

Dexmedetomidine as an Adjuvant in Opioid Anaesthesia Induction in Patients with Left Ventricular Dysfunction and Coronary Artery Disease: A Prospective Randomised Observational Study

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Abstract

Background: Patients with coronary artery disease (CAD) and compromised left ventricular (LV) function represent a high-risk subgroup in cardiac surgery. High-dose opioid induction, while widely employed, carries risks of haemodynamic instability. Dexmedetomidine, a highly selective alpha-2 (α_2) adrenergic agonist, offers sympatholysis, sedation, and analgesia, and may attenuate the adrenergic response to laryngoscopy and intubation. This study aimed to compare the haemodynamic effects and opioid requirements during anaesthesia induction with fentanyl alone versus fentanyl supplemented with dexmedetomidine in patients with LV dysfunction undergoing off-pump coronary artery bypass grafting (OPCAB).

Methods: Sixty adult patients with LV dysfunction (ejection fraction <45%) undergoing elective OPCAB were prospectively randomised into two groups of 30 each: Group F (fentanyl alone) and Group D (fentanyl plus dexmedetomidine loading dose 1 mcg/kg over 10 minutes). Haemodynamic parameters and cardiac output indices were recorded at baseline and at one-minute intervals from induction to seven minutes post-induction using a Flo Trac™/Vigileo™ system. Bispectral Index (BIS) monitoring ensured anaesthetic depth. Fentanyl dosage at induction, additional intraoperative fentanyl requirements, and duration of postoperative ventilation were compared between groups.

Results: Demographic parameters were comparable between groups. Heart rate and systolic blood pressure were significantly elevated in Group F compared to Group D across all post-induction time points ($p < 0.001$). Diastolic blood pressure and oxygen saturation (SpO₂) remained similar in both groups. Stroke volume index (SVI) and cardiac index (CI) were significantly better maintained in Group D ($p < 0.001$). Fentanyl induction dosage (325 ± 25.4 mcg vs 253.3 ± 26.0 mcg) and additional intraoperative fentanyl (371.7 ± 28.4 mcg vs 185.0 ± 32.6 mcg) were significantly lower in Group D ($p < 0.001$). Duration of postoperative ventilation was also significantly shorter in Group D (5.6 ± 0.5 hrs vs 9.0 ± 0.9 hrs, $p < 0.001$).

Conclusion: Dexmedetomidine supplementation to fentanyl-based induction in patients with CAD and LV dysfunction provides superior haemodynamic stability, better preservation of cardiac output parameters, reduced intraoperative opioid requirements, and facilitates faster postoperative extubation, enabling early patient fast-tracking.

Keywords: Dexmedetomidine; Fentanyl; Opioid anaesthesia; Coronary artery bypass grafting; Left ventricular dysfunction; Haemodynamic stability; Cardiac index; Fast-tracking; Bispectral index.

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Introduction

Anaesthetic management of patients with coronary artery disease (CAD) and left ventricular (LV) dysfunction undergoing cardiac surgery poses significant challenges. Haemodynamic stability during induction is paramount, as this population is particularly vulnerable to extremes of heart rate,

blood pressure, and alterations in myocardial oxygen supply-demand balance. The principal goals of anaesthetic induction in this cohort include minimising the sympathoadrenal response to laryngoscopy and tracheal intubation, maintaining coronary perfusion pressure, and avoiding

myocardial depression. [1,2] High-dose opioid induction, particularly using fentanyl, has historically been the cornerstone of cardiac anaesthesia for patients with compromised ventricular function. [3] Fentanyl is a synthetic mu-opioid agonist that provides haemodynamic stability by blunting the stress response to noxious stimuli with minimal direct cardiovascular depression. However, high opioid doses contribute to prolonged postoperative sedation, delayed extubation, and increased intensive care unit (ICU) stay, thereby undermining the benefits of fast-track cardiac surgical protocols.

Dexmedetomidine, a highly selective alpha-2 (α_2) adrenoceptor agonist with an $\alpha_2:\alpha_1$ selectivity ratio of approximately 1620:1, has emerged as a promising adjuvant in cardiac anaesthesia. Acting centrally at the locus coeruleus and peripherally at sympathetic nerve terminals, dexmedetomidine produces dose-dependent sedation, anxiolysis, and analgesia through a non-opioid mechanism. By inhibiting noradrenaline release from sympathetic nerve endings, it attenuates the haemodynamic response to laryngoscopy, reduces perioperative tachycardia, and has demonstrated opioid-sparing properties. [4] Its unique quality of producing sedation through endogenous sleep-promoting pathways, without significant respiratory depression, makes it particularly attractive in the cardiac surgical setting.

Several studies have demonstrated favourable outcomes with dexmedetomidine in patients undergoing coronary artery bypass grafting (CABG), including reduction in myocardial ischaemia, attenuation of the intubation stress response, and facilitation of early extubation.^{5,6} However, evidence specifically addressing its role as an induction adjuvant in patients with established LV dysfunction — where cardiac output is already compromised — remains limited. This prospective randomised observational study was designed to evaluate the haemodynamic and cardiac output effects of adding dexmedetomidine to fentanyl during anaesthetic induction in patients with LV dysfunction and CAD undergoing elective off-pump CABG (OPCAB). Secondary objectives included assessment of intraoperative opioid requirements and postoperative ventilatory duration.

Methodology

This prospective, randomized comparative study was conducted over 2 years at a tertiary care

teaching hospital after obtaining informed consent, in accordance with ICH-GCP guidelines. A total of 60 patients (30 per group), determined by power analysis (80% power, $\alpha = 0.05$), were randomized using computer-generated allocation into Group F (fentanyl) and Group D (dexmedetomidine + fentanyl). Adult patients (≥ 18 years) with left ventricular dysfunction (EF $< 45\%$), ASA grade III–IV, undergoing elective off-pump coronary artery bypass grafting were included. Patients with emergency surgery, preoperative mechanical ventilation or IABP support, valvular disease, arrhythmias, redo surgery, or severe non-cardiac illness were excluded.

Standard preanaesthetic evaluation and monitoring (ECG, SpO₂, arterial line, BIS) were performed. Following preoxygenation and midazolam premedication (0.02 mg/kg), Group F received titrated fentanyl for induction, while Group D received dexmedetomidine (1 $\mu\text{g}/\text{kg}$ over 10 min) with fentanyl to achieve BIS 40–50. Vecuronium (0.1 mg/kg) facilitated intubation. Anaesthesia was maintained with oxygen, air, isoflurane (1–2%), fentanyl boluses, and vecuronium supplementation. Invasive monitoring included central venous and femoral arterial lines.

Haemodynamic parameters (HR, SBP, DBP, SpO₂) were recorded at baseline and every minute for 7 minutes post-induction. Cardiac index and stroke volume index were continuously measured using FloTrac™/Vigileo™. Total fentanyl requirement and duration of postoperative ventilation were recorded.

Data were analysed using Student's t-test (continuous variables) and Chi-square test (categorical variables). A p-value < 0.05 was considered significant and < 0.001 highly significant.

Results

Demographic Characteristics: Sixty patients were enrolled and randomly assigned to Group F (n=30) and Group D (n=30). The two groups were well matched for baseline demographic characteristics. The mean age was 61 ± 8.4 years in Group F and 62 ± 7.8 years in Group D (p=0.487). Mean weight (70 ± 7.6 kg vs 71 ± 7.8 kg, p=0.548) and mean height (151 ± 10.9 cm vs 154 ± 9.5 cm, p=0.297) were also comparable. The sex distribution was similar, with 63.3% males in Group F and 60% in Group D. No statistically significant differences in any demographic parameter were found between the groups, confirming adequate randomisation.

Table 1: Demographic Data Comparison between Group F and Group D

Parameter	Group F (n=30)	Group D (n=30)	p-value
Age (years), Mean \pm SD	61 \pm 8.4	62 \pm 7.8	0.487 (NS)
Weight (kg), Mean \pm SD	70 \pm 7.6	71 \pm 7.8	0.548 (NS)
Height (cm), Mean \pm SD	151 \pm 10.9	154 \pm 9.5	0.297 (NS)
Sex (Male/Female)	19/11 (63.3%/36.7%)	18/12 (60%/40%)	NS

NS = Not Significant; SD = Standard Deviation

Heart Rate and Systolic Blood Pressure: There was no significant difference in baseline heart rate (Group F: 72.1 bpm vs Group D: 72.7 bpm, $p=0.80$) or baseline systolic blood pressure (Group F: 139.2 mmHg vs Group D: 137.1 mmHg, $p=0.475$) between the groups.

Following induction, however, heart rate in Group F demonstrated a sustained and significant elevation across all observed time points (1–7 minutes), ranging from 84.6 to 86.9 bpm, compared to Group D where heart rates remained close to baseline (71.1–71.7 bpm), with a highly significant

difference at all time points ($p<0.001$). Similarly, systolic blood pressure was significantly higher in Group F after induction, particularly at 1 minute (141.2 mmHg vs 119.1 mmHg), and remained elevated (127.2–128.7 mmHg vs 108.0–108.5 mmHg) throughout the study period ($p<0.001$ at all time points). Diastolic blood pressure remained comparable between the groups at all time points (Group F: 69.5–70.8 mmHg vs Group D: 69.3–70.3 mmHg; $p>0.05$ at all time points), suggesting that dexmedetomidine's primary benefit was in modulating the sympathoadrenal response reflected in heart rate and systolic pressure.

Table 2: Heart Rate (bpm) and Systolic Blood Pressure (mmHg) After Induction

Time Point	HR - Group F	HR - Group D	SBP - Group F	SBP - Group D	p-value (HR & SBP)
Baseline	72.1	72.7	139.2	137.1	NS
1 min	86.9	71.4	141.2	119.1	<0.001 (HS)
2 min	85.9	71.7	128.3	108.5	<0.001 (HS)
3 min	86.2	71.1	128.7	108.2	<0.001 (HS)
4 min	85.8	71.3	128.0	108.5	<0.001 (HS)
5 min	85.3	71.2	128.0	108.0	<0.001 (HS)
6 min	85.1	71.5	128.1	108.5	<0.001 (HS)
7 min	84.6	71.3	127.2	108.2	<0.001 (HS)

HR = Heart Rate; SBP = Systolic Blood Pressure; HS = Highly Significant; NS = Not Significant

Cardiac Output Parameters: Baseline SVI was identical in both groups (38.2 ml/beat/m², $p=1.0$), and baseline CI was also equivalent (3.6 L/min/m², $p=0.945$).

Following induction, striking differences emerged between the groups. In Group D, SVI increased to values ranging from 43.7 to 44.3 ml/beat/m² across the observation period, while in Group F, SVI declined to 31.6–32.7 ml/beat/m², representing a statistically highly significant difference ($p<0.001$

at all time points). Cardiac index in Group D remained well preserved at 3.9–4.0 L/min/m² post-induction, compared to a notable fall to 2.4 L/min/m² in Group F at all time points ($p<0.001$). These findings indicate that dexmedetomidine supplementation effectively preserved, and even augmented, cardiac output parameters in patients with existing LV dysfunction during the critical induction period. SpO₂ was 100% in both groups after induction, with only minor non-significant differences at baseline (97.4% vs 97.6%, $p=0.669$).

Table 3: Stroke Volume Index (ml/beat/m²) and Cardiac Index (L/min/m²) After Induction

Time Point	SVI - Group F	SVI - Group D	CI - Group F	CI - Group D	p-value (SVI & CI)
Baseline	38.2	38.2	3.6	3.6	NS
1 min	32.7	44.3	2.4	3.9	<0.001 (HS)
2 min	31.6	43.7	2.4	4.0	<0.001 (HS)
3 min	32.7	44.3	2.4	3.9	<0.001 (HS)
4–7 min	31.6–32.7	43.7–44.3	2.4	3.9–4.0	<0.001 (HS)

SVI = Stroke Volume Index; CI = Cardiac Index; HS = Highly Significant; NS = Not Significant

Fentanyl Dosage Requirements: The induction fentanyl requirement was significantly lower in Group D (253.3 ± 26.0 mcg) compared to Group F (325.0 ± 25.4 mcg), a reduction of approximately 22% ($p < 0.001$). This opioid-sparing effect was even more pronounced for additional intraoperative fentanyl requirements: Group D patients required a

mean of 185.0 ± 32.6 mcg compared to 371.7 ± 28.4 mcg in Group F, representing a reduction of approximately 50% ($p < 0.001$).

These findings confirm a robust intraoperative opioid-sparing property of dexmedetomidine when used as an anaesthetic adjuvant.

Table 4: Fentanyl Dosage Requirements (induction and intraoperative)

Fentanyl Dosage	Group F Mean \pm SD (mcg)	Group D Mean \pm SD (mcg)	p-value
Induction Dosage	325.0 ± 25.4	253.3 ± 26.0	<0.001 (HS)
Additional Intraoperative Dosage	371.7 ± 28.4	185.0 ± 32.6	<0.001 (HS)

HS = Highly Significant; SD = Standard Deviation; mcg = micrograms

Duration of Postoperative Mechanical Ventilation: The duration of postoperative mechanical ventilation was significantly shorter in Group D (mean 5.6 ± 0.5 hours; median 6 hours) compared to Group F (mean 9.0 ± 0.9 hours; median 9 hours), with a highly significant p-value

of <0.001 . This reduction of approximately 3.4 hours in median ventilation time represents a clinically meaningful benefit in the context of fast-track cardiac surgery, with implications for ICU resource utilisation, patient comfort, and potentially reduced pulmonary complications.

Table 5: Duration of Postoperative Mechanical Ventilation

Parameter	Group F	Group D	p-value
Duration of Post-op Ventilation (hrs), Mean \pm SD	9.0 ± 0.9	5.6 ± 0.5	<0.001 (HS)
Median Duration (hrs)	9	6	<0.001 (HS)

HS = Highly Significant; SD = Standard Deviation; hrs = hours

Discussion

This study demonstrates that dexmedetomidine, when used as an adjuvant to fentanyl-based anaesthetic induction, confers significant haemodynamic and clinical advantages over fentanyl used alone in patients with CAD and LV dysfunction undergoing OPCAB surgery. The findings are consistent with and extend the existing literature on alpha-2 agonist use in cardiac anaesthesia.

The most clinically salient finding of this study was the sharp divergence in heart rate and systolic blood pressure between the two groups following induction, while diastolic blood pressure remained stable in both. In Group F, there was a sustained tachycardia and systolic hypertension immediately following induction, peaking at 1 minute and persisting throughout the observation period. This haemodynamic profile reflects the residual sympathoadrenal response to the noxious stimuli of mask ventilation and endotracheal intubation that fentanyl alone was unable to completely suppress.

In contrast, Group D patients demonstrated near-baseline heart rates and significantly lower systolic pressures. This is attributable to dexmedetomidine's central sympatholytic action — through stimulation of postsynaptic α_2 receptors in the locus coeruleus — which reduces noradrenaline release, thereby diminishing tachycardia and hypertensive responses. These findings are corroborated by Gumus et al., [5] who similarly reported more

stable systolic arterial pressures with dexmedetomidine-narcotic induction in CABG patients, and by Sulaiman et al., [6] who demonstrated attenuation of the intubation stress response with dexmedetomidine pretreatment in OPCAB patients even in the presence of beta-blocker therapy. The cardiac output data in this study are particularly compelling. The significant fall in SVI and CI in Group F following induction, without a corresponding drop in Group D, suggests that high-dose fentanyl alone may impair cardiac performance during induction in the already-compromised LV. This may be mediated partly through fentanyl's vagotonic bradycardia effect and partly through the unopposed haemodynamic lability of induction in the absence of sympatholytic support. Dexmedetomidine, by providing sympatholysis and a more stable preload/afterload balance, appears to support rather than impair ventricular function during induction. This is consistent with the known favourable effects of dexmedetomidine on myocardial oxygen balance and peri-operative ischaemia reported in both cardiac and non-cardiac surgical contexts. [7-9]

The opioid-sparing properties of dexmedetomidine observed in this study — a 22% reduction in induction fentanyl dose and a 50% reduction in additional intraoperative fentanyl — are clinically significant. Alpha-2 adrenergic receptors in the dorsal horn of the spinal cord modulate substance P release, contributing to the analgesic properties of

dexmedetomidine. [10] The activation of these receptors supplements opioid-mediated analgesia, reducing the total opioid burden. This is particularly relevant in patients with LV dysfunction, where high cumulative opioid doses potentiate the risk of prolonged respiratory depression and delayed extubation. Brandao et al. [8] similarly reported improved outcomes with dexmedetomidine as an anaesthetic adjuvant in patients undergoing CABG and valve surgery, affirming the opioid-sparing and haemodynamic stabilising benefits. The shortened postoperative ventilation time in Group D (5.6 hours vs 9.0 hours) is a direct downstream consequence of the reduced intraoperative opioid load. Faster emergence from opioid-induced respiratory depression allows earlier weaning and extubation, which is the defining goal of fast-track cardiac anaesthesia. Kamel et al. [7] reported analogous benefits of dexmedetomidine in paediatric cardiac surgery, noting earlier extubation and reduced ICU stay. These parallel findings across different populations reinforce the generalisability of the opioid-sparing and extubation-facilitating properties of dexmedetomidine.

The maintenance of diastolic blood pressure and SpO₂ comparably between both groups is reassuring. While dexmedetomidine is known to produce hypotension via central pre-synaptic α_2 stimulation and resulting noradrenaline suppression [11], this was not observed as a clinically problematic effect in our cohort, likely because the drug was administered as a slow infusion over 10 minutes, avoiding the transient initial hypertension associated with rapid bolus administration due to peripheral α_2B receptor stimulation. The haemodynamic safety profile in LV dysfunction patients appears acceptable with this administration protocol. [12-14]

Several limitations of this study warrant acknowledgment. The study endpoint was set at seven minutes post-induction, limiting long-term haemodynamic and outcome data beyond the induction period. The sample size, while adequately powered for primary endpoints, may not be sufficient to detect differences in less common events such as intraoperative ischaemia or arrhythmias.

All patients underwent off-pump CABG, so findings may not be directly extrapolatable to on-pump procedures where cardiopulmonary bypass introduces additional physiological perturbations. Future multicentre studies with larger cohorts and extended follow-up, including long-term outcomes such as mortality, ICU stay, and cognitive function, would further consolidate the role of dexmedetomidine in this high-risk population.

Conclusion

Dexmedetomidine supplementation to fentanyl anaesthetic induction in patients with coronary artery disease and compromised left ventricular function provides superior haemodynamic stability, with significantly lower heart rates and systolic blood pressures during the critical post-induction and peri-intubation period compared to fentanyl induction alone.

Cardiac output parameters — specifically cardiac index and stroke volume index — are significantly better preserved in the dexmedetomidine group, underscoring its haemodynamic safety in this vulnerable population. The adjuvant use of dexmedetomidine substantially reduces intraoperative opioid requirements, translating into meaningfully shorter postoperative mechanical ventilation duration and facilitating fast-tracking of cardiac surgical patients. Dexmedetomidine may be recommended as a standard adjuvant in opioid-based anaesthetic induction protocols for high-risk cardiac surgical patients with LV dysfunction.

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