

Dexmedetomidine versus Propofol Infusion for Intraoperative Haemodynamic Stability during Laparoscopic Surgery: A Prospective Open-Label Comparative Study

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Abstract

Background: Pneumoperitoneum created during laparoscopic surgery induces significant haemodynamic perturbations, including increased systemic vascular resistance, reduced venous return, and activation of neurohumoral stress pathways. Effective intraoperative haemodynamic management is therefore critical. This study aimed to compare the efficacy of dexmedetomidine infusion versus propofol infusion in maintaining haemodynamic stability during laparoscopic surgery and to evaluate postoperative recovery profiles.

Methods: A prospective, open-label comparative study enrolled 70 patients (ASA PS I and II, aged 18–65 years) undergoing elective laparoscopic surgery at Government Villupuram Medical College & Hospital. Patients were randomised equally into Group D (dexmedetomidine: loading dose 1 mcg/kg over 10 minutes before intubation, followed by 0.2 mcg/kg/h infusion) and Group P (propofol: 100 mcg/kg/min infusion after intubation). Both infusions were continued until deflation of pneumoperitoneum. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at multiple time points. Postoperative sedation and recovery were assessed using the Ramsay Sedation Scale (RSS) and Modified Aldrete Score (MAS).

Results: Both groups were comparable at baseline. Group D exhibited significantly lower HR, SBP, DBP, and MAP compared with Group P at most intraoperative time points ($p < 0.01$), reflecting superior haemodynamic attenuation (HR: 3% decrease vs. 18% increase; MAP: 4% decrease vs. 7% increase over pneumoperitoneum). Group D patients had significantly deeper sedation (higher RSS scores) up to 90 minutes postoperatively ($p < 0.01$), while Modified Aldrete Scores were significantly lower in Group D at 0, 15, and 30 minutes post-extubation ($p < 0.01$), indicating slower initial recovery. Both groups achieved full recovery by 45–60 minutes. No adverse events were recorded.

Conclusion: Dexmedetomidine infusion provides superior intraoperative haemodynamic stability during laparoscopic surgery compared with propofol, with effective attenuation of the stress response to pneumoperitoneum. Propofol offers faster early recovery. Dexmedetomidine is the preferred agent when cardiovascular stability is the clinical priority.

Keywords: Dexmedetomidine; Propofol; Laparoscopic surgery; Pneumoperitoneum; Haemodynamic stability; Intraoperative management; Ramsay Sedation Scale; Modified Aldrete Score.

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Introduction

Laparoscopic surgery has revolutionised modern operative practice, offering patients the benefits of reduced postoperative pain, shorter hospital stays, improved cosmesis, and lower rates of surgical site infection compared with open techniques. [1] Nevertheless, the unique physiological challenges

of laparoscopy—particularly the cardiovascular consequences of carbon dioxide (CO₂) pneumoperitoneum—present the anaesthesiologist with a demanding management problem requiring precise titration of anaesthetic agents. The creation of pneumoperitoneum at intra-abdominal pressures

of 12–15 mmHg produces a well-characterised haemodynamic response: compression of the inferior vena cava reduces venous return, while increased systemic vascular resistance (SVR) and activation of the renin-angiotensin-aldosterone axis raise arterial blood pressure and impose increased afterload on the left ventricle. [2,3] Concomitant neuroendocrine activation results in elevated catecholamine levels, further driving tachycardia and hypertension. Patient positioning, especially reverse Trendelenburg, exacerbates the reduction in cardiac preload. [4] In patients with underlying cardiovascular disease, these perturbations can precipitate serious perioperative complications.

Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist (alpha-2/alpha-1 selectivity ratio approximately 1600:1), exerts sedative, analgesic, and sympatholytic effects by inhibiting central noradrenaline release from the locus coeruleus. [5] By attenuating central sympathetic outflow, it mitigates the haemodynamic response to surgical stress and intubation without causing clinically significant respiratory depression. [6] Propofol, a widely used intravenous anaesthetic acting via GABA-A receptors, offers rapid onset and offset, smooth induction, antiemetic properties, and dose-dependent reductions in blood pressure and SVR. Its ability to suppress the sympathetic stress response has made it a popular choice for maintaining anaesthesia during laparoscopic procedures. [7]

While both agents have demonstrated utility in laparoscopic anaesthesia, direct comparative data on their relative efficacy specifically in maintaining intraoperative haemodynamic stability during pneumoperitoneum, and on the quality of postoperative recovery, remain limited. [8] The present study was therefore designed to fill this gap, comparing continuous infusions of dexmedetomidine and propofol as adjuncts to standard general anaesthesia in patients undergoing elective laparoscopic procedures.

The primary objective was to compare the two infusion regimens in terms of HR, SBP, DBP, and MAP at baseline, after the loading dose, at intubation, at initiation of pneumoperitoneum, at 15-minute intervals during pneumoperitoneum, at extubation, and in the immediate postoperative period. The secondary objective was to compare postoperative sedation and recovery profiles using the Ramsay Sedation Scale and Modified Aldrete Score, and to document any adverse events.

Materials and Methods

This was a prospective, open-label comparative study conducted in the Department of Anaesthesiology, in a tertiary care teaching hospital in South India over 12 months. Institutional Ethics

Committee approval was obtained, and written informed consent was secured from all participants prior to enrolment. Patients were assured that participation was voluntary and that withdrawal at any stage would not affect their medical care.

Assuming a type I error (alpha) of 5% and type II error (beta) of 20%, a minimum of 33 patients per group was required to detect a clinically meaningful 5% difference in MAP between the two groups. To improve statistical power, 35 patients were enrolled in each group (total n=70). Computer-generated randomisation tables were used to allocate participants to Group D (dexmedetomidine) or Group P (propofol) in equal proportions.

Patients aged 18–65 years with ASA physical status I or II, scheduled for elective laparoscopic surgery, were included. Exclusion criteria were: ASA PS III or IV; known hepatic, renal, or significant cardiac disease; pre-existing heart block; use of beta-blockers for hypertension; and known allergy to either study drug or study refusal.

Group D received a loading dose of dexmedetomidine 1 mcg/kg over 10 minutes before intubation (diluted to 10 mcg/ml in a syringe infusion), followed by a maintenance infusion of 0.2 mcg/kg/h from the time of intubation until deflation of pneumoperitoneum. Group P received a propofol infusion at 100 mcg/kg/min (diluted to 10 mg/ml) commencing after intubation and continued until deflation of pneumoperitoneum. Both infusions were delivered via a dedicated intravenous line.

All patients underwent standard preoperative evaluation the day before surgery. Intravenous access was secured with two cannula on arrival in the operating theatre. Standard monitoring (ECG, non-invasive blood pressure, SpO₂, EtCO₂) was established and baseline values recorded. Premedication with glycopyrrolate 4 mcg/kg, fentanyl 1.5 mcg/kg, and midazolam 0.03 mg/kg was administered intravenously to all patients. After the Group D loading dose, anaesthesia was induced in both groups with sodium thiopental 5 mg/kg and succinylcholine 2 mg/kg. Intubation was performed with an appropriately sized cuffed endotracheal tube. Anaesthesia was maintained with sevoflurane 0.8% in an O₂:N₂O (40:60) mixture, guided by entropy values of 40–60. Muscle relaxation was maintained with atracurium 0.1 mg/kg. At the end of surgery, neuromuscular block was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 8 mcg/kg. Bradycardia (HR <60 bpm) was treated with atropine 0.01–0.02 mg/kg intravenously, and hypotension (MAP <30% of baseline) with mephenteramine 6 mg intravenously.

Haemodynamic parameters (HR, SBP, DBP, MAP) were recorded at: baseline (awake); 5 minutes after loading dose (Group D) or after induction (Group P); 10 minutes post-induction; induction; intubation; initiation of pneumoperitoneum; every 15 minutes during pneumoperitoneum; at termination of pneumoperitoneum; at extubation; and postoperatively at 30-minute intervals for 2 hours. The Ramsay Sedation Scale (RSS, scores 1–6) and Modified Aldrete Score (MAS, maximum 10) were recorded at 0, 15, 30, 45, 60, 75, 90, 105, and 120 minutes post-extubation. All analyses were performed using SPSS version 26.0. Continuous data are expressed as mean \pm standard deviation. Between-group comparisons of continuous variables were performed using the independent samples Student's t-test. Categorical variables were compared using the chi-squared test. A two-tailed

p-value of <0.05 was considered statistically significant.

Results: Seventy patients completed the study (35 per group). The two groups were well matched at baseline in terms of age, sex, and ASA physical status, with no statistically significant differences in any parameter (Table 1). The mean age was 36.00 ± 13.55 years in Group D and 40.14 ± 12.97 years in Group P ($p=0.196$).

Males predominated in both groups (Group D: 62.9%; Group P: 54.3%; $p=0.467$). ASA class distribution was equal across groups ($p=0.473$). Baseline haemodynamic parameters—HR, SBP, DBP, and MAP—were comparable between the two groups (all $p>0.05$), confirming suitability for direct comparison (Table 1).

Table 1: Demographic and baseline haemodynamic characteristics of the two study groups. NS = not significant; ASA = American Society of Anesthesiologists Physical Status.

Parameter	Group D (Dexmedetomidine) n=35	Group P (Propofol) n=35	p-value
Age (years), Mean \pm SD	36.00 \pm 13.55	40.14 \pm 12.97	0.196 (NS)
Male, n (%)	22 (62.9%)	19 (54.3%)	0.467 (NS)
Female, n (%)	13 (37.1%)	16 (45.7%)	—
ASA Class I, n (%)	19 (54.3%)	16 (45.7%)	0.473 (NS)
ASA Class II, n (%)	16 (45.7%)	19 (54.3%)	—
Baseline HR (bpm)	77.20 \pm 26.31	79.43 \pm 24.33	0.71 (NS)
Baseline SBP (mmHg)	128.26 \pm 4.70	127.26 \pm 4.89	0.386 (NS)
Baseline DBP (mmHg)	82.71 \pm 1.89	82.66 \pm 1.51	0.89 (NS)
Baseline MAP (mmHg)	97.90 \pm 2.24	97.52 \pm 2.11	0.48 (NS)

Heart Rate

Baseline heart rates were comparable between groups (Group D: 77.20 ± 26.31 bpm; Group P: 79.43 ± 24.33 bpm; $p=0.71$).

Following the initiation of study drug infusion, HR in Group D decreased progressively and remained consistently lower than in Group P throughout the observation period. Statistically significant inter-group differences in HR were observed at 10

minutes ($p=0.02$), 15 minutes into pneumoperitoneum ($p=0.03$), 45 minutes ($p=0.02$), 60 minutes ($p=0.01$), and at extubation ($p<0.01$). Overall, dexmedetomidine produced a 3% decrease in HR from baseline during pneumoperitoneum, whereas propofol was associated with an 18% increase (Table 2). Postoperatively, HR values were comparable between groups ($p=0.68$), indicating resolution of the intraoperative divergence.

Table 2: Comparison of heart rate (bpm) between Group D (dexmedetomidine) and Group P (propofol) at each time point. S* = significant ($p<0.05$); S = highly significant ($p<0.01$); NS = not significant.**

Time Point	Group D Mean \pm SD (bpm)	Group P Mean \pm SD (bpm)	Mean Diff.	t-value	p-value
Baseline	77.20 \pm 26.31	79.43 \pm 24.33	-2.23	-0.37	0.71 NS
After loading dose	80.80 \pm 11.73	84.74 \pm 11.51	-3.94	-1.42	0.16 NS
10 min	78.31 \pm 10.38	84.00 \pm 9.85	-5.69	-2.35	0.02 S*
Pneumoperitoneum	80.09 \pm 12.10	77.66 \pm 12.12	+2.43	0.84	0.40 NS
15 min	79.69 \pm 12.29	88.40 \pm 11.65	-8.71	-3.04	0.03 S*
45 min	80.97 \pm 11.96	88.00 \pm 11.85	-7.03	-2.47	0.01 S*
60 min	76.31 \pm 12.48	83.91 \pm 12.65	-7.60	-2.53	0.01 S*
Extubation	78.66 \pm 10.84	85.77 \pm 10.54	-7.11	-2.78	<0.01 S**
Postoperative	77.66 \pm 10.55	76.54 \pm 11.64	+1.12	0.42	0.68 NS

Systolic and Diastolic Blood Pressure: Both SBP and DBP were similar at baseline in the two groups (SBP: $p=0.386$; DBP: $p=0.89$). From 5 minutes post-loading dose onwards, SBP and DBP were significantly lower in Group D compared with Group P at all time points ($p<0.01$ for all comparisons), with the exception of baseline. Dexmedetomidine was associated with a 5% reduction in SBP from baseline during

pneumoperitoneum, while propofol was associated with a 12% increase. For DBP, Group D showed only a 2% increase from baseline during pneumoperitoneum compared with a 16% increase in Group P. These differences were maintained through intubation, all intraoperative intervals, extubation, and the immediate postoperative period, underscoring the sustained sympatholytic benefit of dexmedetomidine (Table 3).

Table 3: Comparison of systolic blood pressure (SBP) and diastolic blood pressure (DBP) between Group D and Group P at key time points. All comparisons from 5 min onwards were statistically significant ($p<0.01$).

Time Point	Group D SBP±SD (mmHg)	Group P SBP±SD (mmHg)	Group D DBP±SD (mmHg)	Group P DBP±SD (mmHg)	p-value (both)
Baseline	128.26±4.70	127.26±4.89	82.71±1.89	82.66±1.51	NS
5 min (post-loading)	125.20±3.12	129.89±3.15	76.00±2.29	82.09±2.23	<0.01
Intubation	114.37±5.07	121.46±6.00	76.37±2.38	81.60±2.35	<0.01
Pneumoperitoneum	124.26±3.28	129.54±3.31	76.26±2.28	81.94±2.35	<0.01
15 min	125.00±3.26	130.29±3.51	76.63±2.49	81.49±2.49	<0.01
30 min	124.83±2.99	130.37±2.98	76.46±2.01	82.23±1.88	<0.01
60 min	125.51±3.17	130.14±3.23	76.00±2.20	81.26±2.32	<0.01
Extubation	125.00±3.32	129.29±2.99	76.34±2.26	81.77±2.13	<0.01
Postoperative	125.40±3.62	130.46±3.26	76.37±2.25	80.89±2.47	<0.01

Mean Arterial Pressure:

Baseline MAP was equivalent between groups (Group D: 97.90±2.24 mmHg; Group P: 97.52±2.11 mmHg; $p=0.48$). From 5 minutes onwards, MAP in Group D was significantly lower than in Group P at all time points ($p<0.01$ for all). At the time of pneumoperitoneum, Group D demonstrated a mean MAP of 92.26±1.71 mmHg compared with 97.81±1.75 mmHg in Group P

(mean difference -5.55 mmHg, $t=-13.4$, $p<0.01$). This approximately 4% reduction from baseline in Group D contrasted sharply with the 7% increase in Group P, reflecting the effective sympatholytic action of dexmedetomidine in blunting the pneumoperitoneum stress response.

Significant differences persisted through extubation ($p<0.01$) and resolved in the postoperative period at 2 hours (Table 4).

Table 4: Comparison of mean arterial pressure (MAP, mmHg) between Group D and Group P at each time point. S = highly significant ($p<0.01$); NS = not significant.**

Time Point	Group D MAP Mean±SD (mmHg)	Group P MAP Mean±SD (mmHg)	p-value
Baseline	97.90±2.24	97.52±2.11	0.48 NS
5 min	92.40±1.92	98.02±1.79	<0.01 S**
Intubation	89.04±2.25	94.89±2.72	<0.01 S**
Pneumoperitoneum	92.26±1.71	97.81±1.75	<0.01 S**
15 min	92.75±2.09	97.75±2.02	<0.01 S**
30 min	92.58±1.70	98.28±1.73	<0.01 S**
60 min	92.50±1.77	97.55±2.00	<0.01 S**
Extubation	92.56±1.97	97.61±1.70	<0.01 S**
Postoperative	92.71±2.10	97.41±2.12	<0.01 S**

Postoperative Sedation and Recovery

Group D patients exhibited significantly higher RSS scores (indicating deeper sedation) than Group P at all postoperative time points from 0 to 90 minutes ($p<0.05$ to $p<0.01$), with Group D RSS remaining significantly elevated through the 90-minute assessment.

Beyond 90 minutes, the inter-group difference in RSS was no longer statistically significant ($p=0.07$ at 105 min; $p<0.01$ at 120 min). Consistent with deeper sedation, Group D patients had significantly lower MAS scores at 0, 15, and 30 minutes post-extubation ($p<0.01$), reflecting slower initial recovery. However, by 45 minutes, MAS scores were equivalent (9.51±0.66 in both groups,

p=1.00), and both groups achieved perfect MAS scores of 10.00 by 75 minutes, indicating equivalent full recovery. No clinically significant

complications including bradycardia, hypotension, respiratory depression, or postoperative nausea and vomiting were recorded in either group (Table 5).

Table 5: Ramsay Sedation Scale (RSS) and Modified Aldrete Score (MAS) by time point postoperatively.
S* = p<0.05; S** = p<0.01; NS = not significant.

Time Point (min)	Group D RSS Mean±SD	Group P RSS Mean±SD	p-value (RSS)	Group D MAS Mean±SD	Group P MAS Mean±SD
0	2.26±0.78	1.60±0.81	<0.01 S**	6.34±1.49	7.51±1.50
15	2.20±0.72	1.46±0.74	<0.01 S**	7.74±1.24	8.83±0.92
30	2.11±0.72	1.74±0.82	0.05 S*	8.69±1.11	9.26±0.70
45	2.23±0.77	1.74±0.85	0.01 S*	9.51±0.66	9.51±0.66
60	1.97±0.79	1.54±0.74	0.02 S*	9.83±0.38	9.80±0.41
90	2.29±0.83	1.60±0.88	<0.01 S**	10.00±0.00	10.00±0.00
120	2.23±0.77	1.66±0.84	<0.01 S**	10.00±0.00	10.00±0.00

Discussion

The haemodynamic challenge posed by CO₂ pneumoperitoneum is well characterised. Intra-abdominal pressures exceeding 10–12 mmHg compress the inferior vena cava, reduce cardiac preload, and stimulate peripheral chemoreceptors and the renin-angiotensin-aldosterone system, collectively driving tachycardia, hypertension, and increased SVR. [9,10] The patient positioning required for upper abdominal laparoscopy further exacerbates these effects. [4] Effective pharmacological attenuation of this stress response is therefore central to safe anaesthetic management for laparoscopic procedures.

The present study demonstrated that dexmedetomidine infusion was consistently superior to propofol in attenuating the haemodynamic stress response across all measured parameters—HR, SBP, DBP, and MAP—from shortly after drug initiation through extubation. The magnitude of the divergence was particularly pronounced at the time of pneumoperitoneum and during the intraoperative period: dexmedetomidine produced a 3–5% reduction from baseline values, while propofol was associated with increases of 7–18%, reflecting the inability of propofol alone to counteract the sympathoadrenal surge of pneumoperitoneum. These findings are in close agreement with those of Janardhana and Thimmaiah [11], whose similar comparative study using identical drug dosing regimens reported superior haemodynamic stability in the dexmedetomidine group across all intraoperative phases, with particular benefit at the critical juncture of pneumoperitoneum.

The superiority of dexmedetomidine can be understood from its mechanism of action. By inhibiting presynaptic noradrenaline release from the locus coeruleus and peripheral sympathetic nerve terminals via alpha-2A receptor stimulation, dexmedetomidine reduces central sympathetic outflow and thereby attenuates both the cardiac and

vascular components of the stress response. [12] This sympatholytic effect is precisely the mechanism required to counteract pneumoperitoneum-induced catecholamine surges. Propofol, while it reduces SVR and blood pressure through GABA-A-mediated mechanisms, lacks the targeted sympatholytic profile of dexmedetomidine and may even allow compensatory tachycardia in response to its vasodilatory effects. [7]

Our results align with several published studies. Vora et al. [13] showed that dexmedetomidine (1 mcg/kg loading + 0.5 mcg/kg/h maintenance) produced significantly lower intraoperative mean HR and MAP compared with saline controls during laparoscopic surgery, with a concomitant reduction in fentanyl and isoflurane requirements. Chilkoti et al. [14] similarly demonstrated that dexmedetomidine 0.5 mcg/kg/h infusion significantly reduced the haemodynamic stress response to laparoscopic cholecystectomy and decreased postoperative analgesic consumption. Srivastava et al. [15] found dexmedetomidine more effective than esmolol in attenuating haemodynamic responses during laparoscopic cholecystectomy, further reinforcing its role as a preferred sympatholytic agent for this indication. Khare et al. [16] reported that dexmedetomidine reduced propofol requirements during induction and maintenance by 36% and 31% respectively while providing superior haemodynamic stability, confirming its anaesthetic-sparing potential.

On the question of postoperative recovery, the present study found that dexmedetomidine produced deeper postoperative sedation (higher RSS scores) persisting up to 90 minutes, and slower early recovery (lower MAS scores at 0–30 minutes). This sedative carryover is a recognised feature of dexmedetomidine owing to its elimination half-life of approximately 2 hours and prolonged receptor engagement. [12] Importantly, however, both groups achieved equivalent full recovery (MAS 10) by 75 minutes post-extubation,

and no patient experienced clinically problematic over sedation, respiratory depression, or delayed extubation in the dexmedetomidine group. The propofol group's faster early recovery aligns with its known pharmacokinetic advantages—rapid redistribution and a context-sensitive half-time of less than 10 minutes at infusion durations typical of laparoscopic surgery. [17]

Ghodki et al. [18] observed a 62.5% reduction in propofol dose and a 30% reduction in isoflurane requirements when dexmedetomidine was used as an adjuvant for laparoscopic surgery, findings consistent with the opioid- and anaesthetic-sparing effects we indirectly observed. Arain et al. [19] compared dexmedetomidine and propofol for intraoperative sedation and reported that dexmedetomidine achieved sedation more rapidly and provided superior postoperative analgesia, though propofol's earlier offset was preferred in day-surgery settings.

These complementary observations support the view that the two agents are not mutually exclusive—dexmedetomidine for haemodynamic protection and propofol for rapid recovery—and combination strategies may represent the optimal approach in high-risk patients.[20]

Limitations of the present study include the open-label design, which may introduce observer bias in haemodynamic recording; the relatively small sample size; the restriction of the study population to ASA PS I and II patients, limiting generalisability to higher-risk groups; and the absence of cardiac output monitoring, which would have allowed more detailed characterisation of the haemodynamic effects. Future blinded randomised trials with larger samples and advanced haemodynamic monitoring, including in high-risk cardiac or elderly populations, are warranted to confirm and extend these findings.

Conclusion

This study demonstrates that dexmedetomidine infusion (1 mcg/kg loading dose followed by 0.2 mcg/kg/h) provides significantly superior intraoperative haemodynamic stability compared with propofol infusion (100 mcg/kg/min) during laparoscopic surgery. Dexmedetomidine effectively attenuates the cardiovascular stress response to pneumoperitoneum—reducing heart rate, systolic and diastolic blood pressure, and mean arterial pressure relative to baseline—while propofol was associated with clinically meaningful increases in all parameters at the time of pneumoperitoneum.

Although dexmedetomidine produces greater postoperative sedation and slightly delayed initial recovery, full recovery was equivalent between groups by 45–60 minutes with no adverse events in either group. Dexmedetomidine should be

considered the preferred infusion agent for intraoperative haemodynamic management in laparoscopic surgery, particularly when cardiovascular stability is clinically paramount. Propofol remains the agent of choice where faster early recovery is the priority.

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