TTJV was provided at a rate of 100 b.p.m., lower frequency might have prevented this possible fatal complication. Egol et al. gave the following suggestions to decrease the risk of barotrauma<sup>6</sup>: (1) Avoid direct pressure against mucosa. (2) Secure catheter to minimize risk of migration. (3) Use catheters with side holes. (4) Use minimal effective driving pressure.

The commonly used iv catheters for TTJV are thin-walled, easily compressed and prone to kinking. Boyce et al. have recommended the use of vessel dilator to provide a secure airway during TTJV<sup>7</sup>. We think there is a risk of endobronchial insertion of long vessel dilator when the tracheal entry site is located near the carina, such as in the case presented. Transtracheal catheters are easily obstructed especially when surgical manipulation is done around the catheter. Craft et al. advocated the use of a second catheter for airway pressure monitoring during TTJV<sup>8</sup>. Although we agree with their report, insertion of another catheter in the small surgical field is not practical. We consider it is important to ensure unobstructed expiration from the patient's stenotic airway in order to prevent serious tension pneumothorax.

Zornow et al. examined three different techniques of transtracheal ventilation to reverse hypoxia and hypercapnea<sup>9</sup>. They reported that both jet and oxygen flush (from fresh gas outlet of anesthesia machine) modes were able to sustain adequate oxygenation/ventilation, whereas circle (standard anesthesia circuit with vigorous man-

ual ventilation) mode failed to decrease  $Pa_{CO_2}$ . In this case, adequate elimination of carbon dioxide was impossible by using manual ventilation with a small-bore ETT. It is well known that acute normalization of arterial carbon dioxide in a patient who has long been hypoxic and hypercapnic results in profound hypotension. This patient also required to be given an inotropic agent of dopamine after establishment of adequate ventilation.

In conclusion, we presented a patient who suffered bilateral tension pneumothorax as a complication of TTJV, which resulted in cardiac arrest. The patient was successfully resuscitated without neurological deficit. We emphasize the necessity to ensure unobstructed exhalation when TTJV is used in the emergency situation.

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