Anesthesia with nontracheal intubation in thoracic surgery

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ABSTRACT

Objective: To study one-lung respiration during VATS wedge resection of bullae and pulmonary nodules with nontracheal intubation, and to explore the changes of vital signs when patients return to two-lung ventilation.

Methods: Twenty-two patients with normal cardiopulmonary function and absence of contraindications to epidural anesthesia were included in this study. VATS wedge resection of bullae or pulmonary nodules was performed. 0.5% Ropivacain was administrated for epidural anesthesia (T8-9), and 2 mL of 2% lidocaine was used for local anesthetic block of the intrathoracic vagus nerves. The BIS value was maintained between 50 and 70 by target-controlled infusion of propofol and remifentanil. Electrocardiogram (ECG), heart rate (HR), blood pressure (Bp), pulse oxygen saturation (SpO_2), respiratory rate (RR), bispectral index (BIS) and urine volume were monitored.

Results: None patients were converted to endotracheal intubation during anesthesia. MAP and SpO_2 after wound disclosure were stable ($P>0.05$), level of CVP significantly elevated, HR and RR increased ($P<0.05$), $PaCO_2$ increased gradually while PaO_2 remained stable. Fifteen minutes after wound closure, MAP, RR and SpO_2 returned to their pre-anesthesia levels, PH value gradually recovered, $PaCO_2$ tended to decrease and returned to normal one hour after wound closure. Physical agitation occurred in one case due to inadequate epidural anesthesia during skin incision. Cough before intrathoracic vagal blockade was noted in two cases (9.1%) because of lobe traction.

Conclusions: Nontracheal intubation is feasible in VATS wedge resection of bullae and pulmonary nodules. The patients are with stable intraoperative vital signs and none experiences hypoxemia; intraoperative hypercapnia is tolerable and transient, which can be improved quickly when bilateral lungs resume spontaneous respiration.

KEY WORDS Anesthesia; nontracheal intubation; thoracic surgery

J Thorac Dis 2012;4(2):126-130. DOI: 10.3978/j.issn.2072-1439.2012.03.10

Introduction

As lung separation techniques and anesthesia management advances in thoracic surgery, video-assisted thoracic surgery rapidly develops as well. As a result, the operating time and surgical trauma in wedge resections of bullae and pulmonary nodules have been significantly reduced. Since Pompe reported awake VATS wedge resection of solitary pulmonary nodules under thoracic epidural anesthesia in 2004, thoracic sympathectomy, lung metastases resection, pulmonary nodule resection, pulmonary bulla resection, biopsy of lung and pleura, resection of mediastinum nodules and pulmonary lobectomy in a similar anesthetic manner have continually been reported (1-7).

Although VATS wedge resection of bullae and pulmonary nodules with nontracheal intubation has been proven to be feasible, various factors, such as spontaneous respiration with one-lung ventilation during operation, intercostal muscle damage induced by thoracic epidural anesthesia intravenous administration of sedatives and analgesics, and operative position, may aggravate respiratory impairment, causing hypoxemia and hypercapnia, or even serious complication as well.

In addition, as for VATS operations under anesthesia with nontracheal intubation, it is not clear how the vital signs, such

No potential conflict of interest.

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Submitted Mar 06, 2012. Accepted for publication Mar 13, 2012.
Available at www.jthoracdis.com

ISSN: 2072-1439

as respiration and circulation, will change during one-lung ventilation. Moreover, the trend and time of vital signs recovery after conversion to two-lung ventilation have not been reported. This clinical observation of 22 cases of VATS wedge resection of bullae or pulmonary nodules under anesthesia with nontracheal intubation explored changes of the vital signs during one-lung ventilation and subsequent two-lung ventilation.

Patients and methods

Research design

Anesthesia protocols were audited and approved by the Hospital Ethical Committee. The inclusion criteria for subjects were ASA I-II, age between 18 and 65, BMI <25, Mallampati grade I-II, little airway secretion and absence of epidural puncture contraindication. VATS wedge resections of bullae and pulmonary nodules were performed. Anesthetic protocols were explained to the participants before the informed consent was obtained.

Anesthesia

Patients received intramuscular Midazolam 0.06 mg/kg and Atropine 0.01 mg/kg 30 minutes before anesthesia. Electrocardiogram (ECG), heart rate (HR), blood pressure (Bp), pulse oxygen saturation (SpO_2), respiratory rate (RR) and bispectral index (BIS) and urine volume were continuously monitored after the patients entered the operation room. The thoracic epidural catheter was inserted at the T8-9 interspace, 3 cm towards the head, after intravenous infusion had been established. 2 mL of 2% Lidocaine was injected with the patients in supine position. Five minutes after the injection when no abnormal reaction to the anesthesia was observed, 3 mL of 0.5% Ropivacain was administrated followed by re-injection of another 3 mL 5 minutes later to reach a level of anesthesia between T2 and T10. Target-controlled infusion of Propofol and Remifentanil was started, and the BIS value was maintained between 50 and 70 by adjusting target concentration. During the whole research process, nasopharyngeal airway and face mask were used for oxygen inhalation, with an oxygen flow of 3-5 L/min.

Catheters were inserted via the right internal jugular vein or the right subclavian vein to continuously monitor the central venous pressure (CVP). And catheterization via the radial artery was performed to continuously monitor the invasive blood pressure (IBP). An incision into the chest wall on the operated side caused pulmonary collapse, leading to iatrogenic pneumothorax. Patients received local administration of 2 mL of 2% Lidocain injected under thoracoscopic guidance to achieve local anesthetic block of the intrathoracic vagus nerves. After the pleural cavity was closed and the wound was sutured, a face mask was used to assist the patients in ventilation to inflate the lung tissue. After the target controlled infusion was stopped and the epidural catheter was removed, the patients were transferred to a post anesthesia care unit (PACU).

If SpO_2 gradually decreased below 90% during anesthesia, a face mask was needed to assist ventilation in order to improve systematic oxygenation; if $PaCO_2$ ≥80 mmHg, operation had to be suspended and mechanical ventilation was delivered via a face mask to assist gas exchange. If ventilation could not be improved by the face mask, endotracheal intubation would be resorted.
Vital signs were monitored at pre-anesthesia, before and 15, 30, 45 minutes after wound disclosure as well as 15, 30, 45 minutes after wound closure. At the above time points, arterial blood was simultaneously extracted for blood gas analysis to detect values of pH, PaO_2, $PaCO_2$ and Lac. Operating time, arrhythmia, physical agitation, coughs before and after local anesthetic block of the intrathoracic vagus nerves and the cases transferred to endotracheal intubation were all recorded.

Statistical analysis

Primary outcome measures included values of HR, SpO_2, RR, Bp, CVP and arterial blood gas analysis. Secondary outcome measures included BIS, operating time, physical agitation and coughs. Age, height, weight and BMI were expressed by average value ± standard deviation. Two-sample t-test was used for statistical analyses. All data were analyzed with SPSS 13.0. A P value of <0.05 was considered statistically significant.

Results

From July to December of 2011, 9 VATS resections of bullae and 13 wedge resections of lung nodules were performed under combined anesthesia with epidural block, local block of the thoracic vagus nerve and analgesic sedation.

The general clinical data of the patients were detailed in Table 1. Their average age was 39.18±18.52 years and their average BMI was 20.57±2.35. No arrhythmia was found by ECG monitoring. No patients needed conversion to endotracheal intubation during anesthesia. Physical agitation caused by inadequate epidural anesthesia was noted in one case (4.5%) during skin incision. Cough occurred before local anesthetic block of the vagus nerves in two cases (9.1%), which was caused by stretching of pulmonary lobes when exploring and exposing the vagus nerves, but no cough occurred after completion of the vagus nerve blockade.

The HR and SpO_2 values before wound disclosure were almost the same as those before anesthesia; the BIS value obviously declined by 28.8% (P<0.01); the mean arterial pressure (MAP) slightly declined by 15.4%; the respiration rate decreased by 30.3% (Table 2). The changes of operative indexes after wound disclosure

Table 1. General information of the patients.

Items	Results
Male/Female (n)	14/8
Age (y)	39.18±18.52
Height (m)	1.66±0.09
Weight (kg)	56. 36±7.30
BMI	20.57±2.35
Arrhythmia (n)	0
Conversion to intubation (n)	0
Agitation (n)	1
Cough before intrathoracic vagal blockade (n)	2
Cough after intrathoracic vagal blockade (n)	0
Mean anesthetic duration (min)	143.9±24.5
Mean operative duration (min)	57.5±14.2
Bullectomy	9
Wedge resection of pulmonary lump	13
Location: right upper lobe	8
Right lower lobe	3
Left upper lobe	8
Left lower lobe	3

Table 2. Vital signs before anesthesia and before wound disclosure ($\bar{x}\pm s$).

	Before anesthesia	Before wound disclosure
BIS	92.5±5.6	66.2±8.5*
MAP (mmHg)	84.9±9.5	71.7±15.1*
HR (bpm)	78.9±13.9	75.1±17.7
RR (bpm)	15.8±1.4	11.0±3.3*
SpO_2 (%)	99.2±1.1	99.8±0.5

Compared with those before anesthesia, *P<0.01.

were detailed in Table 3. The MAP and SpO_2 changed slightly (P>0.05) while the CVP rose significantly; the sedation level deepened with gradually decreased BIS value; HR and respiratory rate (P<0.05) gradually increased; acidemia was gradually aggravated with increasing $PaCO_2$ but no hypoxemia occurred after PaO_2 was maintained stable.

Compared with those before anesthesia, the MAP, RR and SpO_2 values 15 minutes after wound closure returned to their pre-anesthesia levels. Patients were still in light sedation, with slightly increased HR and BIS value of 73.4±13.6 (Table 4). Under spontaneous respiration with oxygen inhalation via a nasal tube (2-3 L/min), arterial blood gas analysis showed that PH value gradually recovered and $PaCO_2$ tended to decrease but returned to normal one hour after wound disclosure. The oxygenation index significantly declined 15 minutes after thoracotomy but recovered to that before thoracotomy 30 minutes later. Although the value of Lac after thoracotomy was higher than that before wound disclosure, both Lac values were within the normal range (Table 5).

Discussion

The present study enrolled subjects to undergo VATS bullectomy or lumpectomy which can be accomplished simply and in a short time. In our series, the operation duration was (57.5±14.2) min, and the duration from wound disclosure to closure when the negative pressure restored in the thoracic cavity in all cases did not exceeded 45 minutes. As a result, we did not have a long time to observe the pathophysiologic changes after pneumothorax.

Incisions through the chest wall for VATS are generally made between the 4th and 7th costal interspace, so we chose the T8/9 thoracic interspace as the puncture site to perform thoracic epidural blockade because it could maintain effective analgesia in the operative field. In this study, limb agitation occurred

Table 3. Vital signs and blood gas analyses before and after wound disclosure ($\bar{x}\pm s$).

	Before wound disclosure	15 min after wound disclosure	30 min after wound disclosure
HR (bpm)	75.1±17.7	85.9±16.3*	89.2±14.8**
MAP (mmHg)	71.7±15.1	71.1±12.2	75.5±7.7
CVP (cmH_2O)	8.2±4.1	11.4±5.0	11.0±4.6
SpO_2 (%)	99.8±0.5	99.1±2.7	99.7±0.7
RR (bpm)	11.0±3.3	14.7±4.4*	14.5±5.6*
BIS	66.2±8.5	62.5±13.3**	62.6±9.9**
pH	7.30±0.06	7.23±0.06**	7.25±0.05**
$PaCO_2$ (mmHg)	57.6±10.6	68.1±12	65.7±8.6
PaO_2 (mmHg)	260.7±119.2	241.0±122.6	248.3±121.8
Oxygenation index	411.1±149.9	358.1±172.8	365.9±179.3
HCO_3^- (mmol/L)	27.7±2.1	28.1±1.9	28.4±1.5
BE (mmol/L)	1.10±1.76	0.56±1.83	1.12±1.69
Lac (mmol/L)	0.65±0.44	0.65±0.51	0.52±0.36

Compared with those before wound disclosure, *P<0.05, **P<0.01.

during skin incision due to insufficient epidural anesthesia in one patient, whose operation was then completed after further TCI anesthesia.

Cough reflex is a complicated process of neuro-physiological reflex. The cough center is located in the solitary nucleus over the medulla oblongata area of the brain, associated with respiratory neurons. Cough receptors are located mainly on the posterior wall of trachea, pharynx, and mucosa of bronchus. Receptors above secondary bronchi are sensitive to mechanical stimuli while those below are sensitive to chemical stimuli. Impulses caused by stimuli travel via the vagus nerve to the medulla of the brain and trigger a cough. Two cases in our study coughed during operative exploration and lobe traction before intrathoracic vagal blockade, but none had operation-irritated cough during the whole procedure after local anesthesia with lidocaine over the intrathoracic vagus nerve. This indicates intrathoracic vagal blockade may effectively prevent cough reflex, which is in consistency with another relevant study (7).

While patients maintained spontaneous breathing during anesthesia, the operated lung collapsed after iatrogenic pneumothorax. Moreover, factors related to operation and anesthesia aggravated the impaired respiratory function, mainly as follows: (I) decreased activity of the non-operated thoracic cage due to operative posture related compression; (II) further decreased activity of the thoracic cage caused by impaired intercostal muscle function following thoracic epidural anesthesia; (III) inhibition of the respiratory center caused by any anesthetic, sedative and analgesic agent; (IV) paradoxical breathing due to the collapse and insufficiency of the operated lung; (V) muscle flaccidity over the laryngopharynx in sedation which may produce and accelerate glossoptosis, leading to upper respiratory obstruction and aggravating paradoxical respiration and mediastinal flutter.

We strictly selected subjects with good cardiorespiratory functions and without difficult airway. The patients breathed oxygen through a ventimask during the procedure to maintain a good oxygenation index, with SpO_2 above 95%. When epidural anesthesia worked and TCI analgesia was administered, the breathing slowed down and hypercapnia was observed. When iatrogenic pneumothorax occurred on the operated side, the respiratory rate grew compensatingly and $PaCO_2$ increased continuously, which reached to the peak 15 minutes after pneumothorax but began to relieve slightly 30 minutes later. We consider that the hypercapnia occurring in this procedure is tolerable and has little effect on the hemodynamics. It is believed that the increase of $PaCO_2$ along with the decrease of pH mainly depends on the increasing speed of $PaCO_2$ and functional compensation of the kidney. The side effects and tolerance of hypercarbia are mainly related to the cardiovascular and cerebrovascular status of the patients. Studies have indicated that permissive hypercarbia relieves as well as deteriorates cerebral-ischemia-reperfusion injury in rats. Which role it will play is closely correlated with its severity. In a range of 60-100 mmHg, $PaCO_2$ relieves cerebral-ischemia-reperfusion injury in rats by inhibiting neuron apoptosis while it aggravates cerebral edema induced by cerebral-ischemia-reperfusion injury in a range of 101-120 mmHg (8). Propofol may significantly decrease the intracranial pressure and maintain the balance of cerebral oxygen supply and demand in patients with permissive hypercapnia (9).

Patients breathed with bilateral lungs after wound closure, lung dilatation and thoracic negative-pressure drainage. The arterial blood gas analysis 15 minutes later showed all values returned almost to their levels before wound disclosure. Our results also showed that the hypercapnia during pneumothorax was quickly and effectively improved after operation, particularly one hour later.

Table 4. Vital signs before anesthesia and 15 min after wound closure ($\bar{x}\pm s$).

	Before anesthesia	15 min after wound closure
BIS	92.5±5.6	73.4±13.6**
MAP (mmHg)	84.9±9.5	84.1±11.4
HR (bpm)	78.9±13.9	90.3±12.9*
RR (bpm)	15.8±1.4	15.0±4.3
SpO_2 (%)	99.2±1.1	99.0±2.6

Compared with those before anesthesia, *P<0.05, **P<0.01.

Table 5. Blood gas analyses before wound disclosure, 15, 30 and 60 min after wound closure ($\bar{x}\pm s$).

	Before wound disclosure	15 min after wound closure	30 min after wound closure	60 min after wound closure
pH	7.30±0.06	7.29±0.06	7.32±0.05	7.34±0.03
$PaCO_2$ (mmHg)	57.6±10.6	54.1±11.3	48.9±7.30*	47.7±4.45**
PaO_2 (mmHg)	260.7±119.2	144.1±98.8*	144.1±69.3**	133.8±42.5**
Oxygenation index	411.1±149.9	289.5±141.7**	410.5±117.6	420.7±146.7
HCO_3^- (mmol/L)	27.7±2.1	25.5±2.5**	25.0±2.6**	25.6±2.2**
BE (mmol/L)	1.10±1.76	-1.00±2.41*	-1.19±2.80*	-0.39±2.43
Lac (mmol/L)	0.65±0.44	1.08±1.33	1.13±0.98	0.97±0.63

Compared with those before wound disclosure, *P<0.05, **P<0.01.

The blood pressure before operation was lower than that before anesthesia, which was induced by epidural anesthesia and TCI sedation. Thoracic epidural blockade may significantly influence the thoracic sympathetic nerve system, inducing vasodilatation and decreased blood pressure. The arterial blood pressure maintained basically normal after iatrogenic pneumothorax, with no arrhythmia noted by continuous ECG monitoring, which indicates that the mediastinal flutter has no significant influence on circulation. The increases of heart rate and CVP may be compensations for the slowed down venous return following the disappearance of negative pressure in unilateral thoracic cavity.

In the present study, we demonstrated that VATS wedge resection of bullae and pulmonary nodules with nontracheal intubation are feasible in operations that can be accomplished in a short time. Patients can maintain stable intraoperative physical signs without severe hypoxemia. The intraoperative hypercapnia is tolerable and transient and can be improved quickly when the bilateral lungs resume spontaneous respiration. Further research, however, is still to be further studied to characterize the hypercarbia 30 minutes after pneumothorax and to explore its systematic impacts.

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Cite this article as: Dong Q, Liang L, Li Y, Liu J, Yin W, Chen H, Xu X, Shao W, He J. Anesthesia with nontracheal intubation in thoracic surgery. J Thorac Dis 2012;4(2):126-130. DOI: 10.3978/j.issn.2072-1439.2012.03.10