Effect of head-down tilt on intrapulmonary shunt and oxygenation during one-lung ventilation in the lateral decubitus position

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Abstract

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Background: During one lung ventilation (OLV), surgical positions significantly affect deterioration of oxygenation and lateral decubitus position (LDP) is superior in preventing dangerous hypoxemia than supine position. However, additional head-down tilt causes more compression of the dependent ventilated lung by the abdominal contents and may result in dangerous hypoxemia as in supine position. Therefore, we evaluated the effect of head-down tilt on intrapulmonary shunt (Qs/Qt) and oxygenation during OLV in the LDP, in a prospective, randomized, controlled trial. **Methods**: Thirty-four patients requiring OLV for thoracic surgery were randomly allocated to either control (n = 17, CG) or head-down tilt group (n = 17, HG). Hemodynamic and respiratory variables were measured 15 min after OLV in the LDP (baseline), 5 and 10 min after 10° head-down tilt (T5 and T10, respectively), and 10 min after the patient was returned to horizontal position (TH) in the HG, while the CG remained horizontal. **Results**: In the HG, cardiac filling pressures were increased after head-down tilt without any changes in cardiac index. Percent change of Qs/Qt to baseline value was significantly increased at T10 and TH in the HG. Percent change of PaO_2 to baseline value was significantly decreased at T5, T10 and TH in the HG, while it was decreased only at TH in the CG. **Conclusions:** Head-down tilt during OLV in the LDP caused significant increase in Qs/Qt and decrease in percent change of PaO_2.

These results were accompanied by increases in cardiac filling pressures without any increase in cardiac output.

Keywords: one lung ventilation, intrapulmonary shunt, head-down tilt

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Ⅰ. INTRODUCTION

During thoracic surgeries requiring one-lung ventilation (OLV), deterioration of oxygenation is of major concern. Arterial oxygen tension (PaO_2) decreases progressively with time toward a steady value after the start of OLV that corresponds to the pulmonary shunt of deoxygenated blood through the nonventilated lung and to the ratio of ventilation and perfusion (V/Q) in the ventilated lung.[1,2] Surgical positions considerably influence the deterioration speed and the nadir value of PaO_2 after the start of OLV.[3] In the supine position, dangerous hypoxemia (PaO_2 < 60 mm Hg) occurred even after 10 min of OLV and lateral decubitus position (LDP) was superior in preventing the occurrence of dangerous hypoxemia.[3] In thoracic surgery with OLV in the LDP, head-down tilt is sometimes necessary to optimize surgical exposure, especially in lower lobectomy by video-assisted thoracoscopic surgery (VATS) and posterior mediastinum surgery. However, this causes more compression of the dependent ventilated lung by the abdominal contents against the paralyzed diaphragm and may result in increased intrapulmonary shunt (Qs/Qt), and subsequently in dangerous hypoxemia as in supine position. Also, head-down tilt is often considered as the first step to treat hemodynamically unstable patients when hypovolemia is suspected in clinical practice. However, the effects of this maneuver on cardiopulmonary performance remains controversial,[4-12] especially in the LDP. Therefore, we evaluated the effect of head-down tilt on Qs/Qt and oxygenation, and hemodynamics during OLV in the LDP in a prospective, randomized, controlled trial.

Ⅱ. MATERIALS AND METHODS

After approval of the institutional review board and patients' consent, 34 patients scheduled for open thoracotomy or VATS requiring OLV under general anesthesia were included. Patients were randomly allocated to either 10° head-down tilt group (HG) or control group (CG) by a computerized randomization table. Patients with coronary artery occlusive disease, high intraocular pressure, cerebrovascular disease were excluded. Patients with preoperative forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) below 80 % of predicted value were also excluded.

Upon arrival at the operating room, standard monitoring devices were applied. Anesthesia was induced with 5 mg/kg of thiopental, and 2 μg/kg of fentanyl. Endobronchial intubation with a left-sided, double-lumen tube (DLT; Robertshaw tube, Mallinckrodt Medical Inc., Athlone, Ireland) was facilitated with 0.9 mg/kg of rocuronium and the position of DLT was verified with a fiberoptic bronchoscope. After induction of anesthesia, a radial artery catheter was placed and a pulmonary artery catheter (PAC, Swan-Ganz CCOmbo® CCO/SvO_2, Edwards Lifesciences LLC, Irvine, CA, USA) was inserted via the right internal jugular vein. The patients' lungs were ventilated with a tidal volume of 8 to 10 ml/kg, an inspiratory /expiratory ratio of 1:1.9, and an inspiratory pause of 10% of total inspiration time at a rate of 8 to 12 breaths/min in 100% oxygen with no positive end-expiratory pressure throughout the study period. The accordance of $PaCO_2$ with end-tidal CO_2 was confirmed before starting the study and respiratory rate was adjusted to maintain $PaCO_2$ within 33–38 mmHg and pH around 7.4. Anesthesia was maintained with isoflurane (0.8%-1%), continuous infusion of remifentanil (0.05-0.2 μg/kg/min) and vecuronium (1-2 μg/kg/min). Central temperature measured by PAC was maintained above 36°C with a warm mattress, forced warm air blanket, and fluid warmer as necessary.

All patients were turned to LDP and OLV was initiated and tidal volume and respiratory rate were adjusted within the predefined range to maintain peak airway pressure below 40 cm H_2O and normocarbia. Fifteen minutes (min) later, patients were 10° head-down tilted for 10 min and then, returned to horizontal position in the HG while the patients remained horizontal throughout the study period in the CG. Ten degree was chosen, because it was the most frequently utilized degree of tilting in most of the clinical cases as measured with

a goniometer in a preliminary study. The pressure transducers were located at the level of the right atrium during all phases of the study and recalibrated after each position change. Arterial blood samples were measured with an automated blood gas analyzer (Stat Profile® CCX, Nova Biomedical, MA, USA).

Hemodynamic variables, arterial and mixed venous blood gas analyses, peak airway pressure and dynamic pulmonary compliance were recorded at following points. Five min after induction of anesthesia, 15 min after OLV in the LDP (T0, baseline), 5 and 10 min after 10° head-down tilt (T5 and T10, respectively), and 10 min after the patient was returned to horizontal position (TH). Hemodynamic measurements included mean arterial pressure (MAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and cardiac index (CI). Corresponding systemic vascular resistance index (SVRI) and pulmonary vascular resistance index (PVRI) were calculated using standard formula. Oxygen content (CxO_2) in arterial and mixed venous blood was calculated using equation: $CxO_2 = 1.36 \cdot Hb \cdot SxO_2 + 0.0031 \cdot PxO_2$, where Hb = hemoglobin concentration (g/dl) and SxO_2 = oxygen saturation. The alveolar-arterial O_2 gradient ($A\text{-}aO_2$) was calculated as the difference between PAO_2 and PaO_2. Qs/Qt was determined using the formula: $Qs/Qt = (CcO_2 - CaO_2)/(CcO_2 - CvO_2)$, CcO_2 = capillary O_2 content calculated assuming that pulmonary capillary O_2 partial pressure is equal to PAO_2.[13] Percent change of Qs/Qt, PaO_2 and $A\text{-}aDO_2$ from the baseline values (%Qs/Qt, $\%PaO_2$, $\%A\text{-}aDO_2$, respectively) were calculated as follows: (value at a given point – baseline value) * 100/ (baseline value).

This study was planned to terminate if MAP was decreased more than 20% of post-induction value requiring vasoactive drug administration or arterial oxygen saturation as measured by pulse oximetry was declined to below 90%, or PaO2 was decreased below 60 mmHg. To control factors affecting hypoxic pulmonary vasoconstriction (HPV), any use of vasoactive drugs and surgical incision were withheld until after completion of study.

Statistical analyses were performed with SPSS 12.0 (SPSS Inc., Chicago, IL, USA). All data are expressed as mean ± standard deviation (SD) or number of patients. Since there was no previous study evaluating the effect of head-down tilt during OLV in the LDP in thoracic surgical patients, sample-size calculation was performed based on a preliminary study with the following assumptions; 80% power to detect a 10% difference in percent change of PaO_2 from baseline value between the groups with an SD of 10% and an alpha

level of 0.05 using an independent t-test. This generates an estimate of 17 patients each group. Data between the groups were compared using Chi-square test, Fisher's exact test or independent t-test as appropriate. Changes between time points within the group were compared using univariate analysis of variance with post hoc comparisons using the Dunnette’s test. A *P* value of less than 0.05 was considered statistically significant.

Ⅲ. RESULTS

The patients in the two groups were similar with respect to age, sex, body surface area, functional vital capacity and hemoglobin values (Table 1).

Table 1. Patients' characteristics

	Control (n = 17)	Head-down tilt (n = 17)	P value
Age (y)	52.7 ± 13.6	53.9 ± 9.0	0.757
Sex (M/F)	10/ 7	13 / 4	0.465
Body surface area (m^2)	1.71 ± 0.20	1.70 ± 0.13	0.968
FVC (% as predicted)	95.8 ± 11.9	92.7 ± 15.1	0.519
FEV_1 (% as predicted)	99.3 ± 16.1	102.4 ± 18.6	0.614
Hemoglobin (g/dl)	11.6 ± 1.1	12.3 ± 0.7	0.063
Operative side (R/L)	14/ 3	9/ 8	0.141

Values are mean ± SD or number of patients. FVC, functional vital capacity; FEV_1, forced expiratory volume in 1 second.

The diagnoses of the patients were primary lung cancer (25 patients), lung metastases (6 patients), thymoma (2 patients), and endobroncheal tuberculosis (1 patient). The surgical interventions that had been performed were pneumonectomy (2 patients), lobectomy (21 patients), wedge resection (8 patients) and VATS (3 patients).

None of the patients developed dangerous hypoxemia, as well as hypotension requiring vasoactive drugs and the study could be successfully performed in all patients. There were no significant differences in baseline hemodynamic and respiratory variables between the groups (Table 2 and 3).

Table 2. Changes in hemodynamic variables.

Group	Baseline	T5	T10	TH
HR (beats/min)				
Control	68 ± 9	67 ± 9	66 ± 9	66 ± 11
Head-down tilt	61 ± 19	64 ± 11	63 ± 11	63 ± 12
MAP (mmHg)				
Control	83 ± 9	84 ± 9	85 ± 8	87 ± 10
Head-down tilt	83 ± 9	87 ± 9	87 ± 15	82 ± 10
PCWP (mmHg)				
Control	9.0 ± 2.9	10.5 ± 3.2	10.7 ± 2.1	10.7 ± 2.7
Head-down tilt	8.8 ± 3.1	11.1 ± 2.7	11.8 ± 3.8*	10.1 ± 1.9
CVP (mmHg)				
Control	4.9 ± 2.7	5.4 ± 2.6	5.7 ± 2.5	5.9 ± 2.5
Head-down tilt	4.8 ± 2.7	7.6 ± 2.8*†	7.5 ± 2.6*	5.2 ± 1.3
CI ($L/min/m^2$)				
Control	3.2 ± 0.6	3.1 ± 0.6	3.2 ± 0.6	3.3 ± 0.8
Head-down tilt	2.9 ± 0.6	3.0 ± 0.6	3.1 ± 0.7	3.1 ± 0.7
SVRI ($dynes \cdot sec \cdot cm^{-5} \cdot m^2$)				
Control	2033 ± 411	2074 ± 382	2067 ± 411	2076 ± 565
Head-down tilt	2133 ± 451	2147 ± 480	2110 ± 521	2049 ± 505
PVRI ($dynes \cdot sec \cdot cm^{-5} \cdot m^2$)				
Control	140 ± 33	145 ± 30	139 ± 35	147 ± 38
Head-down tilt	169 ± 61	176 ± 52†	173 ± 50†	158 ± 50

Values are mean ± SD. Baseline, 15 min after initiation of OLV; T5, 5 min after head-down tilt; T10, 10 min after head-down tilt; TH, 10 min after horizontal position following head-down tilt; HR, heart rate; MAP, mean systemic arterial pressure; PCWP, pulmonary capillary wedge pressure; CVP, central venous pressure; CI, cardiac index; SVRI, systemic vascular resistance index; PVRI, pulmonary vascular resistance index. *$P < .05$ versus baseline in each group; †$P < .05$ versus control group.

Table 3. Changes in respiratory variables

Group	Group	Baseline	T5	T10	TH
PaO_2 (mmHg)	Control	309 ± 54	307 ± 56	307 ± 55	290 ± 52
	Head-down tilt	299 ± 49	279 ± 47	279 ± 47	272 ± 46
% change of PaO_2	Control		- 0.7 ± 5.3	- 0.9 ± 7.1	- 6.5 ± 10.0*
	Head-down tilt		- 6.3 ± 6.7*†	- 6.4 ± 7.8*†	- 6.8 ± 8.8*
A-aO_2 (mmHg)	Control	361 ± 53	364 ± 56	364 ± 55	384 ± 51
	Head-down tilt	372 ± 49	391 ± 48	391 ± 48	400 ± 46
% change of A-aO_2	Control		0.7 ± 5.1	1.1 ± 6.9	7.6 ± 11.3*
	Head-down tilt		5.3 ± 6.7†	5.4 ± 7.8	6.4 ± 8.3*
Qs/Qt (%)	Control	25.5 ± 7.3	32.6 ± 14.5	29.5 ± 13.5	29.9 ± 6.6
	Head-down tilt	21.9 ± 3.0	28.3 ± 9.4	30.8 ± 16.9*	30.1 ± 6.1
% change of Qs/Qt	Control		29.3 ± 60.7	19.9 ± 43.8	22.5 ± 28.0
	Head-down tilt		28.9 ± 33.8	40.7 ± 70.3*	41.8 ± 29.5*
Paw (cmH_2O)	Control	21 ± 4	21 ± 4	21 ± 4	22 ± 4
	Head-down tilt	24 ± 6	25 ± 6†	25 ± 6†	24 ± 5
Cdyn (ml/cmH_2O)	Control	29 ± 9	29 ± 6	30 ± 6	30 ± 6
	Head-down tilt	28 ± 8	27 ± 6	26 ± 6	28 ± 7

Values are mean ± SD. Baseline, 15 min after initiation of OLV; T5, 5 min after head-down tilt; T10, 10 min after head-down tilt; TH, 10 min after horizontal position following head-down tilt; PaO_2, arterial oxygen tension; Qs/Qt, pulmonary shunt fraction; PAW, peak airway pressure; Cdyn, dynamic compliance. *P < .05 versus baseline in each group; †P < .05 versus control group.

In inter-group comparisons of hemodynamic variables, CVP at T5 (p = 0.019) and PVRI at T5 (p = 0.044) and T10 (p = 0.028) were significantly higher in the HG. Other variables were similar.

In intra-group comparisons of hemodynamic variables to baseline values, CVP at T5 (p = 0.003) and T10 (p = 0.007), and PCWP at T10 (p = 0.01) were significantly increased compared to baseline values and returned to baseline values at TH in the HG. HR, MAP, CI and SVRI were maintained during the study period in the HG. In the CG, there were no significant changes throughout the study period (Table 2).

In inter-group comparisons of respiratory variables, there were no significant differences in the absolute values of PaO_2, A-aO_2 and Qs/Qt. %PaO_2 was significantly lower in the HG at T5 (p = 0.013) and T10 (p = 0.044). %A-aO_2 was significantly higher in the HG at T5 (p

= 0.034). %Qs/Qt showed a trend toward higher %Qs/Qt in the HG at TH (p = 0.083) without statistical significance. Peak airway pressure was significantly higher at T5 (p =0.032) and T10 (p = 0.023) in the HG without any differences in dynamic pulmonary compliance (Table 3).

In intra-group comparisons of respiratory variables to baseline values, Qs/Qt was significantly increased at T5 (p = 0.041) only in the HG. $\%PaO_2$ was significantly decreased at TH (p = 0.022) in the CG while it was significantly decreased at T5 (p = 0.021), T10 (p = 0.02) and TH (p = 0.017) in the HG. $\%A\text{-}aO_2$ was significantly increased at TH in both groups (p = 0.01, in CG and p = 0.024, in HG). %Qs/Qt was significantly increased at T10 (p = 0.019) and TH (p = 0.026), only in the HG. Peak airway pressure and dynamic pulmonary compliance did not show any significant changes in both groups (Table 3).

Ⅳ. DISCUSSION

In this prospective, randomized, controlled study assessing the effect of 10° head-down tilt for 10 minutes during OLV in the LDP on the respiratory variables and cardiac performance, we could observe significant increase in %Qs/Qt and decrease in $\%PaO_2$ without eliciting dangerous hypoxemia. The absolute values of Qs/Qt and PaO_2 showed no significant changes except the Qs/Qt at T10 in the HG which was increased compared to baseline value. These changes were accompanied by increases in cardiac filling pressures without any significant changes in cardiac index.

OLV is frequently utilized in thoracic surgeries and in some patients, severe hypoxemia may occur, mandating implementation of other therapies to provide adequate oxygenation. The cause of hypoxemia during OLV is mainly due to an increase in Qs/Qt through the nonventilated lung. The principle mechanism to achieve better V/Q matching is to reduce pulmonary perfusion in nonventilated lung. In a previous study, LDP prevented life-threatening hypoxemia (SpO2 < 90%) in 92% of the patients, which developed about 10 minutes after the start of OLV in 82% of patients in the supine position in anesthetized humans.[3] Both HPV and gravity would cooperate to reduce pulmonary blood flow in the nondependent lung[3] and gravity would be an important factor for the redistribution of pulmonary perfusion with proportionally more blood flow in the dependent areas of a lung in the LDP.[14]

Although safer than supine position, placing anesthetized patients in the LDP results in significant V/Q mismatch as well. With mechanical ventilation, the expansion of the dependent lung is restricted by abdominal contents impinging on the diaphragm, resulting in overventilation of the nondependent lung and underventilation of the dependent lung. This leads to loss of functional residual capacity, atelectasis and decrease in compliance in the dependent lung.[15-17] In thoracic surgery, head-down tilt is sometimes required for optimal surgical exposure during thoracoscopic procedures or managing hemodynamic instability. In anesthetized-paralyzed patients in the LDP, additional head-down tilt might aggravate cephalad displacement of abdominal contents and more decrease in FRC, TLC and compliance[6] which can impose the risk of dangerous hypoxemia upon OLV as in supine position. However, no comprehensive data exist regarding the effect of head-down tilt on Qs/Qt and oxygenation during OLV in the LDP and we observed that 10° head-down tilt for

10 minutes during OLV in the LDP resulted in significant increase in %Qs/Qt and decrease in $\%PaO_2$ without eliciting dangerous hypoxemia.

In this study, in order to minimize factors affecting HPV response, anesthetic agents (narcotic + inhalation agent) and acid-base status were controlled throughout the study period. In addition, surgical incision and vasoactive agents were also withheld. As our results indicate, additional head-down tilt during OLV in the LDP caused faster decrease in $\%PaO_2$ as well as faster increase in $\%A\text{-}aO_2$ and significant increase in %Qs/Qt which persisted after returning to horizontal LDP. The Qs/Qt values at baseline are consistent with previous study with anesthetized mechanically ventilated patients with 100 % oxygen during OLV.[18] The decrease in $\%PaO_2$ at TH that is 35 min after the onset of OLV in the CG is consistent with the regression curve of PaO_2 decrement after OLV start in LDP without head-down tilt.[3] The results that, further increase in %Qs/Qt, which persisted to be somewhat higher in the HG than CG even after the patients were returned to horizontal LDP ($p = 0.083$) and faster decrease in %PaO2 in the HG which also has intergroup statistical significance, indicate clearly the impact of head-down tilt on these parameters. The effect of head-down tilt on these results could be attributable to combination of multiple factors aggravating V/Q mismatch. Possible factors are as follows. First, increased atelectasis formation by impingement of abdominal contents against the paralyzed diaphragm can be considered. It has been demonstrated that the magnitude of Qs/Qt correlates well with the size of atelectasis.[19] Second, although gravity rather than HPV response was suggested to be a more dominant factor in the distribution of pulmonary perfusion in LDP,[3] decreased HPV response should also be considered. HPV response is maximal when pulmonary vascular pressure is normal and is decreased by either high or low pulmonary vascular pressure. HPV response is not only induced by decrease in alveolar oxygen tension, but also by decrease in mixed venous oxygen tension, which under same conditions reflects cardiac output.[20] Since there were no significant differences in CI between the groups, increased peak airway pressure and PVRI in the HG would cause less effective HPV response because the pulmonary circulation is poorly endowed with smooth muscle and cannot constrict against an increased vascular pressure.[21] The results that none of the patients developed dangerous hypoxemia and neither of the absolute values of Qs/Qt or PaO_2 showed clinically significant changes, indicate the dominant role of gravity in

redistribution of pulmonary blood flow in the LDP enhancing V/Q and thereby preventing the occurrence of dangerous hypoxemia.

The limitations of this study are as follows. First, the maximal HPV response in anesthetized humans in the supine position occurred within 15 min,[22] however the time of maximal HPV response during OLV in the LDP has not been demonstrated. For ethical reasons, we had confined the duration of head-down tilt to 10 min. The duration was set with regard to a finding from previous study showing that 92% of patients developed dangerous hypoxemia during OLV in the supine position in 10 min,[3] and to limit the duration of delaying surgical procedure, which was withheld to minimize its' influence on the HPV response. Therefore, the duration in this study might not be sufficient to have induced maximal HPV response. Unlike in the supine position, considering that HPV response seems to contribute only a minor portion in redistribution of pulmonary blood flow in the LDP with more dominant role of gravity, more prolonged duration of head-down tilt would not have resulted in better preservation of either %Qs/Qt or $\%PaO_2$. Second, to avoid distorting comparative outcome, we have excluded patients with decreased pulmonary function (FEV1 and FVC < 80%) who could be more susceptible in developing dangerous hypoxemia during OLV and these results should not be extended to this subset of patients.

Ⅴ. CONCLUSION

In conclusion, additional 10° head-down tilt for 10 min during OLV in the LDP caused significant increase in %Qs/Qt which persisted after returning to horizontal LDP and faster decrease in $\%PaO_2$ as well as faster increase in $\%A\text{-}aO_2$. These changes were accompanied by transient increases in cardiac filling pressures without any changes in cardiac index. However, none of the patients developed dangerous hypoxemia and the absolute values of Qs/Qt and PaO_2 showed clinically insignificant changes between the groups. Therefore, brief periods of additional head-down tilt during OLV in the LDP for optimizing surgical exposure can be well tolerated in patients with preserved pulmonary function.

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국문요약

측와위 일측폐 환기 시 두부하강 체위가 폐내 션트와 산소화에 미치는 영향

< 지도교수 곽영란 >

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연구배경: 일측폐환기 시 수술 자세는 산소화 악화 정도에 영향을 미치며 측와위 자세는 위험한 저산소증 예방에 있어 앙와위보다 월등하다. 그러나 측와위에서 추가적인 두부하강 체위는 복강내 내용물이 의존폐를 압축시켜 앙와위만큼 저산소증의 위험성이 증가할 수 있다. 이에 본 연구에서는 일측폐환기 시 측와위에서 두부하강체위가 폐내션트와 산소화에 미치는 영향을 전향적으로 연구하였다. 연구방법: 폐수술이 예정된 34명의 환자를 임의적으로 측와위 상태를 유지하는 군(n=17, 대조군)과 측와위에서 두부하강체위를 시행하는 군(n=17, 실험군)으로 나누었다. 혈역학 및 호흡기계 변수를 일측폐 시작하고 15분 후(기저치), 10도의 두부하강체위를 시행한 후 5분과 10분 후(T5와 T10), 다시 수평자세로 바꾼 10분 후(TH)에 측정하였다. 실험군에서 션트분율의 퍼센트 변화는 T10과 TH 시기에 유의하게 증가하였고 동맥혈 산소분압의 퍼센트 변화는 T5, T10, TH 시기에 모두 유의하게 감소하였다. 결과: 대조군에서는 두부하강 후 심장충만압이 증가하지만 심박출지수는 변화가 없었다. 결론: 측와위 일측폐환기 시 두부하강 체위는 션트분율을 증가시키고 동맥혈 산소분압의 퍼센트 변화를 감소시켰다. 동시에 심박출지수의 증가를 동반하지 않는 심장충만압을 증가시켰다.

핵심되는 말 : 일측폐환기, 폐내션트, 두부하강체위