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THE EFFECTS OF DEXMEDETOMIDINE ON HEMODYNAMIC RESPONSES TO ENDOTRACHEAL INTUBATION IN HYPERTENSIVE PATIENTS UNDERGOING SURGERY

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Abstract

BACKGROUND: *Dexmedetomidine is an alpha-2 adrenergic agonist with sedative, anxiolytic, and analgesic properties. This study aimed to assess the inhibitory effects of preoperative administration of 0.5 μg/kg dexmedetomidine on hemodynamic responses caused by endotracheal intubation in elderly hypertensive patients.*

METHODS: *Sixty elderly patients (≥ 60 years old) with controlled hypertension and classified as American Society of Anesthesiologists physical status II, scheduled to undergo elective noncardiac surgery, were randomly selected and assigned to 2 groups. Group C received normal saline, and group D received 0.5 μg/kg dexmedetomidine intravenously over 10 min just before endotracheal intubation. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) were recorded preoperatively in the ward, immediately after study drug administration, and at 1, 3, and 5 min after endotracheal intubation.*

RESULTS: *Compared to group C, group D showed significantly lower SBP and MAP at 1, 3, and 5 min as well as significantly lower DBP and HR at 3 and 5 min after endotracheal intubations.*

CONCLUSION: *In elderly patients with hypertension, administering a single pre-anesthetic dose of dexmedetomidine (0.5 μg/kg) significantly reduced the hemodynamic responses during endotracheal intubation.*

INTRODUCTION

General anesthesia during surgery induces a state of controlled unconsciousness making the patient unaware and insensitive to pain. Laryngoscopy and endotracheal intubation are mostly used to maintain airway while the patient is unconscious.[1] Direct laryngoscopy and tracheal intubation can induce significant hemodynamic changes due to sympathetic adrenergic outflow from laryngeal tissue stimulation. This response may lead to hypertension, tachycardia,[2] arrhythmias, and increases in intracranial and intraocular pressures. Reid and Brace first described these changes, which typically occur within five seconds of laryngoscopy, peak in 1-2 minutes, and return to baseline within 5 minutes. [1,3]

While usually inconsequential in healthy individuals, these effects may lead to serious morbidity in patients with coexisting cerebrovascular or cardiovascular conditions.[3] Additionally, those treated for

hypertension may experience an exaggerated hemodynamic response, further increasing the risk of complications. [4,5] Although transient, this exaggerated response may precipitate hypertensive crises, myocardial ischemia, arrhythmias, increases in intracranial pressure [2] , cardiac decompensation, pulmonary edema, and cerebral hemorrhage, especially in elderly patients with comorbid disease.[6]
To mitigate the sympathetic response and prevent cardiovascular reflexes during laryngoscopy and tracheal intubation, several agents have been used perioperatively, such as opioids, N-Methyl-D-aspartate (NMDA) receptor antagonists, alpha-2 agonists, beta-blockers, local anesthetics such as lidocaine, vasodilators, magnesium, or increased concentrations of volatile anesthetics, and calcium channel blockers. [1,3,8]

Dexmedetomidine is a potent and selective agonist of α-2 adrenoceptors, widely utilized in anesthesiology due to its short distribution half-life of around six minutes. [7] Unlike many sedatives, it maintains stable cardiovascular function and causes only mild respiratory suppression while providing sedation, pain relief, anxiety reduction, and inhibition of sympathetic nervous system activity. These effects are chiefly mediated by the stimulation of α-2A receptors located in the locus coeruleus. Its cardiovascular effects result from a dose-dependent reduction in the central sympathetic outflow. An initial transient hypertensive response occurs due to dexmedetomidine's action on vascular smooth muscle α-2B receptors before central sympathetic outflow diminishes. [10] Studies indicate that dexmedetomidine enhances cardiac outcomes by reducing perioperative oxygen demand and attenuating the sympathetic surge caused by laryngoscopy and surgical stress. Postoperative infusions help stabilize hemodynamics, lower plasma catecholamine levels, and decrease the requirement for additional anesthetics and analgesics. [9]
However, much of the existing research has focused on young, normotensive individuals. In contrast, elderly patients with comorbidities face a greater risk of hemodynamic instability due to their reduced cardiovascular reserve capacity, making them more vulnerable than the studied populations. [7]
In general, dose reduction of dexmedetomidine is recommended for elderly patients (≥ 60 years old), and the US Food and Drug Administration recommends a loading dose of 0.5 μg/ kg instead of 1 μg/kg. Furthermore, multiple studies have demonstrated that a 0.5 μg/kg loading dose remains effective across different age groups, including both younger and older patients.
However, this particular study specifically examined elderly hypertensive patients, who typically experience more pronounced hemodynamic reactions to sympathetic activation. Consequently, a standard 0.5 μg/kg dexmedetomidine loading dose may be insufficient to fully suppress the blood pressure and heart rate fluctuations triggered by endotracheal intubation in this high-risk population. Only a few international studies and none from within the country were found in the literature.
This study primarily aimed to evaluate whether dexmedetomidine could effectively attenuate the sympathetic stress response induced by tracheal intubation in hypertensive patients. Specifically, it investigated how a preoperative intravenous dose of 0.5 mcg/kg dexmedetomidine influenced hemodynamic stability in medicated hypertensive individuals during this critical procedure.
The sample size was calculated through Open EPI Sample Size Calculator (Available at http://www.openepi.com/SampleSize/SSMean.html) by taking MAP after 5 min in group C= 103.9± 13.3 Ref [4] and taking MAP after 5 min in group C = 83.40±8.40 Ref [4] , power (1-β) =80%. The total calculated sample size was 10 patients (5 in each group). Assuming a 20% dropout rate, we planned to enroll 60 patients.
Data was analyzed by using IBM SPSS Statistics version 26. Normality was checked by Shapiro w\Wilk test. Mean and standard deviation or median and interquartile range was computed for quantitative variable i.e. age, weight and duration of hypertension, SBP, DBP, MAP at baseline, after drug administration, 1 minute after intubation, 3 minutes after intubation and 5 minutes after intubation. Frequency and percentage were calculated for qualitative variables i.e., gender, comorbid other than hypertension and drug used as per need. Differences in SBP, DBP & MAP between the treatment groups was compared by using independent t test/ Mann-Whitney U-test.

Difference in SBP, DBP & MAP at baseline, after drug administration, after 1 minute, after 3 minutes and after 5 minutes of intubation was done using repeated measures of ANOVA/ Friedman test as appropriate.

Patients were stratified according to age, body weight, duration of hypertension, gender, non-hypertensive comorbidities, and medication regimens. Following stratification, between-group comparisons were performed using independent t-tests or Mann-Whitney U tests as appropriate for the data distribution. P<0.05 was considered as significant.

Materials and Methods:

Following approval by our hospital's institutional review board, written informed consent was acquired from all study participants. This randomized controlled trial was prospectively registered on ClinicalTrials.gov (Registration No. NCT06712186) in December 2024 prior to patient enrollment.

We screened 70 patients and 62 patients of both sexes aged between 60 and 85 years, with American Society of Anesthesiologists physical status II, Mallampati classification I or II, body mass index (BMI) ≤ 35 kg/m2, and scheduled to undergo elective noncardiac surgery were enrolled in this study. All enrolled participants had confirmed hypertension and were receiving ongoing pharmacological management with antihypertensive agents. We excluded 3 patients with BMI ≥ 35 kg/m2. The dropout rate was 7%. Two patients in whom the endotracheal intubation attempt lasted longer than 30s were excluded. The flow chart for the study is presented at Fig. 1.

Through concealed envelope randomization, 62 preoperative patients were equally divided into control (normal saline) and experimental (dexmedetomidine) groups. Study drug preparation was performed by a blinded anesthesiologist not involved in patient assessment or data collection. Patients were premedicated with 0.2 mg glycopyrrolate intravenously and antihypertensive medications were maintained until the day of surgery. Upon entering the operating theater, all participants underwent standard American Society of Anesthesiologists (ASA) monitoring, including continuous electrocardiography, pulse oximetry, and non-invasive blood pressure measurement. Group D patients (n =32) were administered 0.5 mcg/kg dexmedetomidine (PrecedexTM; Hospira Inc., Lake Forest, IL, USA) intravenously over 10 min. Patients in Group C received an equivalent volume of intravenous normal saline infused over 10 minutes. Both saline and dexmedetomidine were delivered via syringe pump under the supervision of a blinded anesthesiologist unfamiliar with the study protocol. Following infusion completion, anesthesia induction was achieved with Propofol (1.5-2.5 mg/kg, titrated to effect) and Atracurium (0.5 mg/kg). Endotracheal intubation was performed precisely two minutes later using direct laryngoscopy, with all procedures completed in under 30 seconds by a single anesthesiologist to ensure consistency.

Anesthesia maintenance initially consisted of 2% Sevoflurane in a 50% nitrous oxide/oxygen mixture for five minutes, followed by 1.5-2.5% Sevoflurane in a 50% air/oxygen blend. Hemodynamic parameters - including systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP), along with heart rate (HR) - were recorded at multiple time points: preoperatively in the ward (baseline), immediately post-drug administration, and at 1-, 3-, and 5-minutes post-intubation.

RESULTS;

Table 1 presents the demographic characteristics of both study groups. The analysis revealed no statistically significant differences in baseline demographic parameters between the control (Group C) and intervention (Group D) cohorts.

Hemodynamic monitoring demonstrated significantly lower mean systolic blood pressure values in Group D compared to Group C at all measured post-intubation time points (1, 3, and 5 minutes), as detailed in Table 2.

In group C, SBP was significantly higher at 1 min (186.5±14.69, P < 0.001) and 3 min (169.23±12.30, P < 0.001) after intubation than at baseline (145.4±13.75), and in group D, SBP was significantly higher at 1 min (136.5±25.04, P < 0.001) but lower at 3 (122.4±20.21, <0.002) and 5 min (112.07±17.22, P < 0.001) after intubation than at baseline (136.8±13.53) (Fig. 2).

The dexmedetomidine group (D) demonstrated significantly lower mean diastolic blood pressure (DBP) compared to the control group (C) at 1, 3, and 5 minutes post-intubation (p<0.05). In Group C, DBP

values showed a marked elevation from baseline (83.37 ± 13.50 mmHg) to 1-minute (105.2 ± 12.65 mmHg, p<0.001) and 3-minute (91.20 ± 8.52 mmHg, p=0.011) measurements following intubation. In group D, DBP was slightly higher at 1 min (81.03±16.36, P = 0.170) and lower at 5 min (64.67±7.64, P < 0.001) after intubation than at baseline (76.10±8.31) (Fig. 3)

Hemodynamic analysis revealed significantly lower mean arterial pressure values in the dexmedetomidine group (D) compared to controls (C) during all post-intubation measurements (1, 3, and 5 minutes; p<0.05), as documented in Table 2. In group C, MAP was significantly higher at 1 min (133.2±12.68, P < 0.001) and 3 min (117.8±9.94, P < 0.001) after intubation than at baseline (105.1±12.79), and in group D, MAP was unchanged at 1 min (98.87±17.67, P=0.676) and significantly lower at 3 min(86.90±10.8,P <0.001) and 5 min (81.07±9.99, P < 0.001) after intubation than at baseline (97.13±10.86) (Fig. 4)

While heart rate values were significantly lower in the dexmedetomidine group at 1 and 3 minutes post-intubation (both p<0.05), both groups showed comparable rates by 5 minutes (Table 2). In group C, HR was significantly higher at 1 min (102.3±11.59, P < 0.001) and 3 min (87.43±11.67, P < 0.001) after intubation than at baseline (79.20±11.32), and in group D, HR was significantly higher at 1 min (86.37±9.21, P < 0.001) but lower at 5 min (72.60±8.14, P <0.001) after intubation than at baseline (80.60±15.26) (Fig. 5).

DISCUSSION

The mechanical stimulation of laryngoscopy and subsequent intubation triggers significant but transient autonomic responses, resulting in unpredictable blood pressure and heart rate variations. This physiological reaction peaks immediately post-procedure (≤30 sec) and typically resolves within 10 minutes.[11] In vulnerable patients, such as those with hypertension, coronary artery disease, or cerebrovascular disease, these hemodynamic alterations may have serious consequences, including ischemia, arrhythmias, cerebrovascular stroke, pulmonary edema, and increased intracranial pressure. [5,12,13]

To date, numerous drugs and various routes have been tried to attenuate this stress response such as opioids, vasodilators, beta-blockers, calcium channel blockers, intravenous lignocaine, topical sprays, volatile agents, and α2 agonists but none of the agents proved to be ideal.[14] Although many reports claim various drugs effectively attenuate hemodynamic responses, they also mention unexpected side effects like hypoventilation, hypotension, bradycardia, and muscle rigidity. [15]

Dexmedetomidine's highly-selective agonistic action on presynaptic α2-adrenergic receptors and subsequent inhibition of norepinephrine release from the locus coeruleus has been hypothesized as the most putative mechanism for its hemodynamic stress response attenuating action [16] Dexmedetomidine has sympatholytic, sedative, amnestic, and analgesic properties. Its pleiotropic effects have led to its increasing use for reducing anesthetic and analgesic requirements in the perioperative period. [17] The efficacy of dexmedetomidine in decreasing the hemodynamic response to laryngoscopy and intubation has been studied through intravenous [18-22], intranasal [23-24], and intramuscular routes. [25]

Furthermore, dexmedetomidine has been shown to significantly reduce the incidence of postoperative cognitive dysfunction (POCD) and improve the Mini-Mental State Examination (MMSE) score. [26] It is equally effective in all age groups and is being used in pediatric age as well as elderly patients.[27]

Various studies have investigated the effects of intravenous dexmedetomidine on the hemodynamic response to laryngoscopy and intubation. [12,14-18,28-30] While doses of 1–2 μg/kg are effective in attenuating this hemodynamic response, they are associated with significant side effects, such as bradycardia, hypotension, or respiratory depression. [12,29]

Neha et al. reported that both the loading doses of 1 μg/kg and 0.5 μg/kg Dexmedetomidine were equally effective in reducing the induction dose of propofol, improving the intubating conditions, and blunting the hemodynamic response to laryngoscopy and intubation. The incidence of adverse effects, such as hypotension and bradycardia, was lower with the loading dose of 0.5 μg/kg. [1]

In a previously conducted study by Lee CH et al. involving 42 patients, use of Dexmedetomidine showed lower MAP at 3 minutes (116.9 ± 16.2 vs 95.4

± 11.7) and at 5 minutes after intubation (103.9 ± 13.3 vs 83.4 ± 8.4) compared to Saline.[4]

Basar et al. [28] reported that 0.5 µg/kg dexmedetomidine decreased thiopental requirements without causing serious hemodynamic effects or affecting recovery time. Propofol is another favorable induction agent with a cardiovascular depressive property, and is more effective at suppressing stress hormone release than is thiopental. [35] Therefore, in the present study, we decided to administer 1.5-2.5mg/kg of Propofol for induction of anesthesia.

Lawrence and De Lange [29] found that a single dose of 2 µg/kg dexmedetomidine caused a higher incidence of bradycardia and hypotension compared with the placebo treatment. Similarly, Mahajan et al. [12] found that with the same depth of anesthesia, there was a significant fall in HR and SBP and DBP in the dexmedetomidine group (1 µg/kg) versus the placebo group, and that this effect lasted until 30 min following drug administration.

However, the aforementioned studies were conducted in young and normotensive individuals. Dose reduction is required in the elderly because of age associated pharmacodynamic changes, and some studies have reported more pronounced hemodynamic responses to drugs in patients with hypertension. [6,13,34]

Some studies indicate that supplemental administration of dexmedetomidine can effectively reduce early postoperative nausea and vomiting (PONV). [31]

Cabrini and colleagues [32] published the recent systematic review of randomized controlled trials that intravenous sedation with dexmedetomidine alone resulted in safety and a few adverse events in awake fiberoptic intubation.

Keniya et al. [33] reported that patients administered 1 µg/kg dexmedetomidine required more treatment for bradycardia than the controls did. In contrast, Scheinin et al. [21] reported that 0.6 µg/kg dexmedetomidine attenuated cardiovascular responses to laryngoscopy and tracheal intubation in healthy individuals without serious side effects.

Additionally, some research has shown severe hypotensive episodes requiring vasoconstrictor treatment after general anesthesia induction in patients chronically using angiotensin II antagonists.[36] Although none of our patients experienced a hypotensive episode, additional study of the hemodynamic responses to different antihypertensive drugs and alpha-2 agonists, and the use of thiopentone for anesthesia induction, may be needed.

In conclusion, this study shows that the preoperative administration of 0.5 µg/kg dexmedetomidine before anesthesia induction effectively suppresses the hemodynamic changes caused by endotracheal intubation in elderly patients (≥ 65 years old) undergoing treatment for hypertension, without causing any severe side effects. All patients in the study group responded very well to the stress induced by endotracheal intubation and the drug provided a stable hemodynamic state

CONFLICT OF INTEREST:

There is no conflict of interest

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Table 01: Comparison of demographic and clinical findings between study groups

	Study Group	
	C	D
Age (years)	68.9±3.74	67.9±3.23
Height (cm)	164.5±4.15	162.4±6.32
Weight (kg)	69.9±7.71	69.1±10.2
BMI (kg/m²)	25.8±2.49	26.1±2.80
Gender		
Male	18(60)	18(60)
Female	12(40)	12(40)
Comorbid		
Asthma	2(6.7)	4(13.3)
BPH	3(10)	2(6.7)
Breast Carcinoma	4(13.3)	3(10)
Diabetes	13(43.3)	14(46.7)
Hyperthyroidism	0(0)	1(3.3)
Hypothyroid	0(0)	1(3.3)
None	8(26.7)	5(16.7)

Values are mean ± SD or number of patients. No significant intergroup difference was found.
Group C: normal saline, Group D: Dexmedetomidine.

Table 02: Mean Comparison of clinical findings between study groups

	Study Group		Effect size	P-value
	C	D		
Baseline				
SBP	145.4±13.75	136.8±13.53	0.633	0.017*
DBP	83.37±13.50	76.10±8.31	0.648	0.015*
MAP	105.1±12.79	97.13±10.86	0.677	0.011*
HR	79.20±11.32	80.60±15.26	-0.104	0.688
After Drug				
SBP	151.±13.37	124.8±14.97	1.893	<0.001*
DBP	88.47±12.09	70.00±8.07	1.796	<0.001*
MAP	110.57±11.27	89.00±8.84	2.129	<0.001*
HR	78.60±11.30	76.47±6.59	0.231	0.376
1 min after intubation				
SBP	186.5±14.69	136.5±25.04	2.439	<0.001*
DBP	105.2±12.65	81.03±16.36	1.652	<0.001*
MAP	133.2±12.68	98.87±17.67	2.235	<0.001*
HR	102.3±11.59	86.37±9.21	1.528	<0.001*
3 min after intubation				
SBP	169.23±12.30	122.4±20.21	2.797	<0.001*
DBP	91.20±8.52	71.00±11.03	2.049	<0.001*
MAP	117.8±9.94	86.90±10.80	2.976	<0.001*
HR	87.43±11.67	77.97±7.98	0.947	0.001*
5 min after intubation				
SBP	147.3±11.17	112.07±17.22	2.430	<0.001*

DBP	80.23±8.52	64.67±7.64	1.923	<0.001*
MAP	102.50±10.16	81.07±9.99	2.127	<0.001*
HR	77.53±17.00	72.60±8.14	0.370	0.157

Independent t-test was applied. Values are expressed as mean ± SD. *Statistically significant (P < 0.05). Group C: normal saline, Group D: dexmedetomidine. SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, HR: heart rate.

Table 03: Mean changes in hemodynamic parameters from baseline at various time points following drug administration within groups.

	Study Group			
	C		D	
	Mean difference	p-value	Mean difference	p-value
Systolic Blood Pressure				
Before vs After drug	-6.26±12.6	0.011*	11.9±20.8	0.004*
Before vs 1min after intubation	-41.1±22.8	<0.001*	0.30±29.4	0.956
Before vs 3min after intubation	-23.8±21.8	<0.001*	14.36±23.5	0.002*
Before vs 5min after intubation	-1.90±14.7	0.487	24.7±19.8	<0.001*
Diastolic Blood Pressure				
Before vs After drug	-5.10±17.9	0.130	6.10±11.1	0.006*
Before vs 1min after intubation	-21.8±17.0	<0.001*	-4.93±19.2	0.170
Before vs 3min after intubation	-7.83±15.7	0.011*	5.10±14.0	0.056
Before vs 5min after intubation	3.13±11.7	0.155	11.43±11.2	<0.001*
Mean Arterial Pressure				
Before vs After drug	-5.40±13.1	0.032*	8.13±12.8	0.002*
Before vs 1min after intubation	-28.0±16.4	<0.001*	-1.73±22.5	0.676
Before vs 3min after intubation	-12.6±15.3	<0.001*	10.2±14.8	<0.001*
Before vs 5min after intubation	2.66±12.47	0.251	16.0±14.4	<0.001*
Heart Rate				
Before vs After drug	0.60±2.31	0.166	4.13±13.9	0.114
Before vs 1min after intubation	-23.1±7.79	<0.001	-5.76±18.06	0.091
Before vs 3min after intubation	-8.23±5.45	<0.001	2.63±14.4	0.327
Before vs 5min after intubation	1.66±12.6	0.477	8.00±11.13	<0.001*

Paired t-test was applied
*p≤0.05, statistically significant

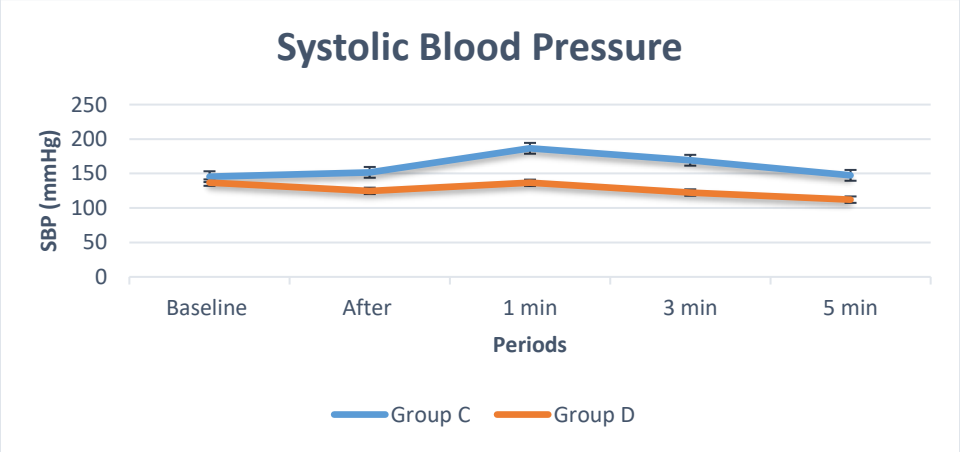

Fig. 2. Changes in systolic blood pressure (SBP) in the 2 groups. Group C: normal saline, Group D: dexmedetomidine.

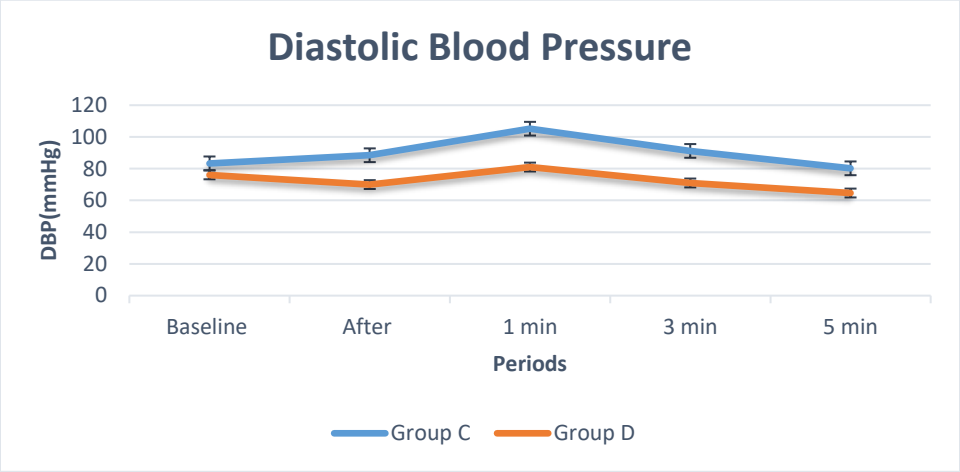

Fig. 3. Changes in diastolic blood pressure (DBP) in the 2 groups. Group C: normal saline, Group D: dexmedetomidine.

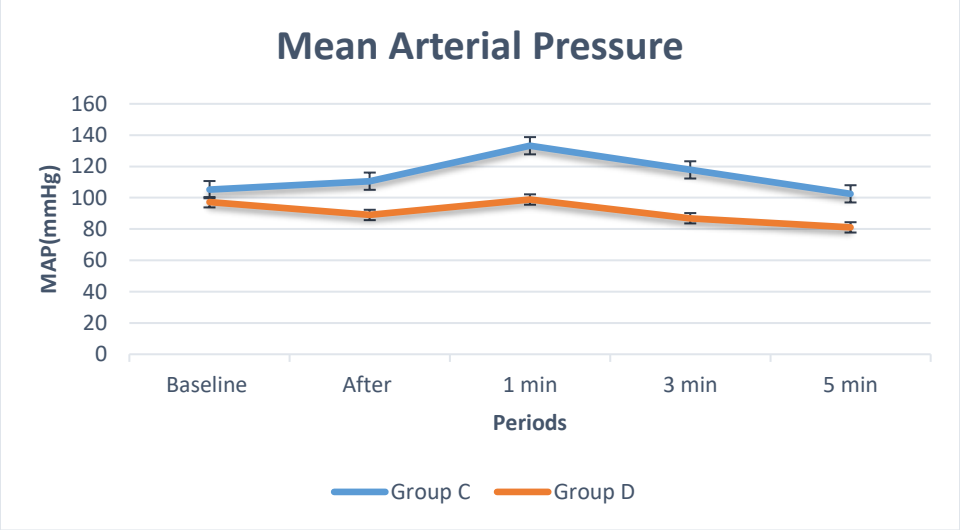

Fig. 4. Changes in mean arterial pressure (MAP) in the 2 groups. Group C: normal saline, Group D: dexmedetomidine.

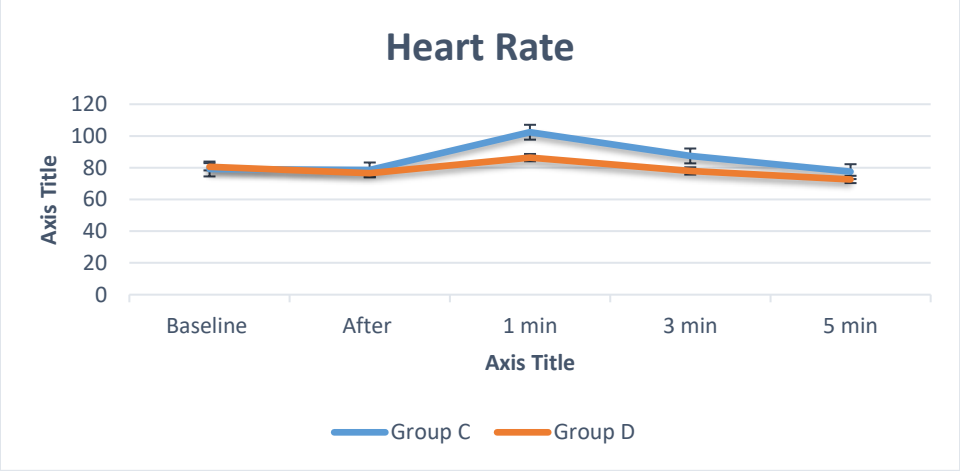

Fig. 5. Changes in heart rate (HR) in the 2 groups. Group C: normal saline, Group D: dexmedetomidine.

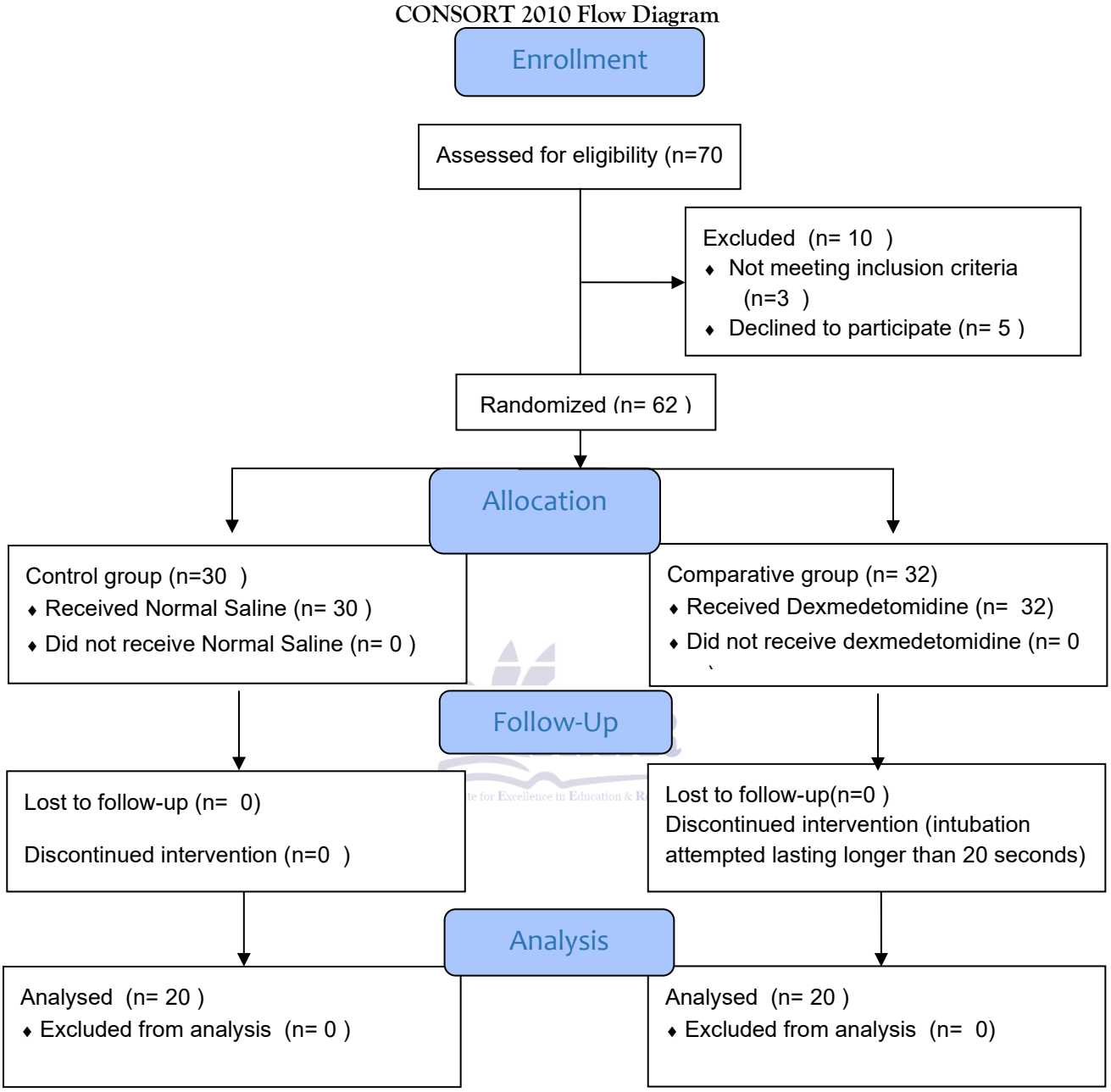

Fig. 1. Flow chart of the study. Seventy patients were recruited, and 3 obese patients (body mass index > 35 kg/m2) were excluded. Randomization was done on 62 patients, with 30 in the control group and 22 in the dexmedetomidine group. Intervention was discontinued in 2 patients in the dexmedetomidine group because the intubation attempt lasted more than 20 s