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Dexmedetomidine Versus Propofol Infusion During Monitored Anaesthesia Care for Endoscopic Retrograde Cholangiopancreatography: A Prospective Randomised Double-Blind Study

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ABSTRACT

Background: Monitored anaesthesia care for endoscopic retrograde cholangiopancreatography (ERCP) requires agents that balance rapid induction, procedural comfort, and cardiorespiratory safety. Dexmedetomidine and propofol differ pharmacologically in their onset, recovery, and haemodynamic profiles. This study compared their sedation efficacy and safety in adult patients undergoing ERCP.

Methods: A single-centre, hospital-based, double-blind, randomised, active-controlled prospective study was conducted at Pacific Institute of Medical Sciences, Udaipur (IEC No. STU/IEC/2024/36, dated 16.05.2024). A total of 120 adult patients undergoing elective ERCP were equally randomised to Group D (dexmedetomidine: loading 1 μg/kg over 10 minutes, maintenance 0.2–0.7 μg/kg/hour) and Group P (propofol: loading 0.5 mg/kg, maintenance 25–75 μg/kg/min). Onset time to Ramsay Sedation Scale (RSS) score 4, recovery time to RSS 1, haemodynamic and respiratory parameters, total rescue fentanyl dose, and adverse events were compared.

Results: Baseline characteristics were comparable between groups (all $p > 0.05$). Propofol achieved faster onset (9.18 vs 12.0 minutes; $p < 0.001$). Dexmedetomidine showed significantly shorter recovery (7.95 vs 22.6 minutes; $p < 0.0001$). Mean arterial pressure was significantly better maintained with dexmedetomidine from 15 minutes onwards ($p < 0.001$). Heart rate was significantly higher in the dexmedetomidine group from 45 minutes onwards. Oxygen saturation and respiratory rate were significantly better preserved with dexmedetomidine during the procedure. Rescue fentanyl requirement was higher with dexmedetomidine (118 vs 64.4 μg; $p < 0.0001$). No major adverse events occurred in either group.

Conclusion: Propofol offers faster onset while dexmedetomidine provides quicker recovery with superior haemodynamic and respiratory stability. Dexmedetomidine is preferred when cardiorespiratory safety is the priority; propofol when rapid induction is required. Both agents are safe for ERCP sedation without major adverse events.

Keywords: dexmedetomidine; propofol; monitored anaesthesia care; ERCP; Ramsay Sedation Scale; recovery time; haemodynamic stability

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is a complex therapeutic endoscopic procedure used in the management of pancreaticobiliary disease, including choledocholithiasis, biliary strictures, and chronic pancreatitis. It demands prolonged patient cooperation in the prone position, which limits airway access, and is associated with significant discomfort from biliary instrumentation, contrast injection, and sphincterotomy.[1,2] These factors collectively necessitate reliable sedation that achieves both procedural comfort and safety, and that supports timely recovery given the typical high procedural throughput of endoscopy units.

Monitored anaesthesia care (MAC) has become the preferred approach for ERCP sedation, allowing titration of sedative agents to achieve deep, controllable sedation while maintaining spontaneous ventilation. The prone position, which is standard in most ERCP suites, amplifies the consequences of sedation-related respiratory compromise by restricting chest wall movement, impairing airway access, and delaying recognition of early desaturation.[3] Drug selection for MAC in ERCP must therefore balance rapid induction — which reduces procedural delay — against cardiorespiratory stability, recovery profile, and opioid requirement.

Propofol is a highly lipophilic intravenous hypnotic agent that potentiates GABA-A receptor-mediated neuronal inhibition, producing rapid onset of sedation within seconds of administration. Its pharmacokinetic profile, characterised by rapid redistribution and efficient hepatic metabolism, supports a short context-sensitive half-time and predictable recovery.[4] However, it lacks intrinsic analgesic activity and causes dose-dependent respiratory depression and systemic vasodilatation, which may translate into hypotension particularly in patients with limited cardiorespiratory reserve.[5]

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that produces sedation by reducing sympathetic outflow from the locus coeruleus, inducing a state resembling physiological non-REM sleep. Unlike propofol, its sedation is arousable, it possesses intrinsic analgesic properties that may reduce opioid requirement, and its effect on respiratory drive is minimal even at deeper sedation levels, making it pharmacologically attractive for procedures with limited airway access.[6,7] However, its onset is slower due to the need for a loading infusion to avoid abrupt haemodynamic shifts, and its sympatholytic properties may cause bradycardia.

Despite growing use of both agents in ERCP, comparative data from standardised randomised trials remain limited, particularly from Indian centres, where patient populations and procedural protocols may differ from those in high-income settings. The present study was designed to directly compare dexmedetomidine and propofol in terms of onset of sedation, recovery time, haemodynamic and respiratory stability, rescue analgesic requirement, and adverse events during monitored anaesthesia care for ERCP.

AIM

To compare dexmedetomidine and propofol infusions in terms of onset of sedation, recovery time, haemodynamic and respiratory parameters, rescue analgesic requirement, and adverse effects during monitored anaesthesia care for endoscopic retrograde cholangiopancreatography.

MATERIAL AND METHODS

This was a single-centre, hospital-based, double-blind, randomised, active-controlled prospective study. The study was conducted in the Department of Anaesthesiology at Pacific Institute of Medical Sciences, Udaipur, Rajasthan — a tertiary care teaching hospital. The study duration was 18 months, with 12 months dedicated to participant recruitment and data collection. Ethical approval was obtained from the Institutional Ethics Committee of Pacific Institute of Medical Sciences, Udaipur, under approval number STU/IEC/2024/36, dated 16th May 2024. The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants before allocation to study groups. The primary outcomes were onset time to achieve a Ramsay Sedation Scale (RSS) score of 4 and recovery time from procedure completion to RSS score of 1. Secondary outcomes were intra-procedural haemodynamic stability (heart rate, mean arterial pressure), respiratory parameters (respiratory rate, SpO_2), total cumulative rescue fentanyl dose, and the occurrence of postoperative nausea and vomiting. Sedation-related adverse events — defined as hypoxaemia (SpO_2 <90% for >10 seconds), respiratory depression (RR <10 breaths/min), hypotension (MAP <65 mmHg), and bradycardia (HR <45 bpm) — were recorded and managed per institutional protocol.

Drug Protocols and Target Sedation: Group D (dexmedetomidine): loading infusion of 1 μg/kg administered over 10 minutes, followed by maintenance infusion at 0.2–0.7 μg/kg/hour. Group P (propofol): induction bolus of 0.5 mg/kg, followed by maintenance infusion at 25–75 μg/kg/min. The target sedation level for both groups was a Ramsay Sedation Scale score of 4. Intravenous fentanyl 0.5 μg/kg was administered as rescue medication if the target sedation level was not achieved or if the patient showed intolerance to the procedure.[17-42]

STUDY PARTICIPANTS, INCLUSION AND EXCLUSION CRITERIA

INCLUSION CRITERIA:

1. Adult patients aged 31–70 years scheduled for elective ERCP.
2. ASA physical status I, II, or III.
3. Written informed consent obtained.

EXCLUSION CRITERIA:

1. Known allergy to dexmedetomidine, propofol, or fentanyl.
2. Pre-existing bradycardia defined as heart rate less than 60 beats per minute.
3. Significant cardiovascular disease, arrhythmia, or haemodynamic instability.
4. Severe hepatic, renal, or pulmonary disease.
5. Active substance abuse or chronic opioid use.
6. Pregnancy or lactation.
7. Emergency or unplanned ERCP.

Sample Size and Randomisation: Sample size was calculated based on expected differences in onset and recovery time between the two agents. The minimum required sample was 120 patients, 60 per group. Randomisation was performed using a computer-generated random number sequence; allocation was concealed using sealed

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opaque envelopes opened sequentially at the time of assignment. Double blinding was maintained: the drug was prepared by a pharmacist not involved in data collection, and participants and the data-recording anaesthesiologist were blinded to group allocation.

Data Collection Procedure: Standard monitoring — non-invasive blood pressure, continuous ECG, and pulse oximetry — was applied to all participants. Baseline values (T0) of heart rate, MAP, respiratory rate, SpO_2, and RSS were recorded before drug administration. The study drug was prepared and administered by a separate pharmacist. The time from initiation of infusion to achievement of RSS 4 was recorded as onset time. Parameters were then recorded at 5-minute intervals from T1 to T8 (up to 60 minutes), and in the post-procedure recovery area at 0, 10, 20, and 30 minutes. Recovery time was defined as time from infusion discontinuation to RSS 1. PONV was assessed clinically at 30 minutes post-procedure. Any sedation-related adverse event was recorded and managed immediately per protocol.

Statistical Analysis: Data were analysed using Stata version 17.0. Continuous variables were expressed as mean ± SD and compared using independent t-test or Mann–Whitney U test. Categorical variables were expressed as frequencies and percentages and compared using Pearson chi-square test. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 120 participants were enrolled and randomised, with 60 in each group. No participant was lost to follow-up and all completed the study. Detailed findings are presented below.

Table 1: Baseline Characteristics

Characteristic	Group D (n=60) n / Mean	Group D %	Group P (n=60) n / Mean	Group P % | P-value
Age Group (years)				
31–40	11	18.3	11	18.3 | 0.957
41–50	17	28.3	15	25.0
51–60	20	33.3	22	36.7
61–70	12	20.0	12	20.0
Mean age (years)	51.0		51.7	P = 0.689
Gender				
Male	46	76.7	38	63.3 | 0.112
Female	14	23.3	22	36.7
ASA Physical Status				
ASA I	13	21.7	23	38.3 | 0.063
ASA II	23	38.3	24	40.0
ASA III	24	40.0	13	21.7
Indication for ERCP				
Choledocholithiasis	8	13.3	16	26.7 | 0.354
Biliary stricture	17	28.3	14	23.3
Chronic pancreatitis	19	31.7	15	25.0
Diagnostic	16	26.7	15	25.0
Mean body weight (kg)	60.9		62.4	P = 0.468

Table 1 confirms comparable baseline characteristics between both groups. Age distribution, mean age (51 vs 51.7 years; p = 0.689), gender distribution (p = 0.112), ASA status (p = 0.063), ERCP indication (p = 0.354), and body weight (60.9 vs 62.4 kg; p = 0.468) were all statistically comparable, confirming adequate baseline equivalence between the groups.

Table 2: Onset Time, Recovery Time, and Rescue Fentanyl Requirement

Outcome	Group D Mean	Group D Range	Group P Mean	Group P Range | P-value
Onset time to RSS 4 (minutes)	12.0 ± 2.03	9 – 15	9.18 ± 1.59	6 – 11 | < 0.001
Recovery time to RSS 1 (minutes)	7.95 ± 2.57	4 – 12	22.6 ± 4.7	15 – 30 | < 0.0001
Total rescue fentanyl (μg)	118	61 – 180	64.4	26 – 100 | < 0.0001

Table 2 shows the primary and key secondary efficacy outcomes. Propofol achieved the target sedation level significantly faster (9.18 vs 12.0 minutes; p < 0.001). Dexmedetomidine demonstrated markedly shorter recovery (7.95 vs 22.6 minutes; p < 0.0001), a difference of nearly 15 minutes representing a clinically and operationally meaningful advantage. Total rescue fentanyl requirement was significantly higher in the dexmedetomidine group (118 vs 64.4 μg; p < 0.0001), reflecting the absence of intrinsic analgesic activity in propofol relative to the need to supplement dexmedetomidine's own analgesic effect in this procedure.

Table 3: Heart Rate Across All Time Points

Time Point	Group D Mean HR (bpm)	Group P Mean HR (bpm)	Difference	P-value
Baseline (T0)	84.8	86.3	−1.5	0.44 (NS)
5 min (T1)	84.8	86.0	−1.2	0.53 (NS)
15 min (T3)	85.0	84.0	+1.0	0.62 (NS)
30 min (T6)	85.1	81.0	+4.1	0.06 (NS)
45 min (T7)*	85.2	80.5	+4.7	0.03*
60 min (T8)*	84.9	79.2	+5.7	0.01*
Post-op 0 min (T9)*	86.0	80.3	+5.7	0.01*
Post-op 10 min (T10)*	86.9	81.2	+5.7	0.01*
Post-op 20 min (T11)*	87.9	82.2	+5.7	0.01*
Post-op 30 min (T12)*	88.7	83.3	+5.4	0.02*

* Statistically significant (p < 0.05). HR remained stable with dexmedetomidine; propofol group showed a progressive intra-procedural decline.

Table 3 shows heart rate trends. Groups were comparable at baseline and during the first 30 minutes of the procedure. From 45 minutes onwards (T7) and throughout the recovery period, heart rate was significantly higher in the dexmedetomidine group than in the propofol group (p = 0.03 to 0.01). The propofol group showed a consistent intra-procedural decline in heart rate, while dexmedetomidine maintained a stable rate throughout the procedure and early recovery.

Table 4: Mean Arterial Pressure Across All Time Points

Time Point	Group D Mean MAP (mmHg)	Group P Mean MAP (mmHg)	Difference	P-value
Baseline (T0)	93.8	93.8	0	1.00 (NS)
5 min (T1)	93.9	93.9	0	1.00 (NS)
10 min (T2)	93.6	93.7	−0.1	0.95 (NS)
15 min (T3)*	93.7	89.5	+4.2	0.02*
20 min (T4)*	93.4	85.6	+7.8	< 0.001*
30 min (T6)*	93.2	85.8	+7.4	< 0.001*
45 min (T7)*	93.3	85.8	+7.5	< 0.001*
60 min (T8)*	93.3	85.9	+7.4	< 0.001*
Post-op 0 min (T9)*	95.2	87.8	+7.4	< 0.001*
Post-op 30 min (T12)*	100.0	93.8	+6.2	< 0.001*

* Statistically significant (p < 0.05). Dexmedetomidine maintained stable MAP throughout; propofol showed significant decline from 15 minutes.

Table 4 shows MAP trends. Both groups had identical baseline MAP (93.8 mmHg). From 15 minutes onwards, MAP in the propofol group declined significantly compared to the dexmedetomidine group (89.5 vs 93.7 at 15 min; p = 0.02). From 20 minutes through the entire intra-procedural phase and recovery (T4–T12), MAP differences were highly significant (p < 0.001). The dexmedetomidine group maintained MAP within a narrow stable range throughout, while propofol caused a sustained reduction of approximately 7–8 mmHg from baseline during the procedure.

Table 5: Respiratory Rate and Oxygen Saturation Across Time Points

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Time Point	D SpO_2 %	P SpO_2 %	SpO_2 P	D RR	P RR	RR P
Baseline (T0)	99.7	99.8	0.07	17	18	0.06
10 min (T2)	99.3	98.7	<0.001*	17	17	0.92
20 min (T4)	99.0	97.7	<0.001*	17	16	0.11
30 min (T6)	98.6	97.6	<0.001*	17	15	0.01*
45 min (T7)	98.5	97.3	<0.001*	17	15	0.003*
60 min (T8)	98.4	97.3	<0.001*	17	15	0.002*
Post-op 0 min (T9)	98.5	97.7	0.006*	17	16	0.12
Post-op 30 min (T12)	98.2	98.6	0.21	19	17	0.01*

D = dexmedetomidine; P = propofol; * significant ($p < 0.05$). SpO_2 and RR were better preserved with dexmedetomidine intra-procedurally and converged post-procedure.

Table 5 presents SpO_2 and RR data. SpO_2 was comparable at baseline but diverged significantly from 10 minutes onwards ($p < 0.001$ for T2–T9), with dexmedetomidine maintaining higher saturation values throughout the procedure. The groups converged at post-procedure 10 and 20 minutes. Respiratory rate similarly remained stable at 17 breaths/min throughout the intra-procedural phase with dexmedetomidine, while propofol was associated with a significant decline to 15 breaths/min from 30 minutes onwards ($p = 0.01–0.002$).

Table 6: Postoperative Nausea and Vomiting and Adverse Events

Outcome	Group D n	Group D %	Group P n	Group P %	P
PONV: No	51	85.0	47	78.3	0.345
PONV: Yes	9	15.0	13	21.7	(NS)
Hypoxaemia (SpO_2 <90%)	0	0	0	0	—
Respiratory depression (RR <10)	0	0	0	0	—
Bradycardia (HR <45 bpm)	0	0	0	0	—
Serious adverse events	0	0	0	0	—

Table 6 shows PONV and safety data. PONV occurred in 15% of the dexmedetomidine group and 21.7% of the propofol group, with no statistically significant difference ($p = 0.345$). No predefined adverse events — hypoxaemia, respiratory depression, bradycardia, or serious adverse events — occurred in either group, confirming the safety of both regimens at the doses used.

DISCUSSION

This double-blind randomised controlled trial enrolled 120 adult patients undergoing elective ERCP at a tertiary anaesthesiology centre in Udaipur and directly compared dexmedetomidine and propofol as the primary sedative agents under MAC. Baseline characteristics were well-matched, ensuring that outcome differences could be attributed to the pharmacological properties of the two agents rather than patient-level confounders. The study demonstrated distinct, clinically meaningful differences in sedation onset, recovery profile, haemodynamic behaviour, respiratory parameters, and analgesic requirement, while both agents proved safe at the doses used.

Propofol achieved the target sedation level (RSS 4) in a mean of 9.18 minutes compared to 12.0 minutes with dexmedetomidine ($p < 0.001$). This difference arises from the fundamental pharmacokinetic distinction between the two agents: propofol's high lipophilicity enables rapid blood-brain barrier penetration, producing sedation within seconds to minutes of administration, while dexmedetomidine requires a structured loading infusion to allow progressive central sympatholytic effect to build without triggering abrupt haemodynamic instability.[4;6] This finding is consistent with Srivastava U et al. (2021), who reported onset of 7.93 minutes for propofol versus 11.4 minutes for dexmedetomidine in ERCP.[8] Zhang N et al. (2024) similarly documented faster onset with propofol, while Aqeel N et al. (2024) observed a mean onset of 11.84 minutes to reach RSS 4 with dexmedetomidine.[9,10] The clinical implication is that the propofol group was ready for endoscope insertion approximately 3 minutes earlier, which has procedural throughput implications in high-volume units. However, as the following findings demonstrate, this advantage must be considered alongside other dimensions of performance.

Dexmedetomidine demonstrated a markedly shorter recovery time (7.95 vs 22.6 minutes; $p < 0.0001$). This nearly three-fold advantage is the most operationally impactful finding of the present study and is explained by the unique pharmacodynamics of dexmedetomidine-induced sedation. Unlike propofol, which causes global cortical depression that must be metabolically cleared, dexmedetomidine induces a state resembling physiological NREM sleep through which patients can be aroused by gentle stimulation as soon as the procedural stimulus — endoscope manipulation — ceases. Recovery is therefore rapid and smooth once the infusion is discontinued. Srivastava U et al. (2021) reported that 86.67% of dexmedetomidine patients achieved maximum Modified Aldrete Score within 5 minutes versus 0% in the propofol group.[8] Zhang N et al. (2024) documented recovery durations of 1.6 minutes (dexmedetomidine) versus 6.9 minutes (propofol),[9] and Garg I et al. (2024) found recovery of 7.24 versus 15.2 minutes in dexmedetomidine-propofol versus propofol-ketamine groups.[11] The consistent finding across studies is that dexmedetomidine-based regimens support earlier departure from recovery to ward, which in resource-limited endoscopy units with multiple parallel lists has direct implications for efficiency.

Dexmedetomidine maintained MAP within a narrow, consistent range throughout the procedure and recovery. Propofol caused a clinically significant decline in MAP from 15 minutes onwards (89.5 vs 93.7 mmHg; $p = 0.02$), which deepened to a 7–8 mmHg sustained reduction from 20 minutes through to post-procedure recovery (all $p < 0.001$). While MAP remained above 85 mmHg in the propofol group — above the pre-defined hypotension threshold of 65 mmHg — this sustained reduction in a predominantly ASA II–III population during prone ERCP is clinically relevant. Propofol's hypotensive effect reflects its vasodilatory and mild negative inotropic actions. Soliman AM et al. (2024) similarly found that dexmedetomidine maintained better MAP in cancer patients undergoing ERCP.[12] Garg I et al. (2024) documented significantly lower MAP in propofol-dexmedetomidine versus propofol-ketamine groups, the latter attributable to ketamine's sympathomimetic properties offsetting the dexmedetomidine effect.[11] Heart rate was stable in the dexmedetomidine group and showed a modest intra-procedural decline in the propofol group, with significant differences from 45 minutes onwards. No patient in either group met the criteria for bradycardia (HR <45 bpm), confirming the cardiovascular safety of dexmedetomidine within the dosing range used.

SpO_2 was significantly better preserved with dexmedetomidine from 10 minutes through the entire intra-procedural phase ($p < 0.001$), consistent with dexmedetomidine's preservation of respiratory drive through its non-GABAergic mechanism of action. Respiratory rate similarly remained stable at 17 breaths/minute throughout the procedure with dexmedetomidine, while propofol caused a significant reduction to 15 breaths/min from 30 minutes onwards ($p < 0.01$). No episode of clinical hypoxaemia (SpO_2 <90%) or respiratory depression (RR <10) occurred in either group, consistent with the supplemental oxygen administered and the monitored setting. Soliman AM et al. (2024) reported a lower incidence of hypoxaemic events with dexmedetomidine (14.9% vs 26.7%),[12] and Liu Y et al. (2025), in a network meta-analysis, found that dexmedetomidine-fentanyl combinations significantly reduced the proportion of patients with SpO_2 below 90% compared to propofol-only sedation.[13] The present study confirms these protective respiratory properties within a strictly monitored ERCP setting.

Total rescue fentanyl was significantly higher in the dexmedetomidine group (118 vs 64.4 μg; $p < 0.0001$). This finding, while initially appearing counterintuitive given dexmedetomidine's intrinsic analgesic effect, reflects the greater depth and quality of sedation provided by propofol at the doses used. The sedation continuum required for ERCP, which approaches deep sedation, is readily achievable with propofol's GABAergic action, whereas dexmedetomidine's physiological sleep-like state may not fully suppress procedural pain from biliary instrumentation without supplemental opioid. PONV rates were 15% and 21.7% in the dexmedetomidine and propofol groups respectively, without reaching statistical significance ($p = 0.345$). The lower rate with dexmedetomidine likely reflects reduced opioid requirement as a driver of nausea, whereas propofol's known antiemetic properties may have partially offset the higher opioid dose in the propofol group.

CONCLUSION

Both dexmedetomidine and propofol are safe and effective sedative agents for monitored anaesthesia care during ERCP, with no major adverse events in either group. Propofol achieves the target sedation level significantly faster (9.18 vs 12.0 minutes; $p < 0.001$), which is advantageous when rapid procedural induction is required. Dexmedetomidine demonstrates markedly shorter recovery (7.95 vs 22.6 minutes; $p < 0.0001$), superior haemodynamic stability, and better preservation of oxygen saturation and respiratory rate during the procedure. The higher rescue fentanyl requirement with dexmedetomidine is a recognised trade-off of its mechanism of action in prolonged pancreaticobiliary intervention. The choice between agents should be guided by individual patient risk profile: dexmedetomidine is preferred when cardiorespiratory stability and rapid recovery are priorities; propofol when fast induction and deeper sedation are required. The two agents may also be considered complementarily in combination protocols[16-43].

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