


High-Flow Nasal Oxygen Prolongs Safe Apnea Time in Obstructive Sleep Apnea Patients Undergoing General Anesthesia: A Randomized Controlled Trial

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Background: Patients with obstructive sleep apnea (OSA) are at high risk for rapid oxygen desaturation during anesthesia induction. Apneic oxygenation with high-flow nasal oxygen (HFNO) has proven effective in prolonging safe apnea time in various patient populations. However, evidence for the efficacy of HFNO in OSA patients remains limited. This study aimed to evaluate whether the use of HFNO during anesthesia induction in OSA patients prolongs safe apnea time.

Methods: In this prospective randomized clinical trial, all participants underwent standardized pre-oxygenation and anesthesia induction. During the apneic period, oxygen was delivered either via HFNO at 60L/min (HFNO group) or with the nasal cannula left in place but disconnected from the oxygen source (control group, no supplemental oxygen). The primary outcome of this study was the time to peripheral oxygen desaturation ($SpO_2 < 95\%$) during apneic oxygenation. Secondary outcomes included minimum SpO_2 , re-oxygenation time, and $tcCO_2$ levels.

Results: The HFNO group demonstrated a significantly prolonged safe apnea time compared to the Control group (18.1 [12.1,18.8] vs 4.2 [2.5,6.3] minutes; $p < 0.001$). Additionally, minimum SpO_2 levels were higher, and re-oxygenation time was shorter in the HFNO group ($p < 0.001$ for both). Kaplan-Meier survival analysis revealed a substantially reduced risk of desaturation in the HFNO group (hazard ratio: 0.071; 95% CI, 0.021–0.222; $p < 0.001$). No serious adverse events were reported.

Conclusion: In this randomized controlled trial, HFNO significantly prolongs safe apnea time and enhances oxygenation during anesthesia induction in OSA patients. These findings highlight the potential of HFNO to improve perioperative airway management and patient safety in this high-risk population.

Keywords: HFNO, OSA, apnea time, difficult airway management, anesthesia induction

Introduction

Difficult airway management remains a persistent challenge in the operating room, particularly in patients with obstructive sleep apnea (OSA), a well-recognized independent risk factor for difficult intubation, difficult mask ventilation, or a combination of both.^{1–3} Moderate to severe OSA patients face a 3-to 4-fold increased risk of difficult intubation, with approximately 1 in 7 (14.5%) severe OSA patients encountering intubation challenges.^{4–6} Physiologically, OSA patients exhibit reduced functional residual capacity and increased oxygen consumption, leading to rapid oxygen desaturation during anesthesia induction.^{7,8} These risks are further compounded by anatomical features such as restrictive cervico-maxillo-mandibular enclosure anatomy, crowded collapsible pharyngeal space, increased oropharyngeal soft tissue. The combination of difficult mask ventilation and rapid desaturation significantly heightens the likelihood of a “cannot intubate, cannot ventilate” scenario, which can result in severe hypoxia, anoxic brain injury, or



even mortality. This underscores the critical importance of employing strategies to optimize oxygenation and extend safe apneic time, ensuring sufficient opportunity to secure the airway and achieve a safe and effective intubation process.^{9,10}

Oxygenation with high-flow nasal oxygen (HFNO) has gained popularity within anesthetic practice for its ability to enhance safety in apneic patients since its introduction by Patel and Nouraei in 2015.¹¹ Studies on HFNO in obese patients, who, like OSA patients, belong to high-risk populations, have demonstrated its potential to significantly extend safe apnea time,^{12–14} offering promise for its application in other challenging patients groups, such as OSA. However, despite those encouraging findings, there is currently no empirical evidence assessing the efficacy of HFNO in OSA patients. Randomized clinical trials evaluating the role of HFNO in OSA patients remain scarce, particularly within the specific perioperative settings which may have the greatest impact.

To date, no randomized controlled trial has evaluated HFNO in OSA patients during anesthesia induction—a phase in which these high-risk patients are particularly prone to rapid desaturation. Addressing this gap, we conducted a single-center randomized controlled study to determine whether HFNO can significantly extend safe apnea time in OSA patients, thereby improving perioperative airway management and patient safety.

Methods

Trial Design and Participants

This single-center, prospective, randomized trial was conducted in the Department of Anesthesiology at Beijing Tongren Hospital in China between April 2022 and July 2024. This study was approved by the Ethics Committee of Tongren Hospital (NO. TREC2021-KY099) and was registered in the Chinese Trials registry (<https://www.chictr.org.cn/showproj.html?proj=132805>) prior to patient enrollment. Written informed consent was obtained from all participants before enrollment. This study adhered to Declaration of Helsinki principles.

Eligible participants were recruited at a national key otorhinolaryngology clinical center. Participants were included if they were >18 yr of age, American society of anesthesiologists (ASA) physical status of I–III, be diagnosed with OSA, and undergoing elective modified uvulopalatopharyngoplasty. Exclusion criteria included: (1) chronic respiratory diseases; (2) oxygen saturation below 98% while on supplemental oxygen; (3) anticipated difficulty with intubation; (4) uncontrolled hypertension; (5) ischemic cardiomyopathy; (6) congestive heart failure; (7) increased intracranial pressure; (8) uncontrolled gastroesophageal reflux disease; (9) known allergy to anesthetics; or (10) inability to use high-flow devices due to nasopharyngeal blockage.

Participants were randomized into two groups: HFNO (HFNO using Optiflow THRIVE™) or Control (no oxygen delivery). Block randomization with a 1:1 ratio was performed using a computer-generated sequence generation and sealed opaque envelopes.

Preoxygenation for both groups was provided via a face mask with an FiO₂ of 1.0 at 10 L/min, with the patient positioned in a 30-degree ramped position. Before induction, tracheostomy trays, cricothyroidotomy equipment, and resuscitation resources were prepared in the operating room. An experienced laryngologist was standby in case of failed intubation or the complete obstruction during induction. One senior anesthetists and an assistant were present throughout the procedure. Anesthesia induction included using opioid, propofol, and rocuronium, with doses selected by the anesthetist, and maintained with intravenous infusions of propofol and remifentanyl to maintain a BIS range of 40–60. Systolic blood pressure was maintained within 20% of baseline values. Manual bag-mask ventilation was performed with PEEP adjusted as needed by the anesthetist. Laryngoscopy was conducted using a CMAC videolaryngoscope, with Cormack-Lehane laryngoscopy grades recorded. Patients with difficulty in manual ventilation or a Grade 3 or 4 laryngoscopy view were excluded at this stage. Following successful ventilation, the videolaryngoscope was removed, and the patient was manually ventilated until an end-tidal oxygen level of 90% was achieved. Nasal oxygen therapy was then applied based on group assignment. An oropharyngeal airway was employed to optimize airway patency until the study endpoint was reached.

Intervention

Participants in the HFNO group received oxygen via the Optiflow THRIVE™ system, with settings of 60 L/min flow rate, 100% inspired oxygen fraction, and a temperature of 37°C (Optiflow; Fisher & Paykel Healthcare, Auckland, New Zealand).

Participants in the Control group wore the nasal cannula of the Optiflow THRIVE™ system with identical device settings (flow rate: 60L / min, FiO₂:100%, temperature: 37°C). However, the oxygen supply tubing was disconnected at the connection point between the oxygen source and the nasal cannula, ensuring no oxygen delivery.

Outcomes

The primary outcome was safe apnea time, defined as the interval from the cessation of mechanical ventilation (start of apnea) until peripheral oxygen saturation (SpO₂) first falls below 95%, or, if desaturation does not occur, until the 18-minute observation limit is reached. The patient remained anesthetized and apneic until one of the following occurred: (1) SpO₂ dropped to 95%, (2) tcCO₂ exceeded 80 mmHg, (3) arrhythmias or other complications arose, or (4) the maximum observation time of 18 minutes was reached. When the endpoint was reached, the nasal cannula was removed, and the patient was manually bag-mask ventilated until SpO₂ reached 98%, followed by intubation performed by attending anesthetists with more than 5 years of airway experience.

Statistical Analysis

The study was powered for the primary outcome. There are few data recommending a certain apnea time in OSA patients under general anesthesia induction. Sample size estimation was based on preliminary data and survival analysis. Using data from a prior study, apnea durations of 261 and 185 seconds were estimated for the HFNO and Control groups, respectively, with an SD of 77 seconds.¹⁴ Type I and type II errors of 5% and 20% were used, yielding an estimated sample size of 36 (18 per group). To account for dropout rates, the sample size was increased by 22%, requiring 44 participants. Survival analysis estimated a failure rate of 90% in the Control group and 12% in the HFNO group at 18 minutes.^{13,15} Adjusted calculations confirmed a total sample size of 44 (Log rank test, Freedman method).

The Shapiro–Wilk test was used to assess the normality of continuous variables. Group characteristics were summarized with means and standard deviations (SD) for normally distributed variables and medians with interquartile ranges (IQR) or 95% confidence intervals (CI) for skewed distributions. Kaplan–Meier curves were used to represent time to desaturation, with comparisons made using the Log rank test. Continuous variables were analyzed with independent *t*-tests or Mann–Whitney *U*-tests as appropriate. Among participants desaturating within 18-minute, the Mann–Whitney *U*-test compared time to desaturation between groups. With statistical significance defined as a two-tailed *p*-value < 0.05. Data were analyzed using SPSS version 28.

Results

Sixty-three participants were assessed for eligibility, and 44 patients were recruited, randomized, and enrolled in the study. One patient developed anaphylactic shock during induction and was excluded from the analysis. A total of 43 patients were included in the final analysis, with 22 in the HFNO group and 21 in the control group. All participants completed the study (Figure 1). No serious adverse events were reported.

The patient characteristics were comparable between the two groups (Table 1). The study was conducted in a population with obstructive sleep apnea, there was a greater percentage of participants were male (79.1%), due to the sex differences and gender bias in OSA. Men are more likely to present with typical symptoms that align with diagnostic criteria. The majority of patients (90.7%) were diagnosed with moderate or severe OSA, and more than half (55.8%) experienced severe hypoxemia.

The primary and secondary outcomes are summarized in Table 2. Median safe apnea time was 18.1 min [12.1–18.8] in the HFNO group versus 4.2 min [2.5–6.3] in the control group (*P* < 0.001), although interpretability is limited by heavy right censoring, as nearly half of the HFNO group did not desaturate within 18 minutes. Among the 30 patients who desaturated, time to desaturation was significantly longer in the HFNO group (mean=10.9; SD=4.6 min; *n*=9) compared to the control group (mean=4.5; SD=2.7 min; *n*=21), and was statistically significant (independent *t* test *P*= 0.009).

The Kaplan–Meier survival curves for each group are shown in Figure 2. All (100%) in the control group and 9 patients (40.9%) in the HFNO group desaturated to an SpO₂ of 95% within 18 min of apnea. The risk of desaturation was significantly lower in the HFNO group compared to the control group (hazard ratio=0.071; 95% CI, 0.021–0.222; Log rank test *P* < 0.001).

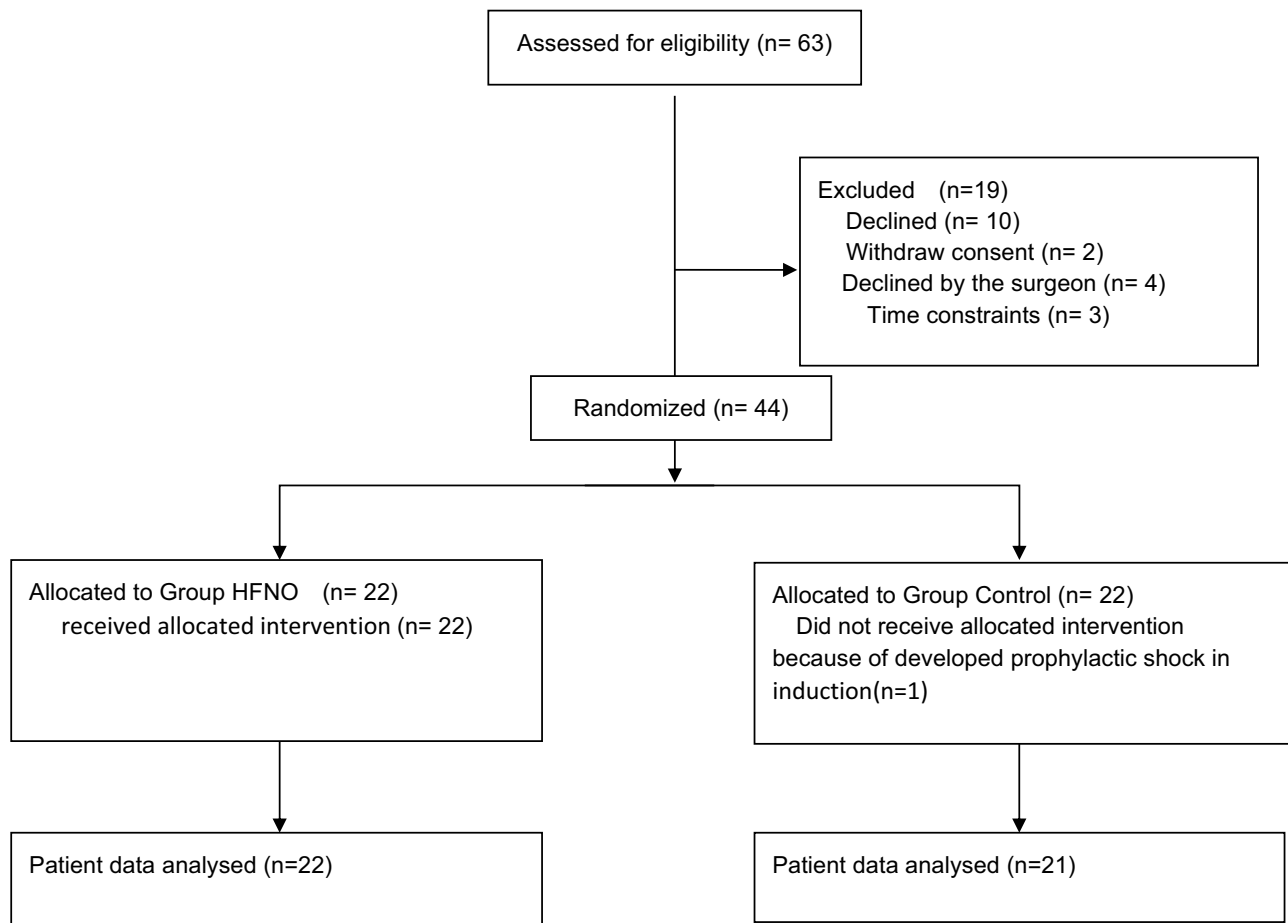


Figure 1 Study flow diagram. Enrollment, randomization, and follow-up of all participants. **Abbreviations:** HFNO, high-flow nasal oxygen.

The mean (SD) CO₂ accumulation was similar between the two groups (Table 2). The median minimum SpO₂ was significantly higher in the HFNO group (Mann–Whitney-*U* test, *P*< 0.001), and the median re-oxygenation time was significantly shorter in the HFNO group (Mann–Whitney-*U* test, *P*= 0.047).

Table 1 Subject Characteristics

	Group HFNO	Group Control	p-value
Participants number	22	21	
Age (yr)	49.2 (15.6)	45.7 (16.7)	0.518
Sex, n (%)			
Female	4 (18.2)	5 (23.8)	0.534
Weight (kg)	84.7 (22.1)	82.4 (17.2)	0.728
Height (cm)	167.27 (9.2)	168.9 (9.7)	0.702
BMI (kg m-2)	29.1 (6.64)	28.5 (4.4)	0.774
ASA physical status, n (%)			0.486
II	20 (90.9)	21 (100.0)	
III	2 (9.1)	0 (0.0)	

(Continued)

Table 1 (Continued).

	Group HFNO	Group Control	p-value
Airway assessments			1.000
History of difficult intubation, n (%)	0 (0.0)	0 (0.0)	
Interincisor distance < 3cm, n (%)	0 (0.0)	0 (0.0)	
Thyromental distance < 6.5 cm, n (%)	10 (45.5)	12 (57.1)	
Neck mobility < 30°, n (%)	0 (0.0)	0 (0.0)	
Neck circumference (cm)	42.5 (5.4)	41.7 (4.8)	0.699
Mallampati score (I/II/III/IV)	5/10/4/3	3/12/4/2	0.846
Cormack-Lehane grade (I/II)	18/4	17/4	1.000
Polysomnography results			
AHI	39.3 [24.2,72.7]	35.0 [23.1,56.3]	0.551
Baseline SpO ₂ (%)	98 [96,100]	98 [97,100]	0.441
Minimum SpO ₂ (%)	77.5 [71.0,84.0]	79.5 [74.7,84.7]	0.432
Severity of hypoxemia, n (%)			0.685
Mild	2 (9.1)	4 (19.0)	
Moderate	7 (31.2)	6 (28.6)	
Severe	13 (59.2)	11 (52.4)	
Severity of OSA, n (%)			0.755
Mild	2 (9.1)	2 (9.5)	
Moderate	6 (27.3)	8 (38.1)	
Severe	14 (63.6)	11 (52.4)	
Tiredness, n (%)	8 (36.4)	9 (42.9)	1.000
Snoring, n (%)	22 (100)	21 (100)	1.000
Duration of snoring history (yr)	8 [5,18]	10 [4.5,20]	0.780
Nocturnal awakening due to dyspnea, n (%)	16 (72.7)	14 (66.7)	1.000
Comorbidities, n (%)			
Hypertension	12 (54.5)	8 (38.1)	0.482
Diabetes	5 (23.7)	5 (23.8)	0.609
Coronary heart disease	1 (4.5)	0 (0.0)	1.000
Hyperlipidemia	12 (59.1)	9 (42.9)	0.732
Smoker (current/ex/never)	4/12/6	5/10/6	0.738
Alcohol consumption	11 (50.0)	7 (33.3)	0.487
COPD	0 (0.0)	0 (0.0)	1.000
Asthma	0 (0.0)	0 (0.0)	1.000
Pulmonary inflammation	0 (0.0)	0 (0.0)	1.000

Notes: Continuous data are presented as median (interquartile range) or mean (standard deviation).

Abbreviations: HFNO, high-flow nasal oxygen; BMI, body mass index; ASA, American society of anesthesiologists; SpO₂, peripheral oxygen saturation; AHI, apnea hypopnea index; OSA, obstructive sleep apnea; COPD, chronic obstructive pulmonary disease.

Table 2 Primary and Secondary Outcomes

	Group HFNO (N=22)	Group Control (N=21)	P-value
Primary outcome			
Safe apnea time (min)	18.1 [12.1,18.8]	4.2 [2.5,6.3]	<0.001
No. patients reached 18 min, n (%)	9 (40.9)	0 (0)	<0.001
No. patients reached desaturated to 95%	9 (40.9)	21 (100)	0.014
tcCO ₂ ≥ 80, n (%)	3 (13.6)	NA	
Arrhythmia, n (%)	1 (4.5)	0 (0)	0.879

(Continued)

Table 2 (Continued).

	Group HFNO (N=22)	Group Control (N=21)	P-value
Secondary outcome			
Minimum SpO ₂ (%)	98 [94, 99]	92 [90, 94]	< 0.001
Rate of tcCO ₂ accumulation (mmHg·min ⁻¹)	2.5 (0.6)	NA	
Rate of ETCO ₂ accumulation (mmHg·min ⁻¹)	2.0 [1.4,2.1]	1.6 [1.3, 3.7]	0.240
Peak ETCO ₂ in first 5 breaths post-apnea (mmHg)	59 [56,66]	34 [32,40]	<0.001
Reoxygenation time (s)	10.5 [5.2,16.5]	28.5 [13.5, 46.7]	0.047
tcCO ₂ post-apnea (mmHg)	58 [50,62]	NA	
SpO ₂ initial-apnea (%)	100 [99,100]	100 [99,100]	0.194
SpO ₂ post-apnea (%)	98 [95,98]	95 [95,95]	0.004
HR initial-apnea	72.0 (10.9)	73.1 (12.3)	0.773
HR post-apnea	84.2 (14.2)	72.7 (9.5)	0.014
Arrived at PACU SpO ₂ (%)	98 [95,100]	97 [95,99]	0.418
Arrived at PACU HR	76.3 (9.8)	74.1 (8.6)	0.550
PACU stay length (min)	41.0 [30.0,52.7]	45.0 [31.5,56.5]	0.685

Notes: Continuous data are presented as median (interquartile range) or mean (standard deviation).

Abbreviations: HFNO, high-flow nasal oxygen; tcCO₂, transcutaneous carbon dioxide; SpO₂, peripheral oxygen saturation; ETCO₂, end-tidal carbon dioxide; HR, heart rate; PACU, post-anesthesia care unit; NA, not applicable (variable not measured in this group by study design).

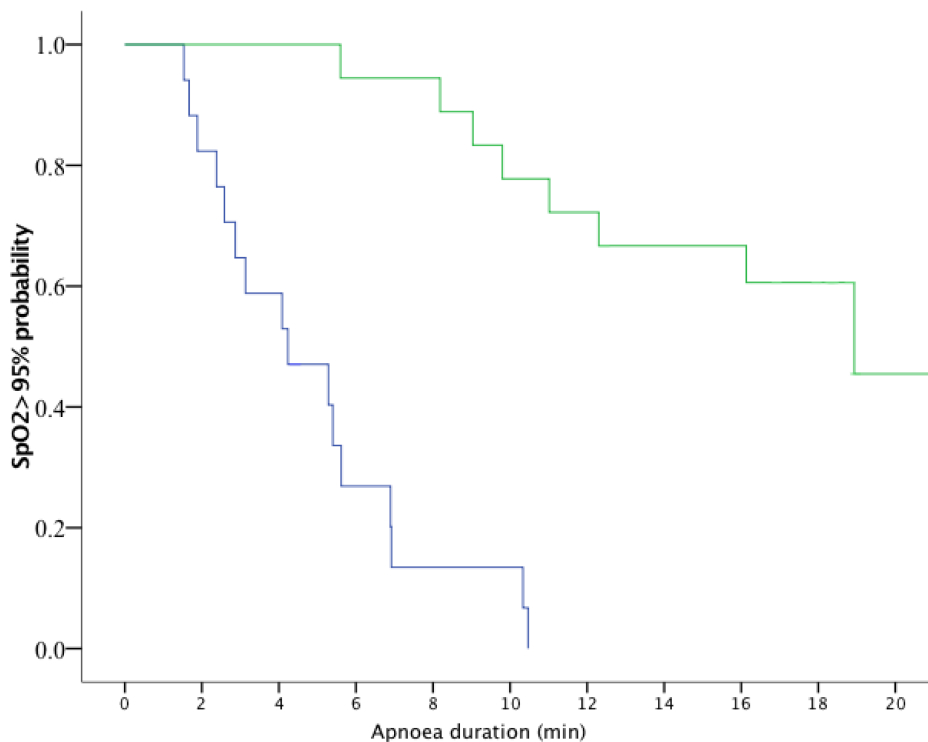
Discussion

Our results suggest a clear effect of HFNO administered at 60L/min in extending safe apnea time and improving perioperative oxygenation in paralyzed OSA patients. To the author's knowledge, based on the literature, this presents the first randomized controlled trial examining HFNO as apneic oxygenation in OSA patients during anesthesia induction. The findings contribute significantly to the limited available evidence on this topic, addressing a critical gap in airway management for this high-risk patient group.

HFNO has previously been shown to prolong apnea duration in obese patients, a population at high risk group for difficult airway management.^{12,14,16–19} Our results extend these findings by demonstrating that HFNO significantly increases safe apnea time in OSA patients, who, beyond sharing reduced functional residual capacity and rapid desaturation risk, also contend with pharyngeal narrowing, soft-tissue collapsibility, and airway collapse under muscle relaxation.^{1,5,20–22} These distinctions underscore the need for OSA-specific investigations, since evidence derived solely from obese cohorts may not capture the full spectrum of airway dynamics in OSA. By prolonging safe apnea time, HFNO enables uninterrupted intubation even during prolonged procedures and alleviates the pressure on anesthesiologists to achieve rapid intubation by preventing desaturation in anticipated difficult airways. Furthermore, HFNO mitigates the risk of catastrophic outcomes, particularly in challenging airway scenarios such as “cannot intubate, cannot ventilate” situations, thereby enhancing patient safety and providing clinicians with critical time for effective airway management.

In our study, 9 of 22 HFNO recipients (40.9%) maintained stable oxygenation throughout the 18-minute observation period. This proportion is lower than the 88% observed by Schutzer-Weissmann et al's in obese patients under identical HFNO settings.¹³ While differences in patient positioning (45°incline versus our 30°ramp) may partially account for this discrepancy, the dominant factor is likely the unique anatomical limitations in OSA, exacerbated by anesthetic muscle relaxation.

Importantly, over 90% of HFNO patients sustained safe oxygenation for at least 10 minutes, a duration sufficient for securing the airway in most clinical scenarios. Conversely, 76% of controls desaturated within 6 minutes, a rate similar to that reported by Guy et al despite their use of 4L/min supplemental oxygen.¹² However, our control group did not receive supplemental oxygen, as we aimed to simulate real clinical scenarios where oxygenation is typically paused during intubation. This differs from previous studies,^{12,13,15,23,24} where face mask is provided, which is impractical during actual intubation. Our results shows the safe apnea time in OSA patients without oxygenation during apnea was 4.2 minutes,



Number at risk	
HFNO	22 22 22 21 20 18 17 15 13 9
Control	21 17 11 5 2 2 0 0 0 0

Figure 2 Kaplan-Meier curve of time to SpO₂≤95% for the two study groups. Group HFNO (green line) and Group Control (purple). Hazard ratio comparing Control and HFNO groups, 0.071 (95% confidence interval, 0.021–0.222; log-rank P< 0.001). Number at risk represents number of patients yet to reach SpO₂≤95% at a time point of every 2 minutes from the beginning of apnea to a maximum of 18 minutes.

Abbreviations: HFNO, high-flow nasal oxygen; SpO₂, peripheral oxygen saturation.

which was comparable or litter shorter than that observed in obese patients in former study.^{14,15} Notably, the BMI of patients in our study was not high, whereas patients in those studies had a higher BMI greater than 40.

In this study, the rate of increase of carbon dioxide was not directly measured to avoid the risks associated with invasive arterial blood gas analysis. Instead, we monitored tcCO₂ in the HFNO arm as a safety threshold, terminating observations at 80 mmHg. In the control arm, apneic intervals were too brief for tcCO₂ to stabilize or reliably track PaCO₂.²⁵ Even if PaCO₂ rose by 10 mmHg in the first minute,²⁶ subsequent accumulation is only ~2 mmHg·min⁻¹,^{27,28} precluding control patients from ever reaching the 70 mmHg safety limit within the observation window. Thus, tcCO₂ monitoring could not have provided actionable data in controls and was reserved for the HFNO arm. Emerging AI-driven predictive models may soon enable noninvasive estimation of PaCO₂ trends, further enhancing perioperative safety.²⁹

Although the rate of increase in ETCO₂ in our study was similar between groups, previous research has demonstrated significant discrepancies between PaCO₂ and ETCO₂ levels, indicating that ETCO₂ is an unreliable indicator of CO₂ accumulation following apnea.¹² Riva et al²⁷ reported a consistent PaCO₂ increase rate of approximately 2 Hg min⁻¹ across various flow rates (0.25–70Lmin⁻¹), comparable to 2.2 Hg min⁻¹ in obese patients.^{12,13,30} Similarly, Schutzer’s study found no significant difference in PaCO₂ increase between HFNO and face mask groups,¹³ reinforcing that the oxygenation method does not effect CO₂ accumulation during apnea. Based on the widely accepted rate of 2 mmHg min⁻¹,^{27,28} the preset 18-minute apnea time in our study was considered safe.

Limitations of the study include the following. First, it was a small single-center study with a larger proportion of male participants, so results may not be generalizable. Second, arterial blood gas was not analyzed in our study, as it is

not a routine measurement taking during surgery and could increase the risk to the risk due to invasive manipulation, carbon dioxide accumulation was unable to be clarified in this study.

In conclusion, apneic oxygenation with HFNO at 60L/min significantly increases safe apnea time and reduces the risk of desaturation in OSA patients during general anesthesia induction. These findings suggest that HFNO is a promising approach to enhance OSA patient safety, further research is needed to confirm its efficacy and guide clinical recommendations.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article or are available from the corresponding author upon reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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