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Hemodynamic Safety of Dexmedetomidine-Fentanyl Sedation During TAVI

ABSTRACT

Background: Transcatheter aortic valve implantation (TAVI) is an established alternative for patients with severe aortic stenosis who are unsuitable for surgical valve replacement. Conscious sedation is preferred to preserve spontaneous respiration and patient cooperation. Dexmedetomidine, a selective α_2-adrenergic agonist, provides sedation, analgesia, and sympatholysis with minimal respiratory depression, making it suitable for high-risk TAVI patients.

Methods: We retrospectively analyzed 53 patients who underwent TAVI under dexmedetomidine-based sedation at a single center between January and July 2025. Patients received an initial loading dose of dexmedetomidine (1 µg/kg over 15 minutes) and fentanyl (1 µg/kg), followed by dexmedetomidine infusion (0.2-1.2 µg/kg/h) to achieve a Ramsay Sedation Score of 3-4 and bispectral index (BIS) 70-80. Hemodynamic parameters were recorded at baseline (T0), post-loading (T1), 10 minutes post-loading (T2), and end of procedure (T3). Hemodynamic compromise was defined as a >30% decrease in systolic or mean arterial pressure(MAP) <65 mm Hg.

Results: The mean age was 76.4 ± 7.3 years, with 58.5% female; all patients were ASA III–IV. Mean arterial pressure (MAP) remained above 65 mm Hg at all time points, with the greatest decrease at T2. Systolic and MAP reductions were consistently below the 30% threshold. Postoperative complications included pacemaker implantation in 2 patients, transient contrast-induced nephropathy in 1, and temporary inotropic support in 4. No anesthesia-related respiratory complications occurred.

Conclusions: Dexmedetomidine combined with fentanyl provides safe and effective sedation for TAVI, maintaining hemodynamic stability and spontaneous respiration. This sedation protocol minimizes perioperative risks and may improve procedural safety in high-risk patients.

Keywords: Conscious sedation, dexmedetomidine, fentanyl, hemodynamic stability, sedation, TAVI, transcatheter aortic valve

ORIGINAL INVESTIGATION

INTRODUCTION

Degenerative calcific aortic stenosis is the most frequent valvular heart disease in Western countries, with a prevalence of about 3% after the age of 75.[1] In severe cases, surgical valve replacement is the standard treatment; however, approximately 30% of these patients are not candidates for surgery due to limited life expectancy and advanced comorbidities. Up to one-third of patients who require lifesaving surgical aortic valve replacement are denied surgery due to a high operative mortality rate.[2] In this patient group, transcatheter aortic valve implantation (TAVI) is considered a life-saving alternative,[3] and this procedure can be performed under conscious sedation. Controlled hypotension is achieved during valve implantation or balloon procedures via a transvenous pacemaker. Transvenous pacing is often used during TAVI procedures to achieve controlled hypotension during valve implantation or balloon valvuloplasty.[4]

Conscious sedation during TAVI aims to prevent pain and discomfort while allowing patient communication.[5] In patients with respiratory instability, severe orthopnea, or those who cannot tolerate sedation, orotracheal intubation and general anesthesia may be necessary during TAVI procedures.

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Dexmedetomidine is a sedative agent with anxiolytic, hypnotic, analgesic, and sympatholytic properties, making it suitable for sedation during TAVI procedures.[6]

It exerts its effects through α_2-adrenergic receptors in the central, peripheral, and spinal cord, without affecting GABA receptors.[7] Dexmedetomidine's ability to maintain spontaneous respiration and patient cooperation makes it a preferred choice in sedative procedures, especially in high-risk patients. Moreover, animal studies have demonstrated that dexmedetomidine protects the heart from ischemic injury, stabilizes cardiac electrophysiology, and prevents arrhythmias.[8] However, its potential to cause bradycardia should be considered, as it may lead to hemodynamic instability in patients with severe aortic stenosis.

This retrospective study aimed to evaluate our institutional experience with dexmedetomidine-based sedation in patients undergoing TAVI.

METHODS

This single-center retrospective study was conducted after obtaining approval from the Local Ethics Committee (decision no. 2025/412, dated 27/08/2025). The need for written informed consent was waived by the ethics committee due to the retrospective nature of the study using the electronic medical records and perioperative anesthesia documents. Data were recorded from the electronic medical records and perioperative anesthesia documents. Patients with missing data, those who received general anesthesia, patients whose anesthesia method was changed for any reason, and those who received sedation techniques other than the routine institutional protocol were excluded from the study.

In our routine protocol, patients undergoing TAVI routinely receive standard ASA monitoring, including invasive arterial pressure monitoring and bispectral index (BIS) monitoring. For sedation, an initial intravenous dose of fentanyl (1 µg/kg) and dexmedetomidine (1 µg/kg over 15 minutes) is administered, followed by a dexmedetomidine infusion at 0.2-1.2 µg/kg/min for maintenance of sedation. If bradycardia occurs during the procedure, the infusion rate is reduced or an alternative anesthetic agent is administered. The maintenance dose is titrated to achieve a Ramsay Sedation Score of 3-4 and a BIS value between 70 and 80.

HIGHLIGHTS

- Dexmedetomidine-fentanyl sedation maintained stable hemodynamics during transcatheter aortic valve implantation (TAVI).
- No anesthesia-related respiratory complications were observed.
- Conversion to general anesthesia was not required in any patient.
- Postoperative complications were minimal and manageable.
- This sedation protocol may enhance safety in high-risk TAVI patients.

Data Collection and Hemodynamic Assessment

Patient demographics, including age, sex, weight, American Society of Anesthesiologists (ASA) physical status classifications, comorbidities, ejection fraction, hospital length of stay, intensive care unit stay, and 1-week and 1-month mortality were recorded. From anesthesia monitoring forms, data on the type of anesthesia administered, drugs used, pre-procedural blood pressure, heart rate, and peripheral oxygen saturation were collected, as well as intra-procedural hemodynamic parameters following drug administration, complications related to anesthesia or the procedure.

Hemodynamic effects after drug administration were assessed by changes in systolic and mean arterial pressures (MAPs). A decrease of more than 30% from baseline in systolic or MAP, or a systolic arterial pressure below 90 mmHg or MAP below 65 mm Hg, was considered a hemodynamic compromise. Baseline hemodynamic data were recorded as T0, hemodynamic parameters after dexmedetomidine administration as T1, measurements 10 minutes after completion of drug administration as T2, and hemodynamic parameters at the end of the procedure as T3.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics Standard Concurrent User V30 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables are presented as mean ± standard deviation (SD).

A 1-sample *t*-test was used to compare the sample means with pre-determined reference values. A ≥30% decrease in systolic blood pressure and MAP was defined as the clinically significant threshold. Additionally, a reference value of 65 mm Hg for MAP was considered. In all analyses, a *P*-value of $< .05$ was considered statistically significant.

RESULTS

During the specified study period, data from 68 patients were collected. Of these, 4 patients received general anesthesia, 2 patients developed cardiac arrest due to mechanical complications following valve opening and were switched to general anesthesia, and 2 patients could not receive dexmedetomidine due to bradycardia. Additionally, 5 patients were excluded due to incomplete data. Consequently, data from 53 patients who received sedation and analgesia with dexmedetomidine were analyzed (Figure 1). The mean age of the patients was 76.4 ± 7.3 years, with 58.5% female and 41.5% male. All patients were classified as ASA III or ASA IV (52.8% and 47.2%, respectively). Comorbidities of the patients were diabetes mellitus (66%), coronary artery disease (39.6%), hypertension (32.1%), heart failure (32.1%), chronic obstructive pulmonary disease (11.3%), renal disease (11.3%), and obstructive sleep apnea syndrome (1.9%). Demographic data are presented in Table 1.

The mean systolic arterial pressure of the patients was highest at T0 and lowest at T2 (T0: 150 [25], T1: 110 [18], T2: 100 [15], and T3: 130 [26]). At all time points, the MAP remained above 65 mm Hg. The hemodynamic parameters and sedation scores of the patients are summarized in Table 2.

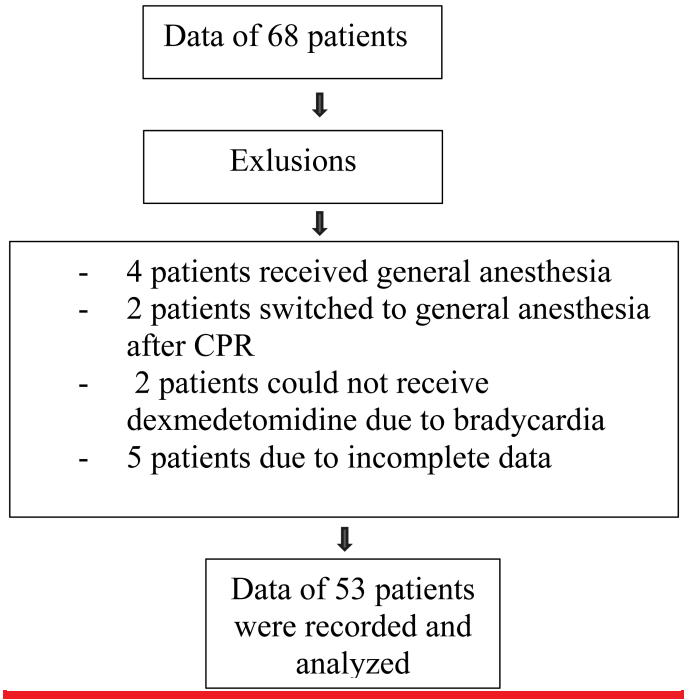

Figure 1. Flow chart.

The mean MAP after drug administration was 86.08 ± 10.6 mm Hg at T1 (difference: +21.07; 95% CI: 18.15-24.00; t(52) = 14.436; $P < .001$), 79.70 ± 12.25 mm Hg at T2 (difference: +14.69; 95% CI: 11.32-18.08; t(52) = 8.731; $P < .001$), and 92.09 ± 13.73 mm Hg at T3 (difference: +27.09; 95% CI: 23.31-30.88; t(52) = 14.358; $P < .001$) (Table 3).

The decrease in MAP after drug administration was significantly lower than the 30% reference value at all time points (Table 4). The mean decrease at T1 was 17.44% ± 15.17% ($P < .001$), at T2 it was 7.17% ± 10.29% ($P < .001$). At T3, the mean change was −7.93% ± 17.51%, indicating that MAP increased by approximately 8 mmHg instead of decreasing ($P < .001$).

Table 1. Demographic Data

Age, years	76.4 ±7.3
Weight	75 [18.5]
EF median	60 [13]
Gender (%)	
Female	31 (58.5)
Male	22 (41.5)
ASA status (%)	
ASA-III	28 (52.8)
ASA IV	25 (47.2)
Comorbidities %	
Diabetes mellitus	35 (66.0)
Coronary artery disease	21 (39.6)
Hypertension	17 (32.1)
Heart failure	17(32.1)
COPD	6 (11.3)
Renal disease	6 (11.3)
OSAS	1 (1.9)
ECG	
SR	48 (90.6)
AF	5 (9.4)
Procedure time (min)	55 [20]

Data are presented as median (IQR), mean ± SD or number (% percentage).
AF, atrial fibrillation; ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; ECG, electrocardiography; OSAS, obstructive sleep apnea syndrome; SR, sinus rhythm.

The decrease in systolic arterial pressure after drug administration varied according to time points when compared with the 30% decrease reference value (Table 5). At T1, the mean decrease was 21.94 ± 9.55%, which was significantly lower than 30% ($P < .001$). At T2, the mean decrease was 28.01% ± 14.46%, showing no significant difference from 30% ($P = .161$). At T3, the mean decrease was 14.30% ± 15.32%, significantly lower than 30% ($P < .001$).

The mean intensive care unit (ICU) stay of the patients was 1.94 ± 1.72 days, and the total hospital stay was 5.3 ±

Table 2. Hemodynamic Parameters and Sedation Score at Different Time Points

Time	SBP (mm Hg)	DBP (mm Hg)	MAP (mm Hg)	HR (beats/min)
T0	154.02 ± 22.6	81.47 ± 10.8	105.58 ± 12.57	81.58 ± 12.5
T1	119 ± 21.3	69.6 ± 8.07	86.08 ± 10.62	67.17 ± 9.9
T2	100.9 ± 18.3	65.06 ± 11.8	79.7 ± 12.2	63.21 ± 9.3
T3	130.53 ± 22.9	73.09 ± 10.5	92.09 ± 13.7	74.75 ± 12.3

Data are presented as mean ± SD, based on distribution. Normality was assessed using the Kolmogorov–Smirnov test.
DBP, diastolic blood pressure; HR, heart rate; MAP, mean blood pressure; SBP, systolic blood pressure; T0, baseline; T1, after loading dose; T2, 10 minutes later; T3, end of the procedure.

Table 3. Comparison of MAP with Reference Value (65 mm Hg)

MAP	Mean ± SD	Mean Difference (mm Hg)	95% CI	t (df)	*P*
T1	86.08 ± 10.6	+21.07	18.15-24.00	14.436 (52)	< .001
T2	79.70 ± 12.25	+14.69	11.32-18.08	8.731 (52)	< .001
T3	92.09 ± 13.73	+27.09	23.31-30.88	14.358 (52)	< .001

$P < .05$ was considered statistically significant.
CI, confidence interval; MAP, mean arterial pressure; SD, standard deviation; t (df), *t*-test statistic and degrees of freedom.

Table 4. MAP Change Compared to 30% Reference Decrease

MAP decrease	Mean ± SD	Mean Difference (%)	95% CI	t (df)	*P*
T1	17.44 ± 15.17	−12.56	−−16.74-8.38	−6.026 (52)	< .001
T2	7.17 ± 10.29	−22.82	−25.65-−19.98	−16.132 (52)	< .001
T3	−7.93 ± 17.51	−37.93	−42.75-−33.10	−15.76 (52)	< .001

P < .05 was considered statistically significant.
CI, confidence interval; MAP, mean arterial pressure; t (df), *t*-test statistic and degrees of freedom; SD, standard deviation.

Table 5. SBP Change Compared to 30% Reference Decrease

SBP decrease	Mean ± SD	Mean Difference (%)	95% CI	t (df)	*P*
T1	21.94 ± 9.55	−8.056	−10.69-−5.42	−6.141(52)	< .001
T2	28.01 ± 14.46	−1.985	−5.97-2.001	−0.999 (52)	.161
T3	14.30 ± 15.32	−15.69	−19.92-−11.47	−7.456 (52)	< .001

P < .05 was considered statistically significant.
CI, confidence interval; t (df), *t*-test statistic and degrees of freedom; SBP, systolic blood [ressure; SD, standard deviation.

2.55 days. Postoperatively, 2 patients required pacemaker implantation due to heart block, 1 patient developed transient contrast-induced nephropathy, and 4 patients received temporary inotropic support. No patient required inotropic support prior to valve opening. No anesthesia-related complications were observed.

DISCUSSION

Dexmedetomidine is a highly selective α_2-adrenergic receptor agonist that has gained widespread use in sedative procedures due to its unique pharmacological profile. Unlike traditional sedatives such as propofol or benzodiazepines, dexmedetomidine provides sedation while preserving respiratory drive, allowing patients to remain arousable and cooperative during procedures. Its anxiolytic, hypnotic, and sympatholytic properties make it particularly valuable in procedures where patient cooperation and spontaneous respiration are essential, including dental sedations, endoscopic interventions, fiberoptic interventions, minor surgical procedures, and cardiac catheterizations.[9-12]

In recent years, dexmedetomidine has also been increasingly investigated as a sedative agent in patients undergoing TAVI. Compared to agents such as propofol, midazolam, and remifentanil, dexmedetomidine has been associated with more stable hemodynamic parameters and lower rates of respiratory depression, which are critical considerations in this high-risk population.[6,13,14] Mayr et al[13] compared dexmedetomidine with a combination of propofol-opioid during transfemoral TAVI and found that dexmedetomidine offered better hemodynamic support, less need for conversion to general anesthesia, and more favorable gas exchange parameters.[13] Another more recent systematic review by Chowdhury et al[15] compared dexmedetomidine vs. propofol for sedation in adult patients undergoing cardiac procedures and reported that dexmedetomidine demonstrated superior respiratory safety—fewer incidents of respiratory depression—while maintaining hemodynamic stability. Moreover, its sympatholytic effects may attenuate peri-procedural stress responses, further supporting its role as a preferred agent in TAVI procedures. Song et al[16] reported that in patients undergoing TAVI, the use of dexmedetomidine was associated with significant reductions in myocardial injury markers such as troponin I and CK-MB, suggesting a potential cardioprotective role. This finding indicates that in procedures with high cardiac stress, such as TAVI, dexmedetomidine may provide not only effective sedation but also myocardial protection.

In the context of TAVI, especially in elderly patients with severe aortic stenosis and multiple comorbidities, sedation management poses significant challenges. Hemodynamic instability, respiratory depression, and procedural complications are major concerns. Our study demonstrates that a combination of dexmedetomidine and fentanyl provides effective sedation while maintaining MAP above clinically significant thresholds and avoiding significant drops in systolic blood pressure. Importantly, no anesthesia-related respiratory complications occurred, highlighting the safety of this regimen in a vulnerable patient population.

These findings align with previous reports suggesting that dexmedetomidine offers superior hemodynamic and respiratory safety compared to traditional sedatives. The absence of severe perioperative complications, along with stable hemodynamics and preserved patient cooperation, underscores the clinical relevance of this sedation strategy.

Clinically, this study provides practical evidence that dexmedetomidine-fentanyl sedation can be safely implemented in routine TAVI procedures in patients at high perioperative risk. This approach may contribute to reduced hemodynamic stress and improved overall procedural safety. Our results may guide anesthesiologists in optimizing sedation protocols for elderly, high-risk TAVI patients, reinforcing the importance of individualized, hemodynamically stable sedation strategies in structural heart interventions.

Study Limitations

This study is retrospective and single-center, with a modest sample size. Future prospective, multicenter studies are warranted to confirm these findings and to further assess the impact of dexmedetomidine-fentanyl sedation on clinical outcomes such as long-term cardiovascular events and hospital resource utilization.

CONCLUSION

In conclusion, dexmedetomidine combined with fentanyl provides safe and effective sedation for TAVI, maintaining hemodynamic stability and spontaneous respiration. This regimen minimizes perioperative risk, improves procedural safety in high-risk patients, and represents a clinically relevant alternative to general anesthesia.

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Ethics Committee Approval: This study was approved by the Ethics Committee of İzmir City Hospital (Approval No.: 225/412; Date: August 27, 2025).

Informed Consent: No informed consent was obtained from the patients due to the retrospective design of the study, and the requirement for informed consent was waived by the Local Ethics Committee.

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Declaration of Interests: The authors have no conflicts of interest to declare.

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