ORIGINAL ARTICLE

COMPARISON OF HIGH-FLOW NASAL OXYGEN AND CONVENTIONAL LOW-FLOW OXYGEN DURING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY UNDER SEDATION IN GERIATRIC AND ADULT PATIENTS

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Abstract

Introduction: Sedated endoscopic retrograde cholangiopancreatography is associated with hypoxaemia and haemodynamic instability, particularly in geriatric patients. High-flow nasal oxygen may improve oxygenation compared with conventional oxygen therapy, yet its benefits in elderly patients undergoing endoscopic retrograde cholangiopancreatography remain unclear.

Materials and Method: In this prospective randomized study, 100 adult patients undergoing elective endoscopic retrograde cholangiopancreatography were randomly assigned (1:1) to receive either High-flow nasal oxygen ($50 L \cdot min^{-1}$ with FiO_2 0.50) or conventional oxygen therapy (4L/min.). Patients were randomly allocated into four groups: Group I:Adult (<65 years)+High-flow nasal oxygen, n=25), Group II:Geriatric (≥65 years)+High-flow nasal oxygen, n=25), Group III:Adult (<65 years) + conventional oxygen therapy, n=25), Group IV: Geriatric (≥65 years)+conventional oxygen therapy, n=25). The primary outcomes were hypoxaemia ($SpO_2 \leq 90\%$) and hypotension (systolic arterial pressure <90 mmHg or ≥20% decrease from baseline). Secondary outcomes included haemodynamic variables, end-tidal CO_2, sedative consumption, and recovery time.

Results: High-flow nasal oxygen significantly reduced hypoxaemic events, particularly in geriatric patients ($p<0.05$). SpO_2 values remained more stable in the elderly High-flow nasal oxygen group, especially at the 15th and 30th minutes. End-tidal CO_2 remained within physiological ranges in all groups. Systolic arterial pressure decreased in all groups at 5 minutes; reductions were more pronounced in geriatric patients irrespective of oxygenation strategy. Recovery time was shorter in adults receiving High-flow nasal oxygen ($p<0.05$).

Conclusion: High-flow nasal oxygen improves oxygenation and reduces hypoxaemia during sedated endoscopic retrograde cholangiopancreatography, with the greatest benefit observed in geriatric patients. However, advanced age remains an independent risk factor for haemodynamic instability. Careful sedative titration and close monitoring are essential in elderly patients.

Keywords: Cholangiopancreatography, Endoscopic Retrograde; Oxygen Inhalation Therapy; Aged; Oxygen Saturation; Hypotension; Procedural Sedation.

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is an advanced endoscopic technique widely utilized in the diagnosis and treatment of biliary and pancreatic disorders (1).

Although it is routinely performed, the procedure is associated with a non-negligible risk of complications, including cardiopulmonary events and a small but measurable mortality rate (2). To improve patient comfort and procedural tolerance, sedation and analgesia are commonly administered during ERCP. However, the use of sedation introduces additional risks, particularly those related to respiratory compromise (3). The prone positioning required for ERCP further exacerbates these risks by altering respiratory mechanics. Increased intra-abdominal pressure and reduced chest wall compliance may impair ventilation, predisposing patients to hypoxaemia. This issue is particularly relevant in older adults. Age-related physiological decline, reduced pulmonary reserve, and the presence of comorbidities increase susceptibility to sedation-related adverse events. Consequently, maintaining adequate oxygenation during ERCP remains a critical challenge in geriatric populations (4,5).

High-flow nasal oxygen (HFNO) has emerged as an alternative oxygen delivery method that provides heated, humidified oxygen at high flow rates. This approach may enhance oxygenation by reducing dead space, ensuring a stable fraction of inspired oxygen, and generating a degree of positive airway pressure. Compared with conventional low-flow oxygen therapy, HFNO has the potential to offer superior respiratory support during sedated procedures (6).

The present study was designed to evaluate the effectiveness of HFNO compared with conventional oxygen therapy in adult and geriatric patients undergoing ERCP under sedation. Additionally, it aimed to identify factors associated with sedation-related complications, with particular emphasis on hypoxaemia and haemodynamic instability.

MATERIALS AND METHOD

This prospective randomized single-center study was conducted in the Department of Anesthesiology and Reanimation at Prof. Dr. Cemil Taşcıoğlu City Hospital, İstanbul, Türkiye, between (April 2025) and (December 2025). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (a revised 2013). The study protocol was approved by the Institutional Ethics Committee (Approval No. 137, Date: 14.04.2025) and was registered at ClinicalTrials.gov (Identifier: NCT07493811.) Written informed consent was obtained from all participants before enrollment. Patients classified as geriatric (≥65 years) or adult (<65 years), with American Society of Anesthesiologists physical status I–III and scheduled for ERCP under sedation, were included in the analysis.

Study Design

Patients were randomly allocated to four groups. Randomization was performed using a computer-generated random allocation sequence prepared by an independent researcher who was not involved in patient care or outcome assessment.

Group I: Adult + HFNO

Group II: Geriatric + HFNO

Group III: Adult + COT

Group IV: Geriatric + COT

Inclusion criteria comprised patient aged ≥18 years who were scheduled for elective ERCP under monitored anaesthesia care, had ASA physical status I–III and were able to provide written informed consent.

The exclusion criteria consisted of severe organ diseases (cardiac, pulmonary, renal, neurological, or hepatic), unstable blood pressure (SAP <90 mmHg or >170/100 mmHg), and baseline hypoxaemia

(SpO_2 <90%). Also excluded were patients with hypersensitivity to the medications, prior adverse sedation events, recent sedative use (within 24 hours), or those who refused consent.

The sample size was calculated a priori using G*Power software version 3.1.9.7. Based on previous studies evaluating hypoxaemia during gastrointestinal endoscopy under sedation, the incidence of hypoxaemia was assumed to be 30% with conventional oxygen therapy and 10% with HFNO. Using a two-sided χ^2 test with $\alpha = 0.05$ and 80% statistical power, the required sample size was calculated as 23 patients per group. To compensate for potential exclusions, 25 patients were recruited into each group.

A total of 105 patients were assessed for eligibility. Five patients were excluded due to baseline SpO_2 <90%. One hundred patients were randomised equally into four groups (n = 25 each). No patients were lost to follow-up, and all randomised participants were included in the final analysis (Figure 1).

Due to the nature of the intervention, the anaesthesiologist administering oxygen therapy was not blinded. However, outcome assessors and statistical analysts were blinded to the group allocation.

Sedation Protocol

All patients underwent overnight fasting prior to the procedure. Approximately 20 min. before sedation, intravenous midazolam was administered at 0.05mg·kg^{-1}, and local anaesthesia of the oropharynx was achieved with 4% lidocaine spray.

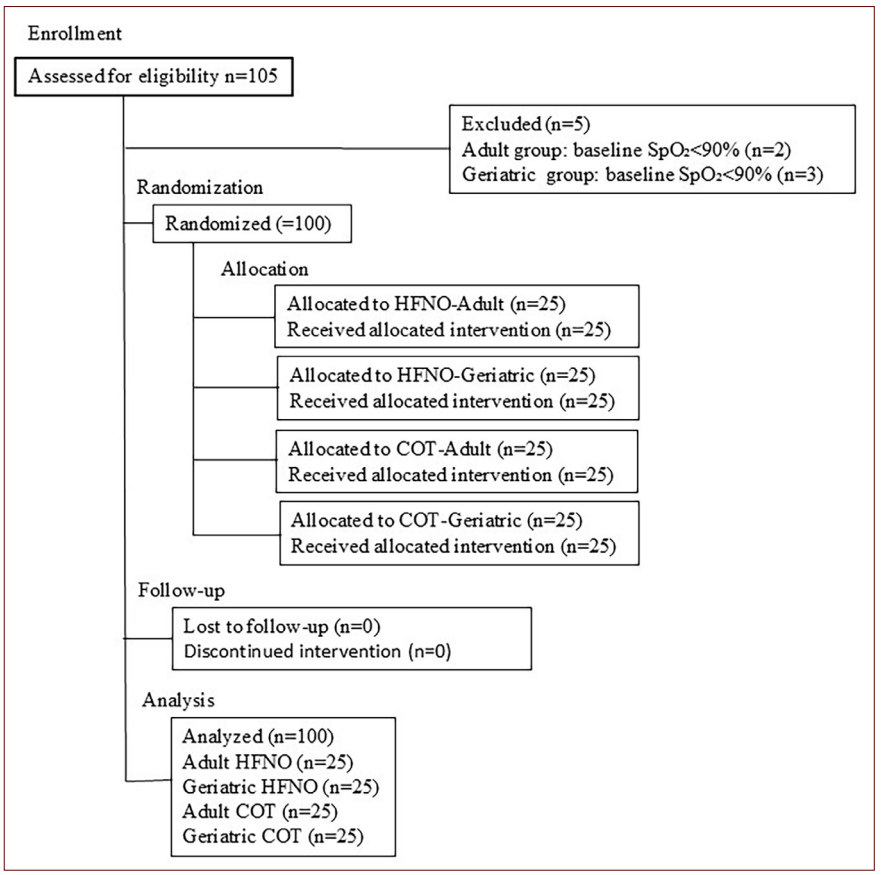


Figure 1. Flow diagram of patient enrollment, randomization, allocation, flow-up and analysis.



Patients were then placed in the prone position with the head turned to the right. Peripheral venous access was established in the left upper extremity using a 20-gauge intravenous catheter, which was connected to an infusion system for drug administration. Sedoanalgesia was initiated with fentanyl in combination with propofol. The induction dose of propofol was age-stratified: 40 mg for patients under 65, 30 mg for those aged 65–89, and 20 mg for individuals 90 years or older.

For maintenance, a ketofol solution in a 1:1 ratio was prepared by combining ketamine, propofol, and saline to achieve final concentrations of 5 mg.mL^{-1} for each agent. This mixture was infused at a fixed rate of 0.8 mL.kg^{-1}h^{-1} across all age groups. Oxygen supplementation differed between groups. In Groups III and IV, patients received low-flow oxygen via nasal cannula at 4 L/min. In contrast, Groups I and II were managed with high-flow nasal oxygen delivered through a heated and humidified system, set at 50 L/min. with an FiO_2 of 0.50, aiming to maintain oxygen saturation at or above 95% and a temperature of 37°C. The high-flow system also enabled continuous end-tidal carbon dioxide ($EtCO_2$) monitoring through modified nasal interfaces suitable for procedural sedation. Standard monitoring included heart rate, non-invasive blood pressure, oxygen saturation, and $EtCO_2$ levels. Additionally, cumulative doses of sedative and analgesic agents were recorded. In cases of mild oxygen desaturation (SpO_2 between 90% and 95%), airway patency was improved using a jaw-thrust manoeuvre. If oxygen saturation dropped to 90% or below, the oxygen flow was increased to 60 L/min., followed by escalation of FiO_2 to 100% if necessary to restore adequate saturation. Maximum limits for flow and saturation were set at 60L/min. and 100%, respectively. In situations where desaturation was accompanied by agitation, particularly in patients receiving midazolam, flumazenil was administered. The endoscopic procedure was discontinued if oxygen saturation remained at or below 90% despite these interventions.

Intraprocedural Management

Hypoxaemia was defined as SpO_2 <90%. Subclinical respiratory depression (90% ≤ SpO_2 <95%) was managed using airway manoeuvres, such as jaw thrust. If SpO_2 ≤90% occurred, HFNO flow was increased to 60 L·min^{-1} and FiO_2 to 100%. If desaturation persisted or was associated with agitation in patients receiving midazolam, flumazenil was administered. The procedure was interrupted if SpO_2 ≤90% did not resolve despite these measures. Hypotension was defined as SAP <90 mmHg or a ≥20% reduction from baseline. Persistent hypotension (>1 min.) was treated with an intravenous fluid bolus and 5 mg ephedrine. In cases of bradycardia (HR <50 bpm), a 0.5 mg intravenous bolus of atropine was administered.

Outcome Measures

Primary outcomes included the incidence of hypoxaemia, hypotension, bradycardia and changes in SAP, HR and SpO_2. Secondary outcomes included total sedative drug consumption and recovery time, defined as the time from cessation of sedative infusion to achievement of a Richmond Agitation–Sedation Scale (RASS) score of 0. Sedation depth was assessed using the Ramsay Sedation Scale, targeting a score ≥ 5. ERCP was performed by an experienced endoscopist who had completed more than 500 procedures.

Following the procedure, all infusions were discontinued, drug doses were recorded and patients were transferred to the post-anaesthesia care unit. Recovery was assessed using the modified Aldrete score, and patients with scores ≥ 9 were transferred to the ward.

Statistical Analysis

Statistical analyses were conducted using Stata/SE 18.0 (StataCorp LLC, USA), with significance set at

a two-tailed p <0.05. Data normality was evaluated through Shapiro–Wilk tests and visual inspection (histograms and Q–Q plots). Continuous variables are reported as mean ± SD or median (IQR) based on their distribution, while categorical data are presented as frequencies and percentages. Group comparisons utilized one-way ANOVA with Tukey's HSD or the Kruskal–Wallis test for continuous data, and Pearson's χ^2 or Fisher's exact tests for categorical variables. Clinical relevance was further assessed using η^2 and Cramér's V effect sizes.

Repeated-Measures Analysis: Time-dependent changes in SAP, HR and SpO2 were analysed using a linear mixed-effects model to account for within-subject correlation.

The model included fixed effects group, time and group × time interaction; random effect – subject-level intercept; and estimation method – restricted maximum likelihood. Model assumptions were verified by residual analysis. When a significant interaction term was detected, post hoc pairwise comparisons were conducted with Bonferroni adjustments for multiple testing.

Sensitivity analyses: To evaluate whether age independently predicted hypoxaemia and hypotension, multivariable logistic regression analyses were performed, including age group, oxygenation method, propofol dose and ketofol dose as covariates. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported.

RESULTS

Patients undergoing sedated ERCP were randomly assigned to four distinct groups. Statistical analysis revealed no significant disparities between these groups regarding gender distribution or ASA physical status classification.

In time-based analyses (pairwise comparisons), SAP decreased significantly at 5 min. compared with baseline in all groups ($p < 0.05$). The most pronounced decreases were observed in Groups II and IV. In Group II, the decrease at 5 min. was −14.8 mmHg ($p < 0.001$), and this reduced level remained statistically significant up to 30 min. ($p = 0.005$). In Group IV (≥65 years, COT), a marked decrease was observed at 5 min. (−12.76 mmHg) and 15 min. (−8.4 mmHg); however, unlike the elderly HFNO group (Group II), SAP values at 30 min. approached baseline values more closely.

In Group III (<65 years, COT), a decrease was observed at 5 min. (−8.0 mmHg, p = 0.036); however, no statistically significant difference remained at 15 min., and rapid stabilisation was achieved. In Group I (<65 years, HFNO), a significant decrease was observed 5 min. after induction (−8.16 mmHg, p = 0.032); however, recovery towards baseline values was observed at 15 and 30 min. (Table 1, Figure 2). When arithmetic means of the haemodynamic profile for SAP were evaluated, Group IV (≥65 years, control) started with the highest systolic blood pressure (139 mmHg). At 5 min., systolic blood pressure values in the HFNO groups (Groups I and II) decreased (approximately 121 mmHg) compared with the COT groups. At 30 min., systolic blood pressure in the elderly HFNO group (Group II) remained around 122 mmHg, whereas it recovered to 132 mmHg in the elderly non-HFNO group (Group IV). The association between age group and hypotension incidence showed a moderate effect size (Cramér's V = 0.32).

In mixed-model and post hoc analyses of SpO_2 values, within-group comparisons showed the following results. No statistically significant difference was observed in Group I at 5 and 15 min. compared with baseline. A significant decrease was observed at 30 min. (p = 0.012). No statistically significant difference from baseline was observed in Group II at any time point. This group was the most stable, maintaining SpO_2 values and even demonstrating a numerical increase at 30 min. (99.15%). No statistically significant decrease or increase was observed in Groups III and IV over

Table 1. Characteristics of the patients according to groups

	Group I n=25	Group II n=25	Group III n=25	Group IV n=25	p
Gender					
Female	15 (60.0)	14 (56.0)	13 (52.0)	12 (48.0)	0.848
Male	10 (40.0)	11 (44.0)	12 (48.0)	13 (52.0)	
Ind.HR	82.12±16.12	78.88±11.92	80.08±16.01	83.36±9.46	0.571
5 min. after HR	129.60±18.50	91.20±13.56	95.76±13.85	89.52±16.74	0.257
15 min. after HR	91.16±15.18	87.28±12.48	95.28±11.54	92.40±12.11	0.198
30.min after HR	94.92±6.92	84.31±12.90	95.40±15.15	93.00±16.04	0.037*
Ind. SAP	129.80±18.02	136.16±20.66	135.48±19.23	139.12±21.11	0.584
5 min. after SAP	121.64±18.64	121.36±16.03	131.16±18.95	130.72±23.55	0.474
15 min. after SAP	126.60±18.72	123.48±19.51	135.76±4.29	34.44±3.50	0.167
30 min. after SAP	127.83±14.36	123.23±16.91	135.50±11.94	133.73±23.41	0.340
Ind. ET CO2	34.76±2.86	34.72±2.48	33.92±2.40	34.40±2.77	0.696
5 min. after ET CO2	35.64±3.49	33.80±3.49	35.76±4.29	34.44±3.50	0.167
15 min. after ET CO2	36.36±3.68	34.32±4.43	36.12±4.60	36.04±4.54	0.249
30 min. after ET CO2	36.72±2.99	35.00±3.46	39.00±5.50	37.20±5.02	0.215
Ind. SpO2	99.12±1.20	97.84±2.76	98.44±1.80	97.56±2.12	0.028*
5 min. after SpO2	98.04±2.11	98.28±1.79	97.32±2.70	97.04±5.57	0.556
15 min. after SpO2	98.32±1.70	98.28±2.41	97.24±3.44	96.36±4.93	0.404
30 min. after SpO2	97.08±5.82	99.15±1.68	98.10±1.45	96.13±2.70	0.003**
Medicines used					
Midozolam (mg)	2.48±0.92	2.00±0.71	2.44±0.82	1.92±0.57	0.102
Fentanyl (µgr)	66.00±25.90	65.21±26.27	76.88±26.53	58.00±21.31	0.120
Propofol (mg)	74.00±17.08	62.08±19.56	81.20±50.11	58.40±20.75	0.008**
Ketafol (cc/h)	14.88±8.24	12.96±4.99	15.04±4.77	11.72±5.75	0.073
Flumazenil	0.33±0.10	0.30±0.10	0.34±0.09	0.28±0.08	0.085
RASS 0 (min.)	8.08±4.85	9.84±3.58	12.48±7.65	10.52±4.62	0.023*

Data are presented as Mean±SD, Frequency (%). P-values computed using Kruskal-Wallis test for continuous variables and Pearson chi-square for categorical variables. ET CO2 : End-tidal Carbon Dioxide; SAP:Systolic Blood Pressure; SpO2: Peripheral Oxygen Saturation; *p < 0.05, ** p < 0.01 were considered statistically significant.

time. The corresponding effect size was moderate ($\eta^2 = 0.11$), indicating clinically meaningful between-group variation.

When groups were compared with each other, no statistically significant differences were found at induction or at 5 min. At 15 min. (Time 3), Group IV had significantly lower SpO_2 values than Groups I ($p = 0.030$) and II ($p = 0.034$). At 30 min. (Time 4), Group IV demonstrated significantly lower values than Group II ($p = 0.029$). A borderline difference was observed between Group II and Group I ($p = 0.055$), with higher values observed in Group II.

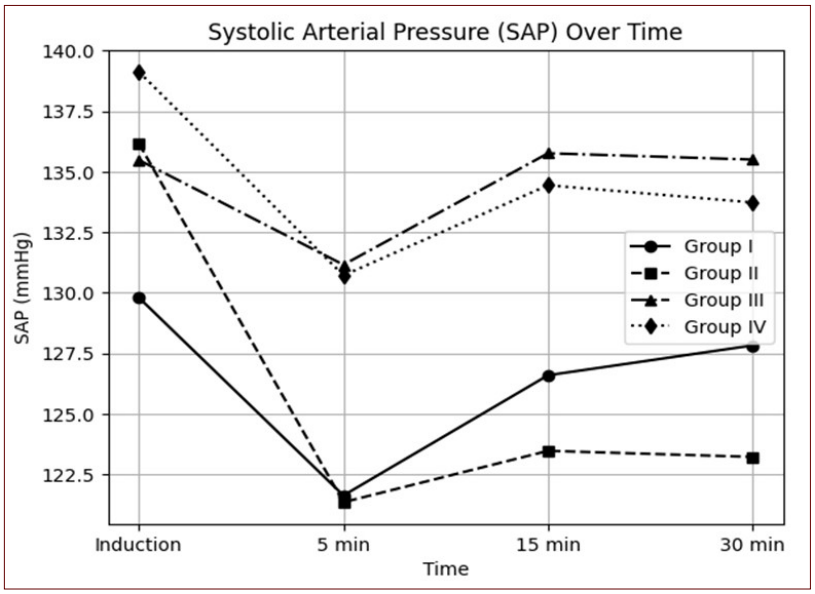


Figure 2. Changes in systolic blood pressure (SAP) over time in the four study groups.

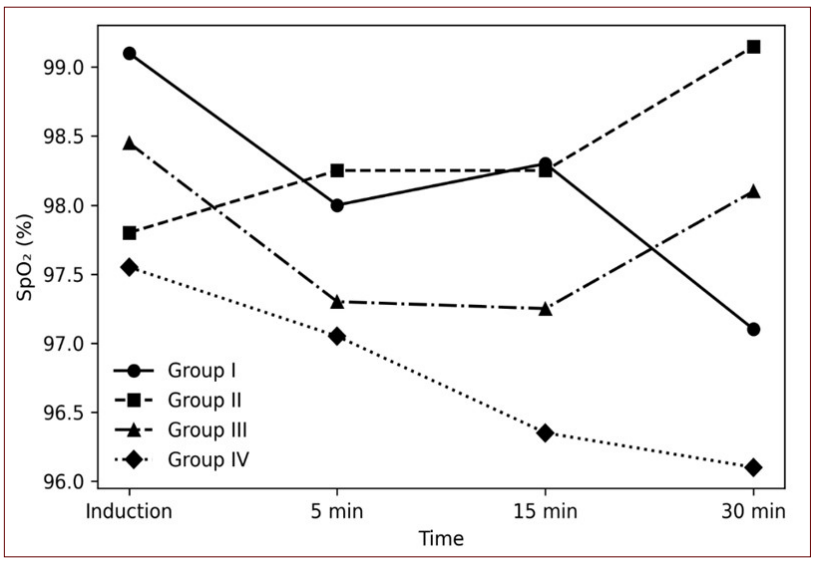


Figure 3. Changes in peripheral oxygen saturation (SpO2) over time in the four study groups.

The association between oxygenation strategy and hypoxaemia incidence demonstrated a moderate effect size (Cramér's V = 0.29) (Figure 3).

Repeated HR measurements were compared using repeated-measures ANOVA with group and time as factors, and post hoc analyses were performed when appropriate. At induction, no statistically significant difference in HR was observed among the four groups ($p > 0.05$). At 5 minutes, a marked increase in HR was observed in Group I (129.6 ± 181.5 bpm). Although intergroup mean values differed at this time point, high variability (wide standard deviation) resulted in no statistically significant intergroup difference ($p > 0.05$). At 15 min., HR values converged across groups, and no statistically significant

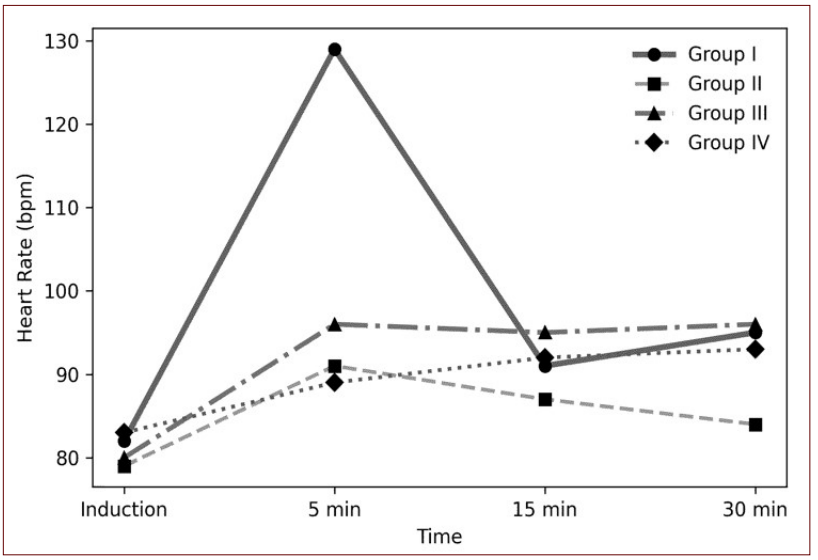


Figure 4. Changes in heart rate over time in the four study groups.

difference was detected ($p > 0.05$). HR values were comparable among groups at induction, 5 min. and 15 min.; however, a statistically significant intergroup difference was observed at 30 minutes ($p = 0.037$) (Figure 4). Post hoc analysis revealed that the significant intergroup difference observed at 30 min. was mainly driven by lower HR values in Group II compared with Groups I and III.

Baseline $EtCO_2$ levels were comparable across all four groups ($p > 0.05$) and remained within physiological limits (30–40 mmHg) throughout the procedure. Similarly, $EtCO_2$ values at 5, 15, and 30 min. showed no significant intergroup variation (all $p > 0.05$). Regarding sedation, baseline midazolam doses were statistically similar among the groups ($F = 3.61$, $p = 0.102$), with post hoc analysis confirming no significant pairwise differences. The lowest propofol dose was administered in Group IV patients, whereas the highest dose was used in Group III patients ($F = 3.04$, $p = 0.0326$), with a statistically significant difference among groups ($p = 0.008$). The effect size for differences in propofol consumption among groups was moderate ($\eta^2 = 0.09$), indicating that approximately 9% of the variance in propofol dose was explained by group allocation.

A similar pattern was observed for ketofol use (Group IV: 11.72 ± 5.75 vs. Group III: 15.04 ± 4.77; $p = 0.073$). The time to achieve a RASS score of 0 after discontinuation of sedative agents and completion of the procedure differed significantly among groups ($p = 0.023$). While this time was 8.08 ± 4.85 min. in Group I (<65 years + HFNO), the longest time was observed in Group III (<65 years + COT), at 12.48 ± 7.65 min. ($F = 2.85$, $p = 0.0416$). For recovery time (RASS 0), the observed effect size was small to moderate ($\eta^2 = 0.08$) (Table 1).

Multivariable logistic regression analyses were performed to determine whether age and oxygenation strategy independently predicted hypoxaemia and hypotension.

No patient experienced hypoxaemia (SpO_2 ≤90%) at induction. At 5 minutes after induction, one patient in Group IV developed hypoxaemia, with a SpO_2 of 73%. At 15 minutes, hypoxaemia occurred in one patient in Group III (SpO_2 84%) and in three patients in Group IV (SpO_2 values of 89%, 87%, and 80%). At 30 minutes after induction, one patient in Group I developed hypoxaemia, with a SpO_2 of 70%. Overall, hypoxaemic episodes occurred more frequently in the conventional oxygen therapy

groups than in the HFNO groups, although a single late hypoxaemic event was observed in the younger HFNO group.

Hypoxaemia (SpO_2 ≤90%): HFNO use was independently protective against hypoxaemia (OR 0.38 [95% CI 0.17–0.82], p = 0.014). After adjustment for oxygenation method, propofol dose and ketofol dose, age ≥ 65 years was independently associated with hypoxaemia (OR 2.41 [95% CI 1.18–4.93], p = 0.016). Propofol dose was not an independent predictor after adjustment (OR 1.01 per mg increase [95% CI 0.99–1.03], p = 0.21). Ketofol dose was not independently associated with hypoxaemia (OR 1.04 [95% CI 0.96–1.12], p = 0.30). Model goodness-of-fit was acceptable (Hosmer–Lemeshow p = 0.48).

Hypotension (SAP <90 mmHg or ≥20% decrease): After multivariable adjustment, age ≥ 65 years remained an independent predictor (OR 2.87 [95% CI 1.34–6.14], p = 0.007). HFNO use was not independently associated with hypotension (OR 1.22 [95% CI 0.58–2.57], p = 0.59). Higher propofol dose was weakly associated with hypotension (OR 1.03 per mg increase [95% CI 1.00–1.05], p = 0.041). Model fit was acceptable (Hosmer–Lemeshow p = 0.61).

DISCUSSION

Few studies have specifically evaluated the efficacy of high-flow nasal oxygen (HFNO) in geriatric patients undergoing ERCP under procedural sedation while simultaneously assessing both respiratory and haemodynamic outcomes (3,7,8). Previous randomized trials and meta-analyses have consistently shown that HFNO reduces hypoxaemic events and improves respiratory safety during gastrointestinal endoscopy (6). However, most studies included heterogeneous populations or focused on obesity, difficult airways, or mixed endoscopic procedures rather than elderly patients undergoing ERCP (9). Consequently, evidence for the optimal oxygenation strategy in this high-risk population remains limited. Age-related physiological changes, including reduced functional residual capacity, impaired ventilatory responses, increased upper-airway collapsibility, and diminished cardiopulmonary reserve, increase susceptibility to sedation-related respiratory complications (9). Our findings demonstrate that HFNO significantly improved oxygenation but did not prevent age-related hypotension, suggesting that oxygenation and haemodynamic management should be considered complementary yet independent therapeutic goals during procedural sedation in older adults.

Respiratory complications remain one of the most frequently encountered issues during sedated ERCP. Factors such as prolonged procedure duration, deep sedation and prone positioning collectively increase the likelihood of hypoventilation and oxygen desaturation. In this context, the enhanced oxygen delivery provided by HFNO appears to mitigate the risk of hypoxemia, especially in patients with limited physiological reserve (3,5).

Consistent with Cha et al. (3), who demonstrated that HFNO use reduced the frequency of desaturation in patients undergoing ERCP under sedation, our findings showed a significantly lower incidence of hypoxaemia in the geriatric HFNO group than in the geriatric COT group. This supports the notion that HFNO effectively enhances oxygenation in this patient population. The beneficial effects of HFNO can be attributed to several physiological mechanisms. High flow rates facilitate clearance of nasopharyngeal dead space, maintain a consistent inspired oxygen concentration and generate a mild positive airway pressure. These features may partially compensate for sedation-induced respiratory depression (7). Despite improvements in oxygenation, hemodynamic instability was still observed, particularly among elderly patients. This finding indicates that advanced age itself is a major determinant of cardiovascular response during sedation (6). Reduced autonomic responsiveness,



diminished vascular tone and increased sensitivity to sedative agents may all contribute to this vulnerability. Therefore, the observed decreases in systolic blood pressure should not be interpreted as a direct adverse effect of HFNO. Recent studies conducted in selected patient populations have shown that HFNO significantly reduces hypoxia, subclinical respiratory depression and severe hypoxic events during sedated GI endoscopy in obese patients without increasing other adverse events (8).

Age-related decrease in lung elasticity, central chemoreceptor response to CO_2 and cardiovascular autonomic responses, along with higher ASA scores and the presence of concomitant diseases, increases the risk of respiratory and hemodynamic complications during sedation (10). However, in our study, $EtCO_2$ values in all groups remained within clinically acceptable physiological ranges (approximately 30–40 mmHg) throughout the study period. This finding suggests that alveolar ventilation was comparable among groups and that the observed haemodynamic differences (e.g. HR changes at 30 min.) were unlikely to be ventilatory in origin. This shows that despite deep sedation, adequate alveolar ventilation was generally provided. This finding is clinically relevant because oxygenation and ventilation are distinct physiological processes, and normal oxygen saturation may not reliably exclude hypoventilation, particularly in patients receiving supplemental oxygen. Continuous capnography enables the early detection of hypoventilation and apnea before clinically significant oxygen desaturation develops and is therefore recommended during procedural sedation by current international guidelines (10,11).

The stable $EtCO_2$ values observed in our study suggest that both oxygenation strategies were associated with satisfactory ventilatory control under continuous respiratory monitoring, while highlighting the importance of capnography for the early recognition of respiratory compromise. In pointwise comparisons, oxygenation in Group IV (geriatric patients receiving COT) was found to be lower during the later stages of the procedure (15 and 30 min.) compared with other groups, particularly Group II. This finding supports the effectiveness of HFNO as an oxygenation strategy during sedation (6).

During sedated ERCP, HFNO reduced the frequency of desaturation. A statistically significant difference in baseline SpO_2 was observed between the study groups. Although the absolute difference was small and all baseline values were within the clinically acceptable range, this imbalance may have introduced a degree of residual confounding. Therefore, the results related to oxygenation outcomes should be interpreted with caution.

Group II appeared to be the most stable group, exhibiting no decline over time and demonstrating higher oxygen saturation values at 30 min. compared with the other groups, particularly Group IV. The physiological mechanisms underlying these favourable outcomes likely include dead space washout, delivery of stable FiO_2 and generation of a low level of PEEP through high flow rates (3). A borderline difference in SpO_2 values was observed between Groups I and II ($p = 0.055$).

While Group I did not show statistically significant differences at 5 and 15 min. compared with baseline, a significant decrease was observed at 30 min. ($p = 0.012$). In the younger patient population, a clear HFNO superiority was not evident. Preserved respiratory reserve and higher tolerance to sedation in younger patients may have limited the additional benefits provided by HFNO. These findings support the concept that the effects of HFNO are more pronounced in patient populations with reduced physiological reserves. The association between oxygenation strategy and hypoxaemia incidence demonstrated a moderate effect size (Cramér's V = 0.29). These findings indicate that the observed differences were not only statistically significant but also clinically meaningful.

Although our findings support the beneficial effects of HFNO in reducing hypoxaemic events during ERCP under sedation, the available evidence remains heterogeneous. Systematic reviews and meta-analyses have shown that HFNO reduces hypoxaemia and the need for airway interventions compared with conventional oxygen therapy; however, substantial between-study heterogeneity has been reported, and the benefits appear less consistent in patients at high risk of hypoxaemia (12). These discrepancies may reflect differences in patient populations, sedation depth, endoscopic procedures, oxygen flow settings, and definitions of hypoxaemia (13). The association between age group and hypotension incidence showed a moderate effect size (Cramér's V = 0.32). SAP decreased at 5 minutes compared with baseline in all groups ($p < 0.05$). The most pronounced decreases were observed in Group II (−14.8 mmHg) and Group IV (−12.76 mmHg). This finding indicates that patients aged 65 years and older are more hemodynamically sensitive to anaesthesia induction. Younger groups (Groups I and III) fully returned to induction values by 15 minutes. In contrast, in the elderly groups, SAP in Group II remained 13.4 mmHg below baseline, and this difference remained statistically significant ($p = 0.005$).

HFNO may influence cardiovascular physiology by generating low levels of positive airway pressure, which can slightly increase intrathoracic pressure and reduce venous return. However, the airway pressure produced by HFNO (approximately 2–5 cmH_2O) is substantially lower than that generated by CPAP or non-invasive ventilation, and its haemodynamic effects are generally minimal (14,15). Consistent with this physiological mechanism, HFNO was not an independent predictor of hypotension in our multivariable analysis, suggesting that the observed haemodynamic changes were more likely related to age and sedative administration than to HFNO itself. These findings are in agreement with previous physiological studies demonstrating that HFNO improves oxygenation without causing clinically relevant cardiovascular compromise. In addition to statistical significance testing, standardized effect sizes were reported to evaluate the magnitude and clinical relevance of the observed findings. The association between HFNO use and hypoxaemia demonstrated a moderate effect size (Cramér's V = 0.29), while the relationship between age and hypotension also showed a moderate effect size (Cramér's V = 0.32). Similarly, the between-group differences in oxygenation were associated with a moderate effect size ($\eta^2 = 0.11$). These findings indicate that, despite the moderate sample size, the observed differences were not only statistically significant but also clinically meaningful.

Another noteworthy finding of our study was the more pronounced decrease and slower recovery of SAP in the geriatric HFNO group. Although HFNO does not have a direct hypotensive effect, reduced vasomotor tone and increased preload dependency in geriatric patients may contribute to deeper and more sustained hypotension following induction, while improved oxygenation may allow for deeper levels of sedation. In addition, relatively higher cumulative doses of sedative agents in elderly patients may have contributed to blunting of hemodynamic responses (15). The data do not support a link between HFNO and hemodynamic instability. Rather, they show that during sedation, geriatric patients have less robust hemodynamic regulation irrespective of the oxygenation technique employed. This highlights the importance of accounting for age-related physiological vulnerability, prioritizing cautious adjustment of sedative dosing and rigorous hemodynamic surveillance instead of avoiding HFNO in this population (16).

The reduced need for early intervention due to hypoxaemia in the HFNO group may have allowed sedation to be maintained more continuously. In geriatric patients with limited cardiovascular reserve, this may have rendered the impact on systolic blood pressure more pronounced.



The relatively larger variability in heart rate observed in Group I is most likely attributable to physiological inter-individual variability rather than a clinically meaningful effect of the oxygen delivery strategy. Heart rate during procedural sedation is influenced by multiple factors, including baseline autonomic nervous system activity, anxiety, nociceptive stimulation during endoscope insertion, and individual pharmacodynamic responses to sedative agents (17).

Importantly, despite the larger standard deviation, mean heart rate values remained within physiological limits, and no increase in cardiovascular complications or haemodynamic instability was observed (18). Therefore, this finding is unlikely to represent a clinically significant treatment effect and should be interpreted with caution, particularly considering the relatively modest sample size.

Regarding sedative use, HFNO has been reported to reduce incidence of hypoxia and severe desaturation during elective gastroscopy performed under propofol sedation in ASA I-II patients, while maintaining good tolerability and a low rate of adverse effects (19). In our cohort, Group IV received the lowest doses of both propofol and ketofol, whereas Group III required the highest doses ($p = 0.008$ and $p = 0.073$, respectively). Notably, geriatric patients were managed with lower sedative doses compared with younger adults. These observations reinforce the associantion between deeper levels of sedation and increased risk of respiratory complications, regardless of age and are in line with prior studies identifying propofol and ketofol administration as contributors to hypoxemia.

Aging is associated with several physiological changes that increase susceptibility to respiratory compromise during procedural sedation, including increased upper airway collapsibility, reduced functional residual capacity, decreased respiratory muscle strength, blunted ventilatory responses to hypoxia and hypercapnia, and impaired airway protective reflexes. Consequently, even modest levels of sedation may result in upper airway obstruction and ventilation–perfusion mismatch in older adults. These age-related alterations likely account for the increased hypoxemia observed in our elderly cohort despite lower sedative requirements and are consistent with previous reports demonstrating greater respiratory vulnerability during sedation in older patients (5,9,20). Similarly, midazolam has been associated with prolonged sedative effects and a greater risk of respiratory compromise, especially in geriatric patients (21).

Consequently, even when HFNO is employed, vigilant monitoring for desaturation and hypoventilation remains crucial in elderly patients, particularly when higher sedative requirements are present (2).

In agreement whit existing literature, advanced age continues to be an independent risk factor for both respiratory and hemodynamic instability during sedation, despite the use of optimised oxygenation techniques such as HFNO.

Earlier achievement of a RASS score of 0 may facilitate earlier neurological assessment, mobilization, and discharge from post-procedural monitoring, potentially improving endoscopy unit efficiency, particularly in elderly patients (22). Given the increased susceptibility of older adults to delayed recovery and postoperative complications, even modest reductions in recovery time may be clinically relevant. Future studies should determine whether these findings translate into shorter PACU stay, earlier discharge, and improved patient-centered outcomes.

Our findings also suggest that HFNO may provide the greatest benefit in selected high-risk geriatric patients, including those with obesity, obstructive sleep apnea, chronic pulmonary disease, frailty, or prolonged ERCP procedures. These conditions are associated with reduced respiratory reserve and an increased risk of peri-procedural hypoxaemia, potentially enhancing the clinical value of HFNO

(23). Further randomized studies are needed to confirm the benefit of a targeted HFNO strategy in these subgroups.

The mechanisms underlying the beneficial effect of HFNO should be interpreted cautiously. Although our study demonstrated a reduction in hypoxaemic events, the underlying physiological mechanisms were not directly evaluated. Previous studies suggest that HFNO improves oxygenation by delivering a stable inspired oxygen concentration, washing out anatomical dead space, and generating low levels of positive airway pressure (24). These mechanisms provide a plausible explanation for our findings but should be regarded as hypothesis-generating rather than directly demonstrated by the present study.

Beyond its physiological benefits, HFNO may also improve procedural efficiency by reducing hypoxaemic events, airway interventions, and procedure interruptions, particularly in high-risk patients (25). Although HFNO requires specialized equipment and higher initial costs than conventional oxygen therapy, these costs may be offset by fewer respiratory complications and unplanned hospital admissions. Formal cost-effectiveness analyses, however, were beyond the scope of this study. In addition, patient comfort and endoscopist satisfaction were not prospectively evaluated. As HFNO is generally well tolerated during procedural sedation, future studies should assess these patient- and operator-reported outcomes together with economic analyses to better define its overall clinical value.

The single-centre design and moderate sample size may limit generalisability. In addition, the use of a standard ketofol protocol across all age groups, the lack of long-term follow-up, the absence of EEG-based sedation monitoring, and the failure to include patient-reported comfort outcomes are among the study's other limitations. Larger multicentre studies are warranted to confirm these findings and evaluate long-term outcomes.

Conclusion

In elderly patients undergoing ERCP under sedation, HFNO reduced the occurrence of hypoxaemic events compared with conventional oxygen therapy, supporting its role as an effective oxygenation strategy in patients at increased respiratory risk. However, HFNO did not prevent sedation-related hypotension, indicating that optimization of oxygenation alone does not address haemodynamic instability associated with sedative administration. These findings emphasize the need for individualized respiratory and haemodynamic management strategies during ERCP sedation, particularly in older patients.

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