

## Comparison of tidal volume and deep breath preoxygenation techniques undergoing coronary artery bypass graft surgery: effects of hemodynamic response and arterial oxygenation\*

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**Background/aim:** Different techniques exist for the preoxygenation of patients that will be operated on under general anesthesia. Preoxygenation with the deep breath (DB) method may affect cardiovascular stability, which is crucial for coronary artery bypass graft (CABG) patients. In this study, we aimed to compare the effects of the 3 min TVB preoxygenation technique and 1 min 8DBs technique on hemodynamic response and arterial oxygenation in patients with normal ejection fraction that were scheduled for elective CABG surgery.

**Materials and methods:** Forty patients classified as ASA II–III and scheduled for elective CABG surgery were randomly assigned to TVB/3 min or 8DBs/1 min for preoxygenation. Cardiovascular variables, i.e. heart rate, mean arterial pressure, central venous pressure, cardiac index, systemic vascular resistance index, and stroke volume index, and arterial blood gas samples were analyzed before and after preoxygenation and at the end of the apneic period before intubation.

**Results:** The preoxygenation methods affected the hemodynamic response similarly. PaO<sub>2</sub> increased significantly with 8DBs compared to the TVB at the end of preoxygenation but was similar between the groups at the end of the apneic period (respectively, P: 0.03; P: 0.15). PaCO<sub>2</sub> changes were similar between the groups.

**Conclusion:** In patients with normal ejection fraction scheduled for CABG, 8DBs can be an alternative to TVB preoxygenation. Our results should be compared with those of other studies.

**Key words:** Preoxygenation, conventional method, tidal volume method, deep breathing method, CABG surgery

### 1. Introduction

The aim of preoxygenation is to provide the patient with the maximum time for tolerance of apnea (1). The traditional technique for preoxygenation is 3–5 min tidal volume breathing (TVB) (2,3); however, this method can be inapplicable in uncooperative patients and in situations where anesthesia must be induced quickly. In such cases, 4–8 deep breaths every 30/60 seconds (4DBs/30 s, 8DBs/60 s) are alternative methods (4). Many coronary artery bypass graft (CABG) patients may require rapid sequence induction due to obesity and diabetes (5). While both techniques are equally effective in terms of delivering O<sub>2</sub> to the bloodstream (4,6,7), we are of the opinion that using the DB method is more practical. However, DBs may affect cardiovascular stability, which is important for cardiac patients. DBs are known to affect mean arterial

pressure (MAP), heart rate (HR), and stroke volume (SV) (8,9). In this study, we aimed to compare the effects of the 3 min TVB preoxygenation technique and 1 min 8DBs technique on both hemodynamic response and arterial oxygenation in patients with normal cardiac function that were to undergo elective CABG surgery.

### 2. Materials and methods

Forty patients between the ages of 40 and 70 classified as ASA II–III and scheduled to undergo elective CABG surgery were included in this prospective randomized study after ethics approval was obtained from local ethics committee of the university (KAEEK 2013/160). The patients were informed about the study bedside one day prior to the procedure and their informed consents were obtained. The exclusion criteria included expected

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difficult airway, lung disease, abnormal respiratory function test, hypoxia or hypercapnia in arterial blood gas (ABG), ejection fraction (EF) of under 40%, body mass index (BMI) of over 30 kg/m<sup>2</sup>, anemia, severe valve failure, cardiovascular autonomic dysfunction, and noncooperation. Patients were premedicated with intravenous (iv) midazolam before being transferred to the operating room. Next 5 L/min oxygen was given via a face mask and peripheral venous access was achieved in the antecubital area using an 18-gauge catheter. HR was determined by 5-channel electrocardiography, and standard peripheral oxygen saturation (SpO<sub>2</sub>) and noninvasive blood pressure monitoring were performed. After local anesthesia, a specific thermistor-tipped catheter (VolumeView catheter, 5 Fr, 20 cm, Edwards Lifesciences, Irvine, CA, USA) was placed in the right femoral artery by iv application of fentanyl 1–2 µg/kg. Local anesthetic was infiltrated at first and a central venous pressure (CVP) catheter was placed preferably in the right vena jugularis interna. The arterial and venous catheters were connected to an EV1000 CO monitor (Edwards Lifesciences). The patients were divided into two groups of 20 patients each using the closed envelope system. Preoxygenation was achieved in the patients in Group A with 3 min normal TV with a flow rate of 5 L/min (TVB/3 min), and in Group B with eight deep breaths within 60 s (8DBs/60 s), and an oxygen flow of 10 L/min, with 100% oxygen. A 2-L balloon was used for preoxygenation, applied by the same person. After the anesthesia circuit was washed out with oxygen, the right-size polyvinyl chloride anesthesia mask with a silicone part touching the patient was placed on his/her face to fit correctly without air leakage. Reflection of the respiratory status and volumes on the balloon reservoir and display of significant capnograph traces were assured. All the patients included in the study were ventilated using the same anesthesia device (Dräger, Primus, Dräger Medical AG&Co, Germany). Anesthesia was induced with thiopental 3–5 mg/kg, fentanyl 5 µg/kg, and rocuronium 1 mg/kg in all patients following preoxygenation. After a 60 s duration following anesthesia induction, both groups were given oxygen without ventilation with a flow rate of 10 L/min. The patients were intubated at the end of this apneic period. The MAP, HR, CVP, cardiac index (CI), stroke volume index (SVI), systemic vascular resistance index (SVRI), and ABG values were reported at control (prior to preoxygenation), T<sub>1</sub> (end of preoxygenation), and T<sub>2</sub> (end of apneic period) times. In the case of hypotension (decrease in systolic arterial pressure of more than 20% from the baseline) during induction, the patient was first placed in the Trendelenburg position. In nonresponding patients, 250 mL of colloid was infused, and bolus administration of 5–10 mg of iv ephedrine was performed in the case of further unresponsiveness. In the case of

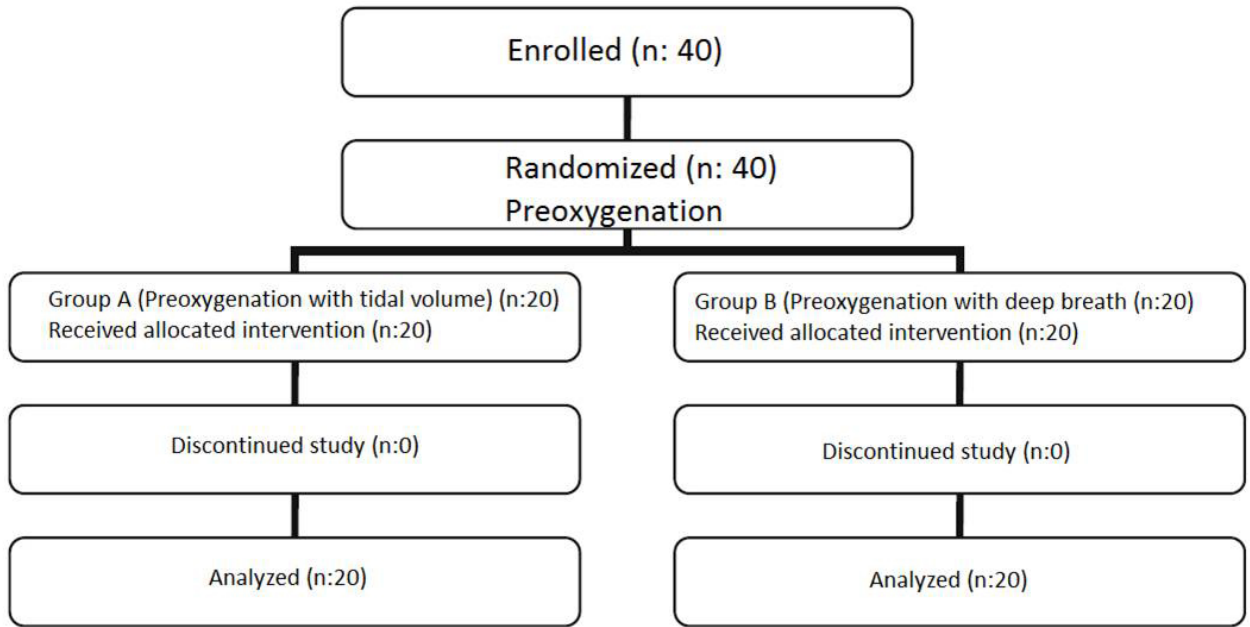
hypertension (increase in systolic arterial pressure of more than 20% from the baseline), anesthesia was deepened and 2 µg/kg fentanyl was applied. For bradycardia (an HR under 50 beats/min), the patients were administered 0.5 mg of iv atropine. Anesthesia was maintained with 30%/60% O<sub>2</sub>/air + desflurane and remifentanyl infusion. Intraoperative fluid management and vasopressor and inotrope requirements were detected according to the MAP and SV variability values, regional cerebral oximetry, hemoglobin, hematocrit, and urine excretion values.

### 2.1. Statistical analysis

Statistical analyses were performed using SPSS for Windows 16.0. In the evaluation of the study data, the Kolmogorov–Smirnov normality test for quantitative data was used besides the descriptive statistical methods (mean ± standard deviation or median (25th–75th percentiles)). The independent samples t-test was applied in the evaluation of the data that followed a normal distribution, whereas the Mann–Whitney U test was used for the assessment of those data that did not follow a normal distribution. The comparisons between the groups were done using two way analysis of variance and the assessment of data that did not follow a normal distribution was performed using Friedman's two way analysis of variance. Sample size was based on the pilot study of 10 patients. The MAP value was detected as 99 ± 15 mmHg in a pilot study of 10 patients that received 3 min TV preoxygenation. For a projected difference of 10% between the groups for MAP for a type 1 error 0.05 and a power of 0.8, 18 patients were required in each group. The number of the cases to be included in each group was increased to 20, taking into account the patients to be excluded from the study. The results were evaluated in a 95% confidence interval and a P value of P < 0.05 was considered statistically significant.

### 3. Results

Data related to all the patients included in the study were included in the statistical analysis (Figure). The demographic data, preoperative characteristics, and medication statuses of the patients were similar (P > 0.05) (Table 1). The MAP, HR, CI, SVI, SVRI, and CVP values did not differ significantly, either within a group or between the groups, at all time points during the study (P > 0.05) (Table 2). SaO<sub>2</sub> was significantly higher in both groups at the end of preoxygenation compared to the control values. Alterations were similar in the two groups. PaO<sub>2</sub> rose significantly at the end of preoxygenation in both groups in relation to the control values. This rise was significantly higher in the 8 DBs/60 s group, compared to the other group (P = 0.03). The PaO<sub>2</sub> values at the end of apnea were similar between the groups. The PaCO<sub>2</sub> levels presented similar changes in both groups. Although pH displayed a statistically significant reduction in the two



**Figure.** Patient randomization and follow-up according to the CONSORT (Consolidated Standards of Reporting Trials) guidelines.

**Table 1.** Patients' characteristics and details.

Characteristics	Group A n: 20 (TV/3 min)	Group B n: 20 (8DBs/60 s)	P
Age (years)	61.75 ± 7.51	59.15 ± 8.02	0.29
Sex (male/female) (n)	15/5	17/3	0.42
BMI (kg/m <sup>2</sup> )	28.24 ± 4.42	27.3 ± 3.39	0.68
ASA (II/III) (n)	2/18	3/17	0.48
EF (%)	56.45 ± 14.15	57.75 ± 11.36	0.75
Drugs (Beta blocker/ACEI /Ca channel blocker)	15/3/2	16/2/2	1.00

TV: Tidal volume; DB: Deep breath; ASA: American Society of Anesthesiologists; BMI: Body mass index; EF: Ejection fraction; ACEI: Angiotensin converting enzyme inhibitors. Data are presented as mean ± SD, numbers, or %.

groups at the end of preoxygenation, the reduction was not considered clinically significant as the values were within the physiological ranges. A statistically significant reduction in pH outside the physiological ranges was evident at the end of apnea in both groups, compared to the control and the end of the preoxygenation values (Table 3).

#### 4. Discussion

Our study revealed that the 8 DBs/60 s and the TVB/3 min techniques affect the patients' hemodynamic response similarly. Arterial oxygen pressure was higher in the 8DBs

method, but it was similar in the two groups at the end of the apneic period.

Myocardial protection in CABG operations must start at anesthesia induction, and all situations that may affect the hemodynamics should be avoided. For this reason, in our study, we aimed primarily to investigate the effects of the preoxygenation methods on hemodynamics during preoxygenation. DBs trigger the Hering-Breuer reflex (10). This reduces chemoreflex sensitivity by augmenting baroreflex, and reduces blood pressure and sympathetic activity (11,12). Slow DBs improve autonomic balance and respiratory control while reducing blood pressure

**Table 2.** Comparison of the hemodynamic parameter values.

Characteristics	Group A n: 20 (TV/3 min)	Group B n: 20 (8DBs/60 s)	P
MAP <sub>Cont</sub> (mmHg)	100.10 ± 14.75	93.65 ± 11.38	0.13
MAP <sub>T<sub>1</sub></sub>	100.80 ± 14.09	93.20 ± 14.08	0.09
MAP <sub>T<sub>2</sub></sub>	92.10 ± 16.01	85.55 ± 9.37	0.41
HR <sub>Cont</sub> (beat/min)	70 (65.0–76.25)	72 (59.25–85.25)	0.92
HR <sub>T<sub>1</sub></sub>	69.10 ± 11.72	77.15 ± 16.23	0.08
HR <sub>T<sub>2</sub></sub>	68.50 (64.25–79.75)	74.50 (67.25–89.75)	0.37
CI <sub>Cont</sub> (L/min/m <sup>2</sup> )	2.54 ± 0.44	2.80 ± 0.71	0.16
CI <sub>T<sub>1</sub></sub>	2.41 ± 0.49	2.76 ± 0.67	0.06
CI <sub>T<sub>2</sub></sub>	2.23 ± 0.56	2.61 ± 0.61	0.05
SVI <sub>Cont</sub> (mL/m <sup>2</sup> /pulse)	35.85 ± 7.78	38.75 ± 9.34	0.29
SVI <sub>T<sub>1</sub></sub>	34.20 ± 7.64	37.45 ± 9.55	0.24
SVI <sub>T<sub>2</sub></sub>	30.70 ± 8.02	34.90 ± 8.12	0.10
SVRI <sub>Cont</sub> (dynes-s/cm <sup>-5</sup> /m <sup>2</sup> )	2920 ± 673.7	2525 ± 632.0	0.06
SVRI <sub>T<sub>1</sub></sub>	3052 ± 748	2608 ± 700	0.06
SVRI <sub>T<sub>2</sub></sub>	2829 ± 801	2610 ± 837	0.40
CVP <sub>Cont</sub> (mmHg)	12.20 ± 4.25	12.85 ± 4.06	0.62
CVP <sub>T<sub>1</sub></sub>	12.40 ± 4.44	10.70 ± 4.11	0.21
CVP <sub>T<sub>2</sub></sub>	11.80 ± 3.95	12.70 ± 4.60	0.51

TV: Tidal volume; DB: Deep breathing; MAP: Mean arterial pressure; HR: Heart rate; CI: Cardiac index; SVI: Stroke volume index; SVRI: Systemic vascular resistance index; CVP: Central venous pressure; Cont: Control prior to preoxygenation; T<sub>1</sub>: End of preoxygenation; T<sub>2</sub>: End of apnea.

Data presented as mean ± SD or median (25th–75th percentile).

in patients with hypertension (13). Further respiratory sinus arrhythmia can also lead to DBs (14). We predicted a higher decrease in blood pressure with the DB method compared to the TVB technique and an increased HR at the start of our study, yet our results did not support our hypothesis. All the hemodynamic data appeared similar to the control values throughout preoxygenation and apneic period in both groups. In our study, the hemodynamic effects of hyperoxemia may have interfered with those of the preoxygenation method, which may have affected our results. Hyperoxia induces a drop in HR and CO and a rise in blood pressure and vascular resistance occurs in healthy individuals (15–17). In contrast, some studies showed no relation between blood pressure and hyperoxia or a reduction in blood pressure (18,19). These differences are induced by a chemoreceptor or baroreceptor reflex (15). The cardiovascular response to hyperoxia may

vary between different patient groups. Peripheral blood pressure does not change in patients with hypertension or it can also decrease (20–23). In patients with heart failure, hyperoxia reduces CO and SV and increases SVR and pulmonary capillary wedge pressure resistance (24,25). Therefore, we preferred to conduct our study in patients with an EF greater than 40% in order to avoid the possible detrimental effects of hyperoxia. Because we compared the cardiovascular effects of different preoxygenation techniques rather than the cardiovascular effects of hyperoxemia, we did not have a nonpreoxygenation control group.

The hemodynamic response during preoxygenation has not been sufficiently investigated (26). There are only a few studies in the literature on the hemodynamic effects of the preoxygenation methods (27,28). In a noncardiac surgery study, no changes were detected in blood pressure

**Table 3.** Comparison of arterial blood gas values.

Characteristics	Group A n: 20 (TV/3 min)	Group B n: 20 (8DBs/60 s)	P
SaO <sub>2cont</sub> (%)	96.84 ± 1.70	97.54 ± 0.79	0.10
SaO <sub>2T1</sub>	99.30 (98.80–99.57)	99.40 (99.20–99.67)	0.22
SaO <sub>2T2</sub>	99.05 (98.80–99.27)	99.10 (98.35–99.47)	0.93
P**	P < 0.001 <sup>x,y</sup>	P < 0.001 <sup>x,y</sup>	
PaO <sub>2cont</sub> (mm Hg)	98.50 ± 25.06	95.0 ± 12.74	0.18
PaO <sub>2T1</sub>	256.50 ± 92.24	319.05 ± 96.79	0.03*
PaO <sub>2T2</sub>	252.65 ± 99.05	301.01 ± 100.39	0.15
P**	P < 0.001 <sup>x,y</sup>	P < 0.001 <sup>x,y</sup>	
Ph <sub>cont</sub>	7.40 ± 0.04	7.40 ± 0.01	0.84
PhT1	7.36 ± 0.05	7.39 ± 0.05	0.10
PhT2	7.31 ± 0.05	7.31 ± 0.04	0.85
P**	P < 0.001 <sup>x,y,z</sup>	P < 0.001 <sup>y,z</sup>	
PaCO <sub>2Cont</sub> (mm Hg)	37.50 ± 4.66	37.45 ± 3.03	0.96
PaCO <sub>2T1</sub>	41.90 ± 6.03	38.75 ± 6.19	0.11
PaCO <sub>2T2</sub>	52.35 ± 6.53	52.90 ± 5.84	0.78
P**	P < 0.001 <sup>y,z</sup>	P < 0.001 <sup>y,z</sup>	

\*P < 0.05; there were statistically significant differences between the groups

\*\*P < 0.05; there was a statistically significant difference in the groups.

Cont: Control prior to preoxygenation, T1: End of preoxygenation, T2: End of apnea

x: Statistical significance in comparison with the control and T1 values within the group,

y: Statistical significance in comparison with the control and T2 values within the group,

z: Statistical significance in comparison with the T1 and T2 values within the group.

Data presented as mean ± SD or median (25th–75th percentile).

or HR with either of the two methods, while in another study it was reported that preoxygenation did not induce any alterations in blood pressure but the TV method induced a higher decrease in MAP, compared to the 8 DBs/60 s method at the end of apnea (27,28). On the other hand, hemodynamic response is not reported in studies where preoxygenation is investigated in cardiac surgery (4,29).

Studies related to preoxygenation have focused on efficacy and efficiency. While alveolar O<sub>2</sub> and alveolar N<sub>2</sub> or PaO<sub>2</sub> measurements are related to the efficacy of preoxygenation, decrease in the SaO<sub>2</sub> levels during apnea indicates efficiency (7). It is not ethical to lower the crucial SpO<sub>2</sub> levels to critical levels in this group of patients with coronary arterial disease. For this reason, we were not able to investigate the desaturation period in our study. Thus, in our study, we evaluated the efficacy of the preoxygenation

technique only. The fact that the duration of desaturation was not recorded may be one of the limitations of our study.

Baraka et al. (4) found similar PaO<sub>2</sub> levels with both methods in CABG patients and a longer desaturation period with the SDBs technique. In another study, the 8DBs method was related to both increased PaO<sub>2</sub> levels and a longer desaturation period compared to the TV technique (27). Different desaturation threshold levels are defined in these two studies, as 95% by Baraka et al. (4) and 92% by Sing et al. (27). According to Nimmagada et al. (7) the reason for the long desaturation times with 8 DBs is the shift of the oxyhemoglobin dissociation curve as a result of hypocapnia to the left. However, some authors have stated that hypocapnia may occur with 8DBs and thus caution should be paid to its harmful effects (30,31). We did not observe hypocapnia with the 8DBs technique.

According to Nimmagada et al. (7), 60 s is not adequate for the storage of O<sub>2</sub> in tissue and venous compartments; it should be extended to 1.5–2 min. Rapid preoxygenation techniques quickly increase the arterial oxygen pressures without leading to an increase in the tissue oxygen stores, and, for this reason, rapid hemoglobin desaturation is more likely to occur compared to the conventional methods (30,31). For maximal preoxygenation, alveolar, vascular, and tissue oxygen saturations should be achieved (31). In our study, arterial oxygen pressure rose more with 8DBs than TV, but fell to similar levels at the end of apneic periods. Our results contradict those of studies that report longer desaturation times with DB as compared to TVB (4,27).

The difference between the results of the preoxygenation studies may be attributed to different patient populations, different anesthesia systems used, and the wide range of fresh gas flows (4 L/min–35 L/min) (6,7). It is reported in the literature that optimal oxygenation may be provided by 3 min normal TVB, with a 5 L/min oxygen flow with conventional respiratory circuits, and that a minimum of 10 L/min gas flow is necessary and adequate for nitrogen washout with the 8DBs method (6,27). We utilized these reports in preparation of our study protocol. We washed out the anesthesia system by O<sub>2</sub> prior to the preoxygenation. We chose the right-size face masks for the patient and placed them to fit correctly without air leakage.

Hyperoxia and preoxygenation in CABG is a subject of debate. The reason is that arterial oxygen tension is the major determinant of coronary artery tonus and

hypoxia is its potent vasodilator (32). Hyperoxia causes a decrease in coronary blood flow and myocardial oxygen consumption. Oxidative inactivation of nitric oxide is responsible of the coronary vasoconstrictor effect (33,34). For this reason, some authors suggest that coronary spasm may be induced with preoxygenation and hyperventilation should be avoided within this period (35,36). On the other hand, the possibility of an unexpected difficult airway in patients that are to undergo general anesthesia is still at a rate of 1%–3% despite advances in airway management techniques (37). Patients with coronary artery disease have been identified as patients at risk to the extent of not being able to tolerate even moderate level hypoxemia; therefore, filling their oxygen stocks with preoxygenation can be more vital than the harmful effects of hyperoxia (38). In addition, although hypoxemia has harmful effects during CPB, oxygen pretreatment in the pre-CPB period may be cardioprotective. Therefore, the benefit/harm rate of hyperoxemia should be evaluated on a case by case basis (39,40).

In conclusion, preoxygenation by 8DBs/60 s/10 L/min and TVB/3 min/5 L/min affected hemodynamics similarly in patients scheduled to undergo CABG surgery. Increased arterial oxygen pressure by the 8DB method indicates the efficacy of this technique. 8DBs can be an alternative to TV only in patients with normal cardiac function. However, our study is a preliminary one carried out only in patients with coronary artery disease with normal ventricular function. Our results should be compared with those of other studies.

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