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Effect of Mild Hypercapnia on Lung Oxygenation in Sitting Position During Shoulder Arthroscopy Under General Anesthesia

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Background: Mild hypercapnia is permitted during surgeries in sitting position under general anesthesia to maintain cerebral regional oxygen saturation (rSO_2). However, since hypoventilation may cause gas exchange impairment, we evaluated effects of mild hypercapnia on lung oxygenation during shoulder arthroscopy in sitting position.

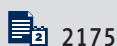
Material/Methods: Forty patients were randomly allocated to a normocapnia group ($ETCO_2$ 35 mmHg, $n=20$) or a hypercapnia group (45 mmHg, $n=20$). The mean arterial pressure (MAP), heart rate (HR), and rSO_2 were measured 5 min after intubation in supine position (T0), and at 2, 4, 6, 8, 10, 20, 30, 40, 50, and 60 min of remaining in sitting position (T1–10). Arterial blood gas was analyzed at T0 and T5. The oxygenation index (PaO_2/FiO_2) and dead-space ventilation ratio (Vd/Vt) were calculated.

Results: There were no differences in PaO_2/FiO_2 at T0 and T5 between the 2 groups. At T5, the Vd/Vt was higher in the normocapnia group than in the hypercapnia group ($p=0.04$). The Vd/Vt at T5 increased from T0 in the normocapnia group. The incidence of cerebral desaturation in the hypercapnia group (0/20) was lower than in the normocapnia group (5/20) ($p=0.047$). Among rSO_2 , MAP, and HR, only changes in rSO_2 over time between the 2 groups differed significantly ($p=0.048$).

Conclusions: Mild hypercapnia did not decrease lung oxygenation in sitting position, probably due to attenuation of the increase in dead-space ventilation ratio. Since hypercapnia maintained rSO_2 without changes in oxygenation index and hemodynamic parameters, mild hypercapnia should be maintained during shoulder arthroscopy in sitting position under general anesthesia.

MeSH Keywords: **Hypercapnia • Hypoventilation • Respiratory Dead Space**

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Background

Shoulder arthroscopy is often performed in the sitting position because the position provides better visualization without the distortion of the shoulder anatomy. The sitting position under general anesthesia carries potential for neurologic complications ranging from transient visual loss to cerebral and spinal cord ischemia, which are associated with decreased mean arterial pressure (MAP) and cardiac output [1–3]. To prevent cerebral complications, mild hypercapnia is permitted because it was reported to attenuate the decrease in cerebral regional oxygen saturation (rSO_2) [4]. Hypoventilation increases arterial carbon dioxide tension ($PaCO_2$), which in turn increases rSO_2 via its vasodilatory effect on cerebral vasculature. A previous study in patients without vascular disease reported that rSO_2 changed according to the change in $PaCO_2$ [5]. However, hypoventilation of the lungs may result in atelectasis and increase in intrapulmonary shunt, which leads to hypoxemia and lung injury [6,7]. In previous studies on respiratory effect of the sitting position during neurosurgery, sitting position was associated with decreased arterial oxygen tension (PaO_2) and increased dead-space ventilation (Vd/Vt) [8,9].

Hypercapnia is a potent cerebral vasodilator, but it enhances hypoxic vasoconstriction in the pulmonary vascular system [10]. We hypothesized that hypoventilation-induced mild hypercapnia would influence lung oxygenation, hypoxic pulmonary vasoconstriction, and dead-space ventilation in the sitting position. The purpose of this study was to evaluate the effects of hypercapnia on lung oxygenation during shoulder arthroscopy in the sitting position. The primary outcome variable was the oxygenation index (PaO_2/FiO_2) and secondary variables were dead-space ventilation (Vd/Vt), rSO_2 , MAP, and heart rate (HR).

Material and Methods

After the Institutional Review Board of GUGMC (Gachon University Gil Medical Center) approval, written informed consent from all patients was obtained. Patients undergoing elective shoulder arthroscopic surgery were randomly allocated to either the normocapnia group ($n=20$) or the hypercapnia group ($n=20$) using computer-generated random numbers. Exclusion criteria included age >65 years, obesity ($BMI >30$ kg/m²), history of diffuse lung disease, uncontrolled systemic hypertension, intracranial disease, and/or cardiovascular and cerebrovascular disease (Figure 1). The normal range of $PaCO_2$ is between 35 and 45 mmHg; therefore, 45 mmHg is not actually hypercapnia. In this study, $ETCO_2$ of 45 mmHg is defined as 'mild' hypercapnia to clarify the concept of increased $PaCO_2$.

Patients were premedicated with intramuscular midazolam 0.05 mg/kg and glycopyrrolate 0.2 mg before being transported

to the operating room. All patients received volume replacement with 5 ml/kg 6% hydroxyethyl starch (130/0.4) before anesthesia induction. Standard monitoring including electrocardiogram, pulse oximeter, noninvasive blood pressure, and a BIS monitor (BIS Vista monitor Revision 3.0, Aspect Medical Systems, Norwood, MA) was begun in the operating room. For invasive blood pressure monitoring, a 20G catheter was inserted into the radial artery. The pressure transducer was placed at the mid-axillary level when patients were in the supine position and at the level of the external ear canal level when they were in the sitting position. The rSO_2 was measured continuously using the INVOS[®] 5100B cerebral oximeter (Somanetics Corporation, Troy, MI). After cleansing the skin with an alcohol pad, the cerebral oximeter sensors were attached on both sides of frontotemporal area.

After pre-induction measurements of mean arterial pressure (MAP), heart rate (HR), and rSO_2 , anesthesia was induced with propofol 2 mg/kg and alfentanil 10 μ g/kg followed by 2 vol% sevoflurane. The endotracheal intubation was facilitated with rocuronium 0.6 mg/kg. The respiratory rate was adjusted to achieve the objective values of end-tidal carbon dioxide tension ($ETCO_2$) 35 mmHg in the normocapnia group (tidal volume setting of 8 ml/kg) or 45 mmHg in the hypercapnia group (tidal volume setting of 6 ml/kg) with 50% oxygen in air. The inspiratory-to-expiratory ratio was set at 1:2, with no positive end-expiratory pressure. Anesthesia was adjusted in a range of sevoflurane 2–3 vol% to maintain BIS values of 40–50.

Approximately 5 min after intubation, when MAP and HR became stable, patients were placed in the 70° sitting position using a beach chair (Allen[®] Lift-Assist[®] Beach Chair, Allen Medical System, Acton, USA). The MAP, HR, and rSO_2 were measured 5 min after intubation in the supine position (T0), 2, 4, 6, 8, 10, 20, 30, 40, 50, and 60 min (T1, T2, T3, T4, T5, T6, T7, T8, T9, and T10, respectively) after being in the beach chair position. The incidence of hypotension (MAP \leq 60 mm Hg) was recorded and treated with phenylephrine 100 μ g (HR \geq 60 BPM) or ephedrine 5 mg (HR $<$ 60 BPM). A cerebral desaturation event (CDE) was defined as a drop in rSO_2 of 20% or greater from baseline (pre-induction) for 15 s or greater [11]. When CDE occurred, the first step was to check and correct the circuit, hemoglobin, fraction of oxygen, and mean arterial pressure. When the first step was ineffective, MAP was corrected with fluid replacement and phenylephrine.

Arterial blood gas was analyzed at T0 and T5. The oxygenation index (PaO_2/FiO_2) and dead-space ventilation ratio (Vd/Vt) were calculated according to the Hardman and Aitkenhead equation:

$$Vd/Vt=1.135 \times (PaCO_2 - ETCO_2) / PaCO_2 - 0.005 \quad [12].$$

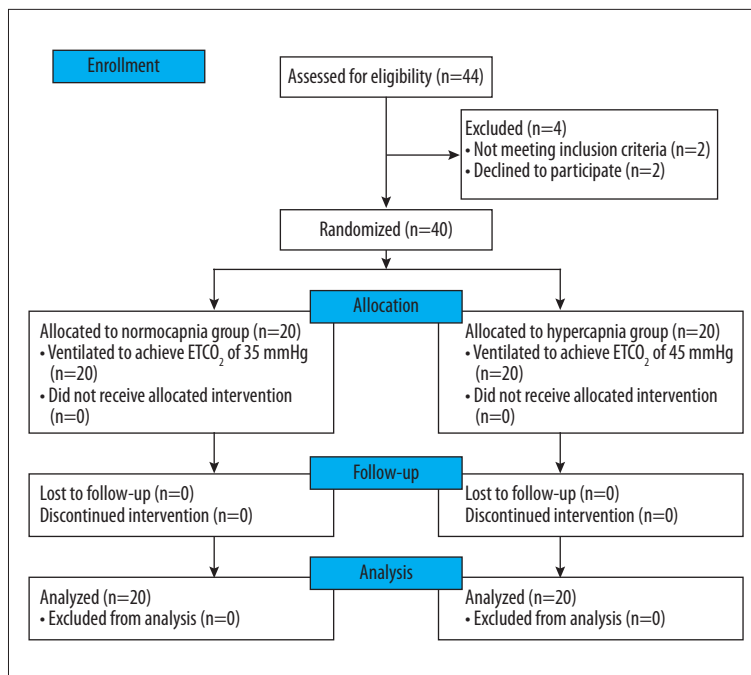


Figure 1. Flow diagram of patient allocation.

Forty-four patients were assessed for eligibility. Two patients were excluded after screening and 2 patients refused to participate. Forty patients completed the study.

Sample size was calculated based on a previous study [13]. In each group, 18 patients were needed to achieve an alpha-error of 0.05 and a power of 80%, assuming the mean intergroup difference of 48 in oxygenation index (SD=50). To compensate for a dropout rate of 10%, 40 patients were included in this study. Statistical analyses were performed using SPSS 13.0 for Windows (SPSS Inc., Chicago, IL). Normal distribution of data was tested by Kolmogorov-Smirnov tests. Variables of patients' characteristics and induction profiles were compared using an unpaired t-test or chi-square test. Changes in MAP, HR, and rSO_2 over time between the groups were compared using repeated-measures ANOVA, and post hoc comparison within the group was performed using the LSD test. The intergroup difference in respiratory parameters at each time point was compared using the unpaired t-test. Statistical significance was considered as $P < 0.05$.

Results

Forty-four patients were assessed for eligibility. Two patients were excluded after screening and 2 patients refused to participate. Forty patients completed the study (Figure 1). Patients' characteristics and data from the perioperative period are listed in Table 1. There was no difference in patients' characteristics and pre-induction hemodynamics, including rSO_2 . The incidence of CDE was lower in the hypercapnia group compared to that in the normocapnia group ($p = 0.047$). The oxygen fraction was raised to 1.0 in all patients with CDE. There was no difference in the incidence of hypotension between the 2 groups.

The respiratory variables are listed in Table 2. There were no differences in PaO_2/FiO_2 at T0 and T5 between the 2 groups. Compared to baseline (T0), the PaO_2/FiO_2 at T5 did not change significantly in either group. At T5, the Vd/Vt was significantly higher in the normocapnia group compared to that in the hypercapnia group ($p = 0.04$). The Vd/Vt at T5 increased significantly from T0 only in the normocapnia group. At 2 time points, pH was significantly lower in the hypercapnia group than in the normocapnia group and $PaCO_2$ was significantly higher in the hypercapnia group than in the normocapnia group.

The changes in MAP, HR, and rSO_2 while in beach chair position are shown in Figure 2. The change in MAP over time between the groups did not differ significantly ($p = 0.586$) and there was no significant intergroup difference ($p = 0.405$). In the normocapnia group, MAP decreased significantly from T0 to T1, T2, T3, T4, and T5 and then increased at T7. In the hypercapnia group, MAP decreased significantly from T0 to T4, T5, and T6. The change in HR over time between the groups did not differ significantly ($p = 0.545$), but there was a significant intergroup difference in HR ($p = 0.048$). Compared to baseline (T0), HR decreased at T5 and T6 in the normocapnia group and at T3, T4, T5, and T6 in the hypercapnia group. The change in rSO_2 over time between the groups differed significantly ($p = 0.048$), with a significant intergroup difference ($p = 0.003$). The rSO_2 decreased significantly from T0 to T1, T2, T4, and T5 in the normocapnia group, but it did not change in the hypercapnia group.

Table 1. Patient characteristics and data from perioperative period.

	Normocapnia (n=20)	Hypercapnia (n=20)
Sex (M/F)	10/10	13/7
Age (years)	53±8	50±9
Weight (kg)	64±10	69±10
Pre-induction hemodynamics		
Mean arterial pressure (mmHg)	108±17	102±10
Heart rate (beats/min)	72±12	75±15
Regional cerebral oxygen saturation (%)	65±5	68±7
Incidence of cerebral desaturation event (n)	5	0*
Incidence of hypotension (n)	9	7
use of ephedrine (n)	4	2
use of phenylephrine (n)	6	6

Values are means ±SD or numbers of patients. * P<0.05 vs. normocapnia group.

Table 2. Changes in respiratory variables during anesthesia and operation.

	T0	T5
pH		
Normocapnia	7.44±0.04	7.46±0.03
Hypercapnia	7.40±0.04*	7.38±0.03***
PaO ₂ /FiO ₂		
Normocapnia	423±81	400±69
Hypercapnia	404±101	410±79
PaCO ₂ (mmHg)		
Normocapnia	37.6±6.1	37.9±3.2
Hypercapnia	42.8±5.8*	46.3±4.4***
ETCO ₂ (mmHg)		
Normocapnia	33.6±2.1	33.7±1.9
Hypercapnia	40.6±4.2*	43.0±2.3***
Vd/Vt (%)		
Normocapnia	6.3±11.3	12.7±9.4**
Hypercapnia	5.2±8.9	7.3±8.9*

Values are means ±SD. PaO₂/FiO₂ – oxygenation index; ETCO₂ – end-tidal carbon dioxide tension; Vd/Vt – dead space ventilation ratio; T0 – 5 min after intubation in the supine position; T5 – 10 min after beach chair position. * P<0.05; vs. normocapnia group, ** P<0.05; vs. T0.

Discussion

This study demonstrated that hypoventilation-induced mild hypercapnia did not decrease lung oxygenation in the sitting position under sevoflurane anesthesia, probably due to the attenuation of the increase in dead-space ventilation in the sitting position. Mild hypercapnia also prevented the decrease in the rSO₂ while in sitting position under general anesthesia.

Previous studies on the respiratory effect of sitting position during neurosurgery reported the decrease in lung oxygenation and increase in dead-space ventilation, which are partly consistent with the results of the present study [8,9]. In the present study, the dead-space ventilation was increased in the normocapnia group but the oxygenation index was not changed in the sitting position. The difference may be due to the elimination of nitrous oxide in this study, which attenuates hypoxic pulmonary vasoconstriction [14]. The effect of hypercapnia on the respiratory system has been studied in patients with severe respiratory failure, in which the hypercapnia was a result of lung protective ventilation to decrease further lung injury [15]. This concept of ‘permissive’ hypercapnia is changing to ‘therapeutic’ hypercapnia, in which CO₂ may be added to increase PaCO₂ to 70 mmHg, because hypercapnic acidosis is reported to be associated with reduced lung injury due to its anti-inflammatory effect, as well as ventilation-perfusion matching [7,16]. Despite all the advantages of hypercapnia in patients with respiratory failure, hypercapnia is not used in normal lungs because hypoventilation may lead to atelectasis and increase in intrapulmonary shunt, which may lead to hypoxemia [6,7]. However, when patients in the hypercapnia group were raised to the sitting position in this study, enhanced hypercapnic pulmonary vasoconstriction may have

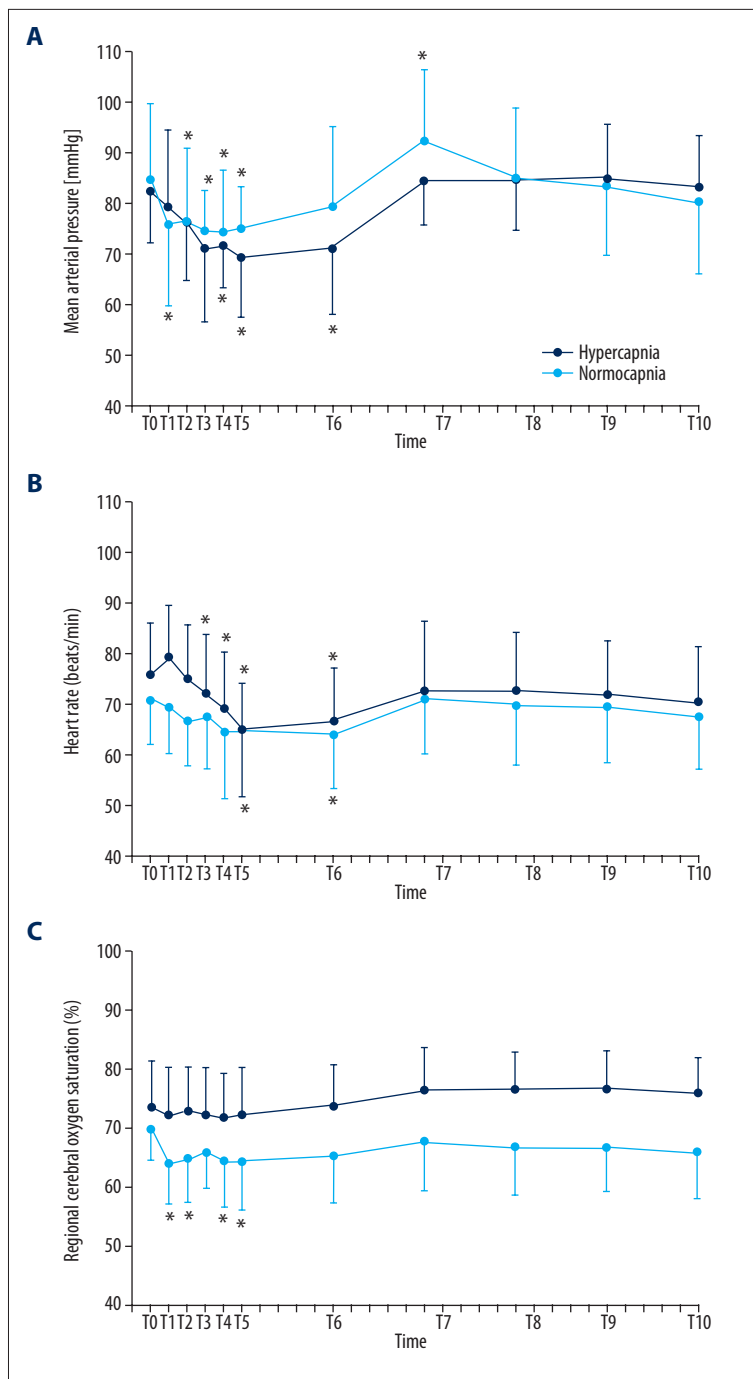


Figure 2. The changes in: (A) Mean arterial pressure (MAP); (B) Heart rate (HR); (C) Regional cerebral oxygen saturation (rSO₂). Filled circles represent hypercapnia group and unfilled circles represent normocapnia group. T0: 5 min after intubation in the supine position; T1, T2, T3, T4, T5, T6, T7, T8, T9, and T10: 2, 4, 6, 8, 10, 20, 30, 40, 50, and 60 min, respectively, after being in the beach-chair position. * P<0.05 vs. T0.

decreased dead space, which may have compensated for the effect of hypoventilation on oxygenation index.

Mild hypercapnia prevented the decrease in rSO₂, and no patient in the hypercapnia group experienced cerebral desaturation after being in the sitting position in this study. On the other hand, rSO₂ decreased with normocapnia, and 5 patients in the normocapnia group experienced cerebral desaturation events. The cerebral blood flow is easily modulated by changes

in PaCO₂ within a normal range of PaCO₂. However, during anesthesia, anesthetic agent, position, and patient vascular condition also affect the cerebral blood flow. The CO₂ responsiveness is well maintained during sevoflurane anesthesia [17]. During general anesthesia in patients without lung injury, only mild hypercapnia in the range of PaCO₂ between 40 and 45 mmHg is maintained to increase cerebral oxygenation in special circumstances such as surgeries in the sitting position and carotid endarterectomy [4,18]. When mild hypercapnia was instituted

in patients undergoing carotid endarterectomy, middle cerebral flow velocity was increased without carotid cross-clamping and rSO_2 was increased during carotid cross-clamping [18]. A recent study on the effect of ventilation on rSO_2 in the sitting position using a beach chair reported that maintaining $ETCO_2$ between 40 and 42 mmHg improved cerebral oxygenation [4]. In the present study, mild hypercapnia also prevented the decrease in rSO_2 after the sitting position but there was no difference in lung oxygenation or MAP. Since the change in rSO_2 was not associated with lung oxygenation or hemodynamic variables, cerebral vasodilation due to hypercapnic acidosis is the likely cause of increased cerebral perfusion in this study.

The hemodynamic instability associated with the beach chair position is well known. It is mainly caused by decreased preload [3]. The upright position causes a shift in blood from the intra- to the extra-thoracic compartment, which decreases cardiac output and mean arterial pressure [3]. This arterial hypotension associated with the sitting position may be aggravated by the vasodilating effect and myocardial depressant effect of anesthetic agents and changes in venous return due to positive-pressure ventilation [19]. The systemic effect of acute hypercapnic acidosis is complex. Although its direct effect is to impair contractility of cardiac and vascular smooth muscle, hypercapnia indirectly increases myocardial contractility and heart rate via enhanced sympathetic nervous system discharge [20,21]. Therefore, one cannot predict the hemodynamic response to hypercapnia. The change in MAP and HR over time between the groups did not differ significantly in this study.

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Our study has several limitations. First, the shunt fraction, which can support the effect of dead space on the oxygenation index, was not calculated in this study for ethical reasons. To calculate shunt fraction, pulmonary artery catheterization is needed, which is an invasive procedure not needed for shoulder arthroscopy. Second, oxygenation data after being in sitting position was measured only once, right after being in the sitting position. The timing of the sampling (10 min in sitting position) was chosen based on a previous study on rSO_2 in the sitting position [22], which reported mean rSO_2 at 9 min after maintaining the sitting position. Collecting more oxygenation data throughout the surgery may have provided more information.

Conclusions

Mild hypercapnia induced by hypoventilation did not decrease lung oxygenation in the sitting position during general anesthesia, probably due to attenuation of the increase in dead-space ventilation ratio. Since the increase in CO_2 prevented the decrease in rSO_2 without changes in oxygenation index and hemodynamic parameters, mild hypercapnia should be maintained during shoulder arthroscopy in the sitting position under general anesthesia.

Statement

All sources were departmental. All authors declare no competing interests.

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