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ORIGINAL ARTICLE

Compressing the non-dependent lung during one-lung ventilation improves arterial oxygenation, but impairs systemic oxygen delivery by decreasing cardiac output

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Abstract
Purpose We have previously found that compression of the non-dependent lung improves arterial oxygenation during one-lung ventilation (OLV) in patients undergoing esophagectomy. The purpose of this study was to investigate the effects of compression of the non-dependent lung on hemodynamic indices and oxygen delivery using a minimally invasive cardiac output (CO) monitor.
Methods Sixteen consecutive patients undergoing esophagectomy through a right thoracotomy were studied. Under general anesthesia, a left-sided double-lumen tube was placed for OLV, and the dependent lung was mechanically ventilated with a tidal volume of 8 ml kg^{-1} body weight and a fraction of inspiratory oxygen of 0.8 during OLV. CO was monitored continuously using a FloTrac/Vigileo (Edwards Lifesciences) system. Surgeons compressed the non-dependent lung several times during surgery using a lung retractor to improve exposure of the surgical field. The oxygen delivery index was roughly estimated as the product of the cardiac index (CI) and arterial oxygen saturation as monitored by pulse oximetry (Spo_2).
Results Just before non-dependent lung compression, mean (±SD) CI and Spo_2 were 2.6 ± 0.6 L min^{-1} m^{-2} and 95.0 ± 3.9%, respectively. At 1 min after non-dependent lung compression, Spo_2 increased significantly to 97.8 ± 2.2% ($P < 0.05$), but CI decreased significantly to 2.0 ± 0.4 L min^{-1} m^{-2} ($P < 0.05$). The product of CI and Spo_2 at 1 min was significantly lower (192.7 ± 37.3) than baseline levels (250.5 ± 66.3, $P < 0.05$).
Conclusion Although non-dependent lung compression may be a potentially effective measure to treat hypoxemia during OLV, it should be noted that CO and systemic oxygen delivery may be decreased by this maneuver.

Keywords One-lung ventilation · Esophagectomy · Arterial oxygenation · Systemic oxygen delivery · Non-dependent lung

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Introduction

Hypoxemia is a problem in 1–9% of patients during one-lung ventilation (OLV) [1, 2]. Despite treatment with nitric oxide [3], prostaglandin E1 [4], prostaglandin F2 alpha [5], almitrine [6], or different anesthetic drugs [7–10], hypoxemia during OLV can remain a problem.

During the thoracic part of esophagectomy, OLV is used while the non-dependent lung is usually compressed by surgeons using a lung retractor to improve visualization of the surgical field. In our previous study [11], we showed that arterial oxygenation can improve during OLV in some patients undergoing esophagectomy, and that this improvement is partly related to a marked increase in arterial oxygen tension (Pao_2) during compression of the non-dependent lung. One of the limitations of that study was that a pulmonary catheter was not used, and thus cardiac output (CO) and oxygen delivery data were lacking, since we did not consider it ethical to insert a pulmonary catheter solely for the purpose of the study. Thus, it has remained unclear whether oxygen delivery as well as

Pao_2 improves with non-dependent lung compression during OLV in patients undergoing esophagectomy.

The FloTrac sensor and Vigileo monitor (Edwards Lifesciences, Irvine, CA, USA) have recently been introduced as devices that calculate continuous CO using arterial pressure waveforms and individual demographic data to estimate arterial compliance. This FloTrac/Vigileo system is considered to be less invasive than pulmonary artery catheters or other devices that require calibration, since it can measure CO without calibration by connecting to an arterial catheter. Recent research has demonstrated that the cardiac index (CI) obtained by the FloTrac/Vigileo system (version 1.10) showed good intraoperative and postoperative agreement with intermittent pulmonary artery thermodilution CI measurements in patients undergoing coronary artery bypass graft surgery [12].

The main objective of the present study was to analyze the effects of non-dependent lung compression on hemodynamic indices and arterial oxygenation. If arterial oxygenation is improved without deterioration of hemodynamic indices by compression of the non-dependent lung, this surgical maneuver may be an effective measure for treating hypoxemia during OLV and may reduce the fraction of inspiratory oxygen (FIo_2) that is required to avoid hypoxemia during OLV. Furthermore, by analyzing CO and arterial blood gases repeatedly, sequential changes in oxygen delivery were assessed in patients undergoing esophagectomy.

Materials and methods

Sixteen consecutive patients scheduled to undergo esophagectomy using a right thoracotomy and OLV were considered for the study, which was approved by the University Ethics Committee. Written informed consent was obtained from all patients. Patients with pacemakers, history of cardiac arrhythmias, severe peripheral vascular disease, cardiac support (intra-aortic balloon pump), mitral or aortic dysfunction, and intracardiac shunt were excluded.

No premedication was administered before the patients were taken to the operating room. Before induction of general anesthesia, an epidural catheter was inserted at the 6–7th or 7–8th thoracic interspace. General anesthesia was induced with intravenous propofol (2 mg kg^{-1}) and tracheal intubation was facilitated with intravenous vecuronium (0.2 mg kg^{-1}). A left-sided double-lumen tracheal tube (Broncho-Cath®, Mallinckrodt Inc., Argyle, NY, USA) was placed for OLV, with correct positioning being confirmed by auscultation and fiber-optic bronchoscopy. Anesthesia was maintained with sevoflurane at an end-tidal concentration of 1.0–2.5%. Patients were ventilated mechanically at a constant tidal volume (10 ml kg^{-1}), and the respiratory rate was adjusted to maintain end-tidal carbon dioxide pressure at approximately 35 mmHg. The FIo_2 was set at 0.8. After positioning the patient in the left lateral decubitus position, correct positioning of the double-lumen tube was reconfirmed by bronchoscopy. Rectal temperature was measured and kept constant using a warm-water blanket. A bolus dose of 0.3% ropivacaine (8–12 ml) was injected epidurally and continuously administered at 4–8 ml h^{-1}.

A 20-gauge intravascular catheter was inserted into the left radial artery and connected to a Vigileo monitor (software version 1.10) through the FloTrac pressure transducer for less-invasive determination of continuous CO. The Vigileo monitor was connected to a personal computer (ThinkPad X23, IBM, USA) by a USB port, and CO was recorded every 20 s.

OLV was started just before the pleura were opened. The tracheal lumen of the double-lumen tube was simply opened to the atmosphere at the beginning of OLV. The dependent lung was ventilated with a tidal volume of 8 ml kg^{-1} and FIo_2 of 0.8. The respiratory frequency was adjusted to maintain $Paco_2$ at approximately 40 mmHg. If arterial oxygen saturation (Spo_2), monitored by pulse oximetry, decreased below 90% during OLV, tube malposition, circulatory problems, leaks or disconnection were excluded as possible reasons for hypoxemia. If required, FIo_2 was increased to 1.0 and continuous positive airway pressure (CPAP) and/or intermittent positive pressure ventilation [13] were applied to the non-dependent lung and data from these patients were excluded from analysis. Systolic blood pressure was maintained within 20% of the preoperative value by controlling the concentration of sevoflurane, rate of continuous epidural administration, rate of intravenous fluid infusion, or by intravenous administration of inotropic agents (ephedrine or dopamine) or vasodilator (nicardipine), as needed.

To analyze sequential changes of Pao_2, arterial oxygen content (Cao_2), and systemic oxygen delivery (DO_2) during esophagectomy, arterial blood samples were collected at 15 min after the patients were placed in the lateral position [two-lung ventilation (TLV)], and then at 10 min (OLV 10), 60 min (OLV 60) and 120 min (OLV 120) after the start of OLV. Since the non-dependent lung is usually compressed by the surgeons at about 15 min after the pleura are opened at our institution, one of the arterial samples was collected at OLV 10 to gain arterial blood gas data before the compression of the non-dependent lung. Cao_2 and DO_2 were calculated using the following standard equations:

$$\begin{aligned} Cao_2\,(\mathrm{ml/dl}) &= 1.34 \times \text{hemoglobin concentration}\,(\mathrm{g/dl}) \\ &\quad \times \text{arterial oxygen saturation}\,(\%)/100 \\ &\quad + 0.0031 \times Pao_2\,(\mathrm{mmHg}) \end{aligned}$$

$$DO_2\,(\mathrm{ml/min}) = CO\,(\mathrm{l/min}) \times Cao_2\,(\mathrm{ml/dl}) \times 10.$$

The non-dependent lung was gently wrapped in gauze and compressed by the surgeons using a retractor to improve exposure of the surgical field several times during the surgical procedure. Application and release of the retractor were done at the discretion of the surgeon. This maneuver has been routinely used at our institution during esophagectomy in more than 1000 cases over 35 years. To analyze the effects of the application of the lung retractor on hemodynamic parameters and arterial oxygenation, we analyzed the first 3 min of the first application of the lung retractor during the thoracic part of the esophagectomy, as changes in CO and Spo_2 were most dynamic during this period. For 3 min after the first application of the retractor, systolic blood pressure, heart rate (HR), stroke volume (SV), CO, CI, and Spo_2 were recorded every minute. The oxygen delivery index was roughly estimated as the product of CI and Spo_2, as changes in hemoglobin concentration and dissolved oxygen content during the course seemed very small. We did not sample arterial blood and calculate DO_2 during this period because sampling arterial blood requires the interruption of continuous CO monitoring via the FloTrac/Vigileo system. Since application of the retractor is usually accompanied by a decrease in blood pressure, a systolic blood pressure of 70 mmHg or higher was allowed for the first 3 min after application, while it was maintained within 20% of the preoperative value before and after the 3-min observation period. At the end of the thoracic part of esophagectomy, systolic blood pressure, CI, Spo_2 and the product of CI and Spo_2 were recorded for 3 min after the release of the retractor to analyze the effects of the release of the retractor on hemodynamic parameters and arterial oxygenation.

Sequential changes in systolic blood pressure, HR, SV, arterial blood gas data, CO, CI, Cao_2, and DO_2 were analyzed using one-way repeated measures analysis of variance followed by Bonferroni multiple comparison test. Statistical significance was established at the $P < 0.05$ level.

Results

All patients included in the study had ASA physical status I or II. Demographic data of the patients are shown in Table 1. In all patients, anesthesia and surgery proceeded without major complications. Since no patient suffered hypoxemia with a Spo_2 of less than 90%, measures to improve arterial oxygenation (increase in FIo_2, CPAP, intermittent positive pressure ventilation) were not required. The non-dependent lung was compressed at 958 ± 296 s after the pleura were opened. None of the non-dependent lungs were compressed in 10 min after the pleura were opened. The mean (±SD) time periods of the first, second, third and fourth application of the lung retractor were 63.8 ± 20.0, 44.4 ± 44.7, 22.7 ± 26.0, and 72.7 ± 51.4 min, respectively. At OLV 120, the number of patients who had second, third, and fourth applications were 6, 3, and 7, respectively. Ephedrine was administered in 9 patients before the 3-min observation period and in 5 patients after the 3-min observation period, while none required intravenous ephedrine during the 3-min observation period after compression of the non-dependent lung. Dopamine was intravenously administered at 3–5 μg kg^{-1} min^{-1} in 8 patients before the non-dependent lung compression to keep systolic blood pressure within 20% of the preoperative value. The speed of continuous dopamine was not changed during the 3-min observation period.

Table 1 Demographic data

	Patients ($n = 16$)
Gender (M/F)	15/1
Age (years)	65.3 ± 10.2
Height (cm)	164.3 ± 7.1
Weight (kg)	59.4 ± 7.6
%VC (%)	109.2 ± 21.1
FEV 1.0% (%)	73.9 ± 8.9
[a]Estimated blood loss (ml)	336 ± 142

Data are presented as mean ± SD

%VC, vital capacity (VC)/predicted VC ratio

FEV 1.0%, forced expiratory volume in 1 s (FEV 1)/forced VC ratio

[a] Estimated blood loss during the thoracic part of esophagectomy

Systolic blood pressure, HR, CO and CI were not significantly different at different time points during the thoracic part of esophagectomy (Table 2). Pao_2 and arterial oxygen saturation were significantly decreased after the start of OLV, with significant improvements at OLV 120 compared to OLV 10. Cao_2 at OLV 10 and OLV 120 was significantly lower than that at TLV; however, DO_2 did not show any significant difference during the thoracic part of esophagectomy.

After the non-dependent lung was compressed, systolic blood pressure and CI significantly decreased at 1 min (83 ± 11 mmHg and 2.0 ± 0.4 L min^{-1} m^{-2}, respectively) and 2 min (82 ± 15 mmHg and 2.1 ± 0.5 L min^{-1} m^{-2}, respectively), compared with just before compression (99 ± 18 mmHg and 2.6 ± 0.6 L min^{-1} m^{-2}, respectively). In contrast, Spo_2 at 1 min (97.8 ± 2.2%) was significantly higher than just before compression (95.0 ± 3.9%). The product of CI and Spo_2 significantly decreased at 1 min after compression (193 ± 37 L min^{-1} m^{-2} × %) compared with just before compression (251 ± 66 L min^{-1} m^{-2} × %). There were no significant changes in HR and SV at different

Table 2 Sequential changes in hemodynamics, arterial blood gases, and other variables related to arterial oxygenation during the thoracic part of esophagectomy

	TLV	OLV 0	OLV 10	OLV 60	OLV 120
Systolic blood pressure (mmHg)	110 ± 18	129 ± 25	124 ± 33	117 ± 18	106 ± 15
Heart rate (bpm)	63 ± 10	68 ± 12	72 ± 15	76 ± 17	80 ± 14
Cardiac output (L/min)	4.2 ± 0.8	4.9 ± 0.8	4.7 ± 1.0	4.6 ± 0.5	4.4 ± 0.9
Cardiac index (L/min/m^2)	2.6 ± 0.6	3.0 ± 0.6	2.9 ± 0.7	2.8 ± 0.4	2.7 ± 0.5
pH	7.41 ± 0.04		7.41 ± 0.04	7.41 ± 0.05	7.41 ± 0.03
Pao_2 (mmHg)	392 ± 61		98 ± 44 **	129 ± 52 **	209 ± 78 **,++,§§
$Paco_2$ (mmHg)	42 ± 5		41 ± 5	39 ± 4	40 ± 4
Arterial oxygen saturation (%)	99.6 ± 0.2		95.2 ± 3.2 **	97.3 ± 1.9 **,+	98.8 ± 0.7++
Hemoglobin concentration (g/dl)	12.0 ± 1.1		12.1 ± 1.1	12.0 ± 1.1	11.3 ± 1.2
Oxygen content (ml/dl)	17.3 ± 1.4		15.8 ± 1.3*	16.0 ± 1.3	15.6 ± 1.6 *
Oxygen delivery (ml/min)	728 ± 128		730 ± 158	729 ± 84	681 ± 152

Data are expressed in mean ± SD

TLV, two-lung ventilation; *OLV 0*, *OLV 10*, *OLV 60*, *OLV 120*, 0, 10, 60, 120 min after the commencement of one-lung ventilation

* $P < 0.05$ versus TLV

** $P < 0.01$ versus TLV

+ $P < 0.05$ versus OLV10

++ $P < 0.01$ versus OLV10

§§ $P < 0.01$ versus OLV60

time points during the 3-min observation period (Fig. 1). SV tended to decrease at 1 min of the non-dependent lung compression compared with baseline; however, the change in SV did not reach statistical significance ($P = 0.054$).

Hemodynamic data and arterial oxygenation were analyzed at the release of the retractor in 13 patients. Three patients were excluded because surgeons asked the anesthesiologists to inflate the non-dependent lung immediately after the release of the retractor. There was no significant change in systolic blood pressure, CI, Spo_2, or the product of CI and Spo_2 (Fig. 2).

Discussion

We assessed the effects of non-dependent lung compression on hemodynamic indices as well as arterial oxygenation during OLV in patients undergoing esophagectomy. Pao_2 significantly improved with time during 2 h of OLV, as was observed in our previous study, but CO and DO_2 did not change significantly. No improvement in oxygen content was observed during OLV, possibly because hemoglobin concentration tended to decrease with time during the surgical procedure.

Although we advocated that compression of the non-dependent lung during OLV may be a potentially useful measure to treat hypoxemia in our previous study [11], the present study shows that oxygenation seems to improve with non-dependent lung compression at the expense of CO. Although the mechanisms behind the decrease in CO that accompanies non-dependent lung compression are unclear, there are three possible explanations. First, the heart may have been directly compressed by the lung retractor, thus impairing cardiac performance. Second, the inferior vena cava was compressed by the lung retractor, thus decreasing venous return to the right atrium. Finally, since the right lung was compressed by the lung retractor, the afterload of the right ventricle was augmented, leading to a decrease in the right ventricular stroke volume. Although CO and blood pressure decreased when the non-dependent lung was compressed, its effects on hemodynamic parameters were transient in the present study.

Since there are many factors that influence arterial oxygenation during OLV, we assume that the mechanisms behind the improvement in arterial oxygenation by the non-dependent lung compression are multifactorial. We assume that the most likely explanation of changes in arterial oxygenation after lung retraction application is that blood flow to the non-dependent lung is redirected to the dependent lung by physical compression and kinking of the lung vessels, although it is difficult to prove this idea. The effects of the release of the lung retractor on hemodynamic parameters and arterial oxygenation were negligible in the present study. However, it should be noted that arterial oxygenation may be impaired after the release of the retractor, since our previous study showed that there was a mild drop in Pao_2

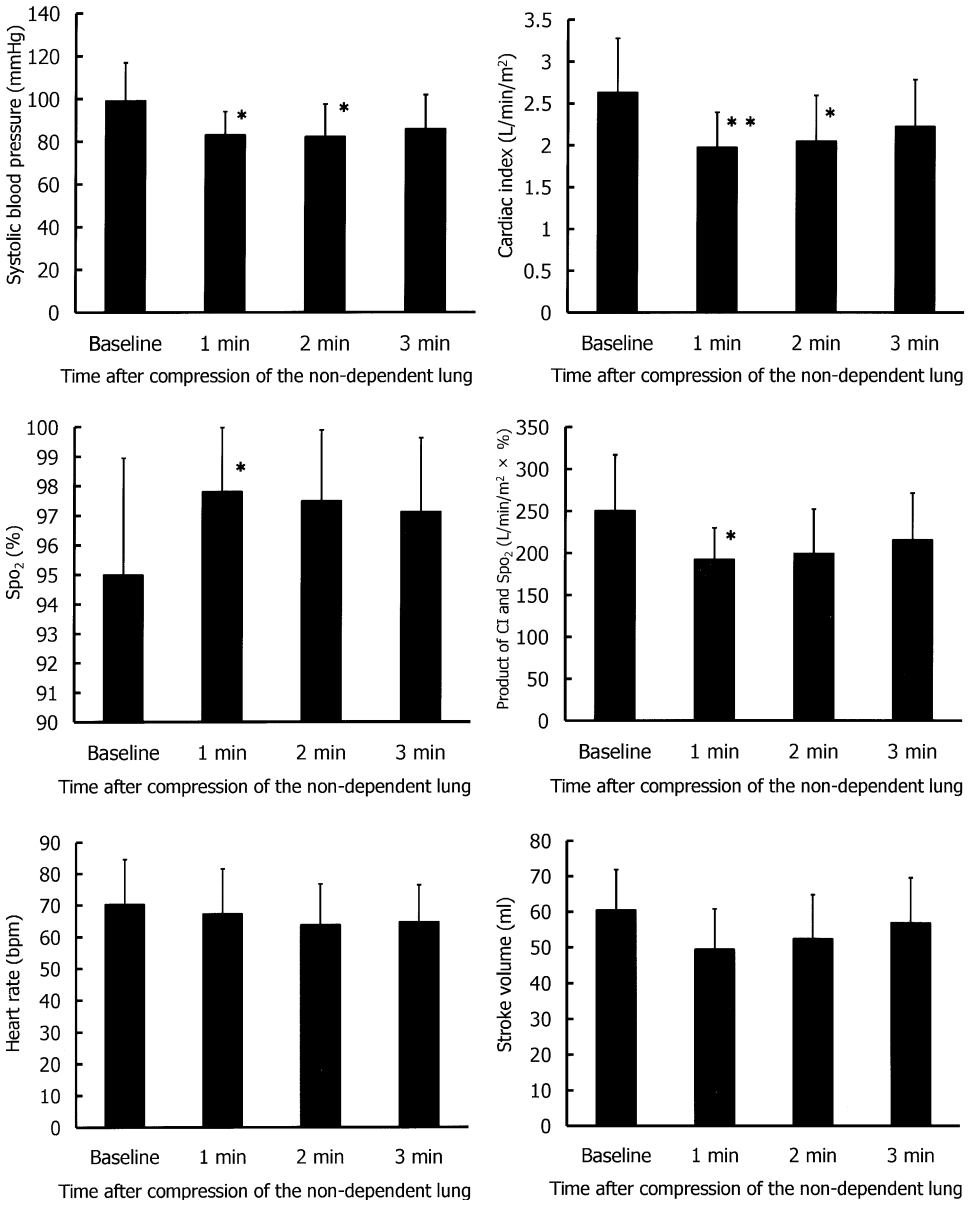

Fig. 1 Sequential changes in systolic blood pressure, cardiac index, Spo_2, the product of CI and Spo_2, heart rate, and stroke volume just before (*baseline*) and at *1*, *2*, and *3* min after non-dependent lung compression. *Spo_2*, arterial oxygen saturation monitored by pulse oximetry; *CI*, cardiac index. $^{**}P < 0.01$ versus baseline, $^{*}P < 0.05$ versus baseline

(approximately 43 mmHg on average) in 2 min after decompression of the non-dependent lung by using continuous intra-arterial blood gas monitoring [11].

Since oxygenation is affected by several factors during OLV, such as the time course of hypoxic pulmonary vasoconstriction [14], CO [15–17], and epidural anesthesia [18], it is very difficult to analyze the effects of the non-dependent lung compression on the gradual improvement in arterial oxygenation. We previously found that improvement in arterial oxygenation during OLV was partly related to a marked increase in Pao_2 during the second compression of the non-dependent lung [11]. Pao_2 markedly improved during the surgical procedure in patients whose increase in Pao_2 in response to the second compression of the non-dependent lung was more than 100 mmHg [11].

To both improve arterial oxygenation and maintain CO and DO_2, increasing CO by inotropic agents in combination with non-dependent lung compression may be useful. Two human studies have demonstrated an increase in CO by approximately 25% using dobutamine 5 μg kg^{-1} min^{-1}, leading to significant improvements in arterial oxygenation during OLV [17, 19]. The increase in CO may not increase the shunt flow via the non-dependent lung during OLV due to the fact that the non-dependent lung is compressed by the lung retractor and the non-dependent lung vessels are most likely kinked. However, since increasing CO to very high values can cause deterioration in arterial oxygenation [20, 21], CO monitoring is recommended when inotropic agents are administered for the purpose of improving arterial oxygenation during OLV.

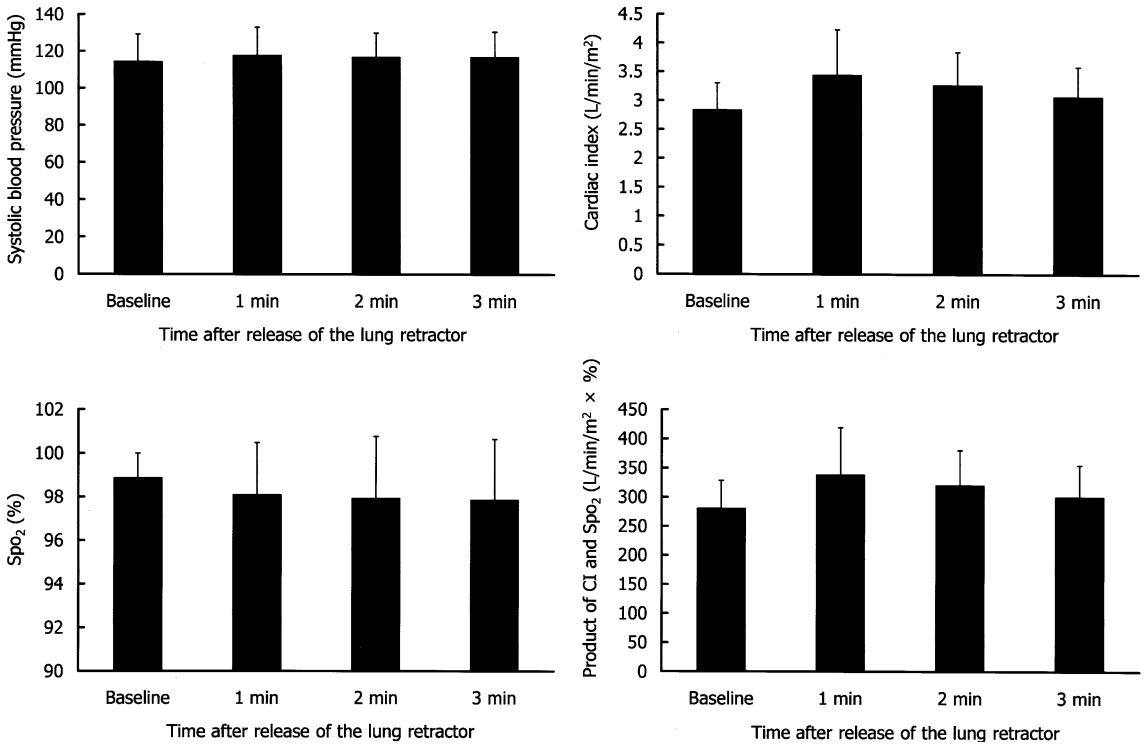

Fig. 2 Sequential changes in systolic blood pressure, cardiac index, Spo_2, the product of CI and Spo_2 just before (*baseline*) and at *1*, *2*, and *3* min after the final release of the retractor. *Spo_2*, arterial oxygen saturation monitored by pulse oximetry; *CI*, cardiac index. There was no significant change in any of these parameters at different time points

There is a limitation in the present study. As far as we know, there has been no published report that has evaluated the accuracy of the FloTrac/Vigileo system in patients during OLV. This system should be validated in clinical settings of thoracic anesthesia in the future.

In conclusion, compression of the non-dependent lung during OLV improves arterial oxygenation, but may impair both DO_2 and CO. When the non-dependent lung is compressed, the heart and inferior vena cava may be compressed simultaneously or SV may decrease due to an increase in right ventricular afterload. Although we previously considered non-dependent lung compression as a potentially effective measure to treat hypoxemia and to reduce the FIo_2 during OLV, which is required to avoid hypoxemia, it should be noted that CO and DO_2 may be decreased by this maneuver.

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