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# Effect of Dexmedetomidine Infusion on Hemodynamic Response During Laparoscopic Cholecystectomy

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**KEYWORDS**

Dexmedetomidine; Hemodynamic responses; Laparoscopic cholecystectomy; Post-operative pain; General anesthesia

**ABSTRACT:**

**Introduction:** Laparoscopic cholecystectomy is a minimally invasive procedure associated with significant hemodynamic changes, primarily due to pneumoperitoneum. This stress response often elevates mean arterial pressure (MAP) and heart rate (HR), posing challenges for patients with cardiovascular conditions. Dexmedetomidine, an alpha-2 adrenergic agonist, has shown potential in stabilizing hemodynamics through its sedative, anxiolytic, and sympatholytic effects. This study aims to compare the efficacy of a dexmedetomidine loading dose followed by continuous infusion versus only a loading dose in maintaining hemodynamic stability during laparoscopic cholecystectomy.

**Methodology:** A prospective, randomized, double-blind study was conducted with 120 ASA Grade I and II patients undergoing laparoscopic cholecystectomy from October 2019 to March 2021. Participants were divided into two groups: Group D received a 1 µg/kg loading dose of dexmedetomidine followed by a continuous infusion of 0.2 µg/kg/hr, while Group NS received the same loading dose followed by a placebo. Hemodynamic parameters (HR and MAP) were monitored throughout the procedure. Postoperative pain and sedation were assessed using Visual Analogue Scale (VAS) and Ramsay Sedation Score (RSS).

**Results:** Group D showed significantly lower HR and MAP compared to Group NS, particularly after 40 and 60 minutes, respectively. Group D also demonstrated reduced postoperative analgesic requirements and better pain control. However, Group D had a higher incidence of bradycardia and hypotension, though no significant adverse effects like dry mouth were observed.

**Conclusion:** A dexmedetomidine regimen consisting of a 1 µg/kg loading dose followed by a continuous infusion of 0.2 µg/kg/hr is more effective in attenuating hemodynamic fluctuations and reducing postoperative analgesic needs compared to only a loading dose. This approach provides improved hemodynamic stability during laparoscopic cholecystectomy while maintaining acceptable sedation levels.

## 1. Introduction

Laparoscopic cholecystectomy, a minimally invasive procedure for gallbladder removal, has become the standard of care due to its advantages over open surgery, such as reduced postoperative pain, shorter hospital stays, and quicker recovery.[1-3] However, the procedure is associated with significant hemodynamic changes, primarily due to the

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pneumoperitoneum created by carbon dioxide insufflation and patient positioning.[4] These hemodynamic alterations include increases in mean arterial pressure (MAP), heart rate (HR), and systemic vascular resistance, which can be particularly challenging in patients with pre-existing cardiovascular conditions.[4-7]

Dexmedetomidine, an alpha-2 adrenergic agonist, has gained attention for its potential to modulate these hemodynamic responses. Its sedative, anxiolytic, and analgesic properties, combined with its ability to reduce sympathetic tone, make it a valuable adjunct in anesthesia management, particularly for procedures like laparoscopic cholecystectomy where stable hemodynamics are crucial.[8,9]

The use of dexmedetomidine in laparoscopic cholecystectomy has been shown to significantly improve hemodynamic stability. Studies indicate that patients receiving dexmedetomidine infusion exhibit lower MAP and HR compared to those who receive a placebo or other anesthetic agents. This effect is primarily due to the drug's ability to decrease norepinephrine release, leading to a reduction in sympathetic outflow. By attenuating the stress response associated with pneumoperitoneum and surgical stimuli, dexmedetomidine helps maintain hemodynamic parameters within a more stable range, reducing the risk of perioperative complications.[8-10]

When compared to other agents like clonidine,[8,11] esmolol,[12,13] or opioids,[9] dexmedetomidine[1,10] demonstrates superior efficacy in controlling hemodynamic fluctuations. While clonidine, another alpha-2 agonist, also provides hemodynamic stability, dexmedetomidine offers additional benefits such as sedation without significant respiratory depression.[8,11] Esmolol, a beta-blocker, can effectively blunt the heart rate response, but it may not provide the same level of MAP control as dexmedetomidine.[18,19] Moreover, opioids, while useful for analgesia, do not have the same degree of impact on hemodynamic stability and may cause adverse effects like respiratory depression.

The use of dexmedetomidine not only improves intraoperative hemodynamic stability but also has favorable effects on postoperative recovery. Patients receiving dexmedetomidine tend to have smoother emergence from anesthesia, lower postoperative pain scores, and reduced need for analgesics. Additionally, the sedative properties of dexmedetomidine contribute to a more relaxed state, reducing the incidence of postoperative agitation or delirium, which is particularly beneficial in elderly patients or those with pre-existing cognitive dysfunction.[14]

## 2. Objectives

Primary aim

- To compare the effect of a loading dose of dexmedetomidine followed by infusion,with that of only a loading dose of dexmedetomidine,on hemodynamic response to pneumoperitoneum during laparoscopic cholecystectomy.

Secondary aim

- To study the side effects of dexmedetomidine
- To study the requirement of post operative analgesia
- To study the post operative sedation score

## 3. Methods

This prospective, randomized, double-blind, comparative study was conducted to evaluate the efficacy of dexmedetomidine in maintaining hemodynamic stability during laparoscopic cholecystectomy. The study was carried out at a tertiary hospital from October 2019 to March 2021, following approval from the ethical committee. Informed consent was obtained from all participating patients.

A total of 120 patients classified as ASA Grade I and II, scheduled for laparoscopic cholecystectomy, were included in the study. The participants were randomly divided into two groups of 60 patients each using a computer-generated randomization technique.

**Inclusion criteria** encompassed patients aged 18-65 years, of either sex, weighing 30-70 kg, and undergoing laparoscopic surgery.

**Exclusion criteria** included allergy or intolerance to the study drugs, preoperative heart rate below 45 bpm, use of antihypertensive medications involving α-2 adrenergic agonists or beta-blockers, renal or hepatic insufficiency, and pregnancy or lactation.

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All participants underwent a thorough pre-anesthetic evaluation, which included assessments of their general condition, nutritional status, cardiovascular and respiratory systems, associated diseases, and airway status. Routine investigations such as hemoglobin levels, complete blood count, renal function tests, ECG, chest X-ray (if indicated), and fasting blood sugar levels (for diabetics) were performed. Patients were instructed on using a 10 cm Visual Analogue Scale (VAS) to assess pain levels.

On the day of surgery, patients were kept nil by mouth for six hours for solid food and two hours for clear liquids. Upon arrival in the operating room, an 18 or 20 gauge intravenous cannula was inserted, and IV fluids were administered. Patients were connected to a multi-parameter monitor displaying heart rate (HR), non-invasive blood pressure (NIBP), end-tidal carbon dioxide (EtCO2), arterial oxygen saturation, and continuous ECG monitoring.

Premedication included IV ondansetron (0.1 mg/kg), IV glycopyrrolate (0.01 mg/kg), IV midazolam (0.05 mg/kg), and IV fentanyl (2 µg/kg). Preoxygenation was provided with 100% oxygen. General anesthesia was induced with IV propofol (2 mg/kg), and intubation was facilitated by IV atracurium (0.5 mg/kg). Anesthesia was maintained with oxygen, air, IV atracurium (0.1 mg/kg), and isoflurane using a circle absorber system. The patients were mechanically ventilated, and intra-abdominal pressure was maintained between 12 and 14 mmHg throughout the procedure.

The patients were randomly assigned to one of two groups:

**Group D**: Received an IV dexmedetomidine loading dose of 1 µg/kg over 10 minutes before pneumoperitoneum, followed by a continuous infusion of dexmedetomidine at 0.2µg/kg/hr.

**Group NS**: Received an IV dexmedetomidine loading dose of 1 µg/kg over 10 minutes before pneumoperitoneum, followed by a placebo infusion of normal saline.

The preparation of the study drug was blinded, with dexmedetomidine diluted in normal saline to a final concentration of 4 µg/ml for Group D, and normal saline alone for Group NS. Any fall in heart rate below 60 bpm was treated as bradycardia with IV atropine, and hypotension (defined as a blood pressure drop of more than 20% from baseline) was initially treated with a bolus of ringer lactate and, if necessary, IV ephedrine.

During the surgery, HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at multiple intervals, including pre-induction, post-induction, after the dexmedetomidine loading dose, and every 10 minutes during surgery. Postoperative monitoring continued, with sedation assessed using the Ramsay Sedation Score (RSS) and pain using the VAS. Rescue analgesia was provided if VAS >4. Patients were monitored for adverse effects, such as bradycardia and hypotension, as well as postoperative symptoms like nausea and dry mouth.

## 4. Results

The study compared the demographic, physiological, and clinical characteristics between two patient groups, Group D and Group NS, across various parameters.

Table 1 shows that age wise distribution showed a higher proportion of younger patients (<50 years) in Group D, while Group NS had a higher proportion of older patients (51-60 years) but these differences were not statistically significant (p>0.05). There were slightly more females Group NS, and more males were in Group D with no statistically significant between the groups (p>0.05). Group D had more patients <50 kg, while Group NS had more patients in the 51-60 kg range but the differences were insignificant statistically (p>0.05). Group D had slightly more patients in ASA 1, while Group NS had slightly more in ASA 2 with no significant difference between the groups.

Table 2 shows that across various time points, heart rate was generally higher in Group NS. However, these differences were not statistically significant until 30 minutes after administration of anaesthesia. At later time points significant differences with lower HR in Group D were noted with p-values < 0.001.(Figure 1)

Table 3 states that Group NS exhibited slightly higher initial systolic blood pressure (SBP) compared to Group D for 50 minutes post-administration of anaesthesia, this difference was not statistically significant (p>0.05). At later point of times, Group NS demonstrated a significantly higher SBP as compared to Group D with

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p-values indicating strong statistical significance ($p < 0.001$).(Figure 2)

According to table 4, Group D had a slightly higher initial diastolic blood pressure (DBP) than Group NS, though this difference was not statistically significant ($p>0.05$). After 20 minutes, Group NS exhibited significantly higher DBP with strong statistical significance ($p < 0.001$).Figure 3 depicts that Group D had slightly higher MAP for first 20 minutes but differences were not significant ($p >0.05$). After 20 minutes, Group NS had significantly higher MAP with p-values < 0.001 as compared to group D.

Table 5 shows that mean Ramsay Sedation Score of patients in group D was slightly higher with mean of 2.9±0.3, whilst in group NS it was 2.78±0.42 with statistically no significant difference in the sedation scores between the groups. Mean VAS Score of patients in Group D was 2.92±0.33 indicating better pain control as compared to Group NS with significantly higher mean value of 3.12±0.49 ($p<0.05$). Significantly fewer patients in Group D (1.7%) required rescue analgesia as compared to 11.7% patients in Group NS, suggesting that Group D provided more effective pain management.

Table 6 depicts that 23.3% of patients in Group D had hypotension as compared to 6.7% patients in Group NS and the difference was statistically significant ($p<0.05$). Similarly, it was observed that 45% patients in Group D had bradycardia, compared to 26.7% in Group NS with the difference being statistically significant ($p<0.05$). Nausea was experience by only 3.3% patients in group D as compared to 0% in group NS and the difference was not statistically significant ($p>0.05$). None of the patients in both the groups experienced dry mouth.

## 5. Discussion

This prospective, randomized, comparative, double-blind study was conducted at a tertiary hospital between October 2019 and March 2021. The primary aim was to evaluate the hemodynamic response to pneumoperitoneum during laparoscopic cholecystectomy in patients administered a loading dose of Dexmedetomidine, followed by either an infusion or no further dose. The study involved 120 ASA grade 1 and 2 patients, divided into two groups of 60 each: Group D (received Dexmedetomidine infusion) and Group NS (received placebo infusion). Both the groups were comparable and there was no statistically significant difference with regards to mean age, weight and sex. Patients over the age of 65 years were not included due to varied pharmacokinetic profile of Dexmedetomidine in this group of patients. An increased variationin HR is seen with increasing age. These patients are also known to develop excessive sedation and hypotension.[15] Also Patients on Beta- Blockers, Calcium channel blockers and those withPreoperative HR less than < 60bpm were excluded from the study as Dexmedetomidine is known to cause Bradycardia.

Dexmedetomidine, commonly used intravenously, typically follows a loading dose of 1 µg/kg, with an infusion rate of 0.2 to 0.7 µg/kg/hr. Studies have explored various dosing strategies to manage hemodynamic responses during surgeries like pneumoperitoneum. Study by Gourishankar Reddy Manne et al.[1] found that a 0.4 µg/kg/hr infusion was more effective than a 0.2 µg/kg/hr infusion for stabilizing hemodynamics without a loading dose. Vora et al.[16] used a 1 µg/kg bolus followed by a 0.5 µg/kg/hr infusion, achieving stable intraoperative hemodynamics but causing excessive postoperative sedation. J Choi et al.[17] administered a 0.5 µg/kg bolus after induction, improving intraoperative hemodynamics and postoperative outcomes. Qin Ye et al.[10] demonstrated that higher doses of dexmedetomidine (0.4 to 0.8 µg/kg) increased the risk of bradycardia. Bakshi et al.[18] showed that a 1 µg/kg loading dose followed by a 0.2 µg/kg/hr infusion significantly reduced the need for intraoperative fentanyl in robotic-assisted laparoscopic surgeries. Tufanogullari et al.[19] recommended a 0.2 µg/kg/hr infusion for laparoscopic bariatric surgery, minimizing cardiovascular side effects. However, no study has directly compared the hemodynamic responses to pneumoperitoneum using only a loading dose versus a loading dose followed by infusion, which is the focus of the present study. In our study, loading dose of dexmedetomidine 1µg/kg was administered over 10 mins after induction of anaesthesia and infusion of study drug was started from initiation of pneumoperitoneum upto its release to avoid confounding factors of laryngoscopy.

Both groups had comparable baseline HR, with no significant differences (Group D: 86.98 bpm, Group NS: 87.07 bpm). Following the administration of the loading dose, both groups experienced a significant decrease in HR (Group D: 19.12%, Group NS: 18.28%). However,

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the difference between the two groups was not statistically significant, indicating that the loading dose alone effectively reduced HR in both groups. HR was consistently lower in Group D compared to Group NS throughout the pneumoperitoneum, with a significant difference observed after 40 minutes. This suggests that the infusion in Group D provided better hemodynamic stability over time. Kataria et al.[9],showed a mean HR of 78.01± 7.16 bpm at baseline followed by HR of 72.53 ± 4.89 bpm after administration of Dexmedetomidine 1µg/kg bolus with a drop of 15bpm. Vora et al[16],also showed a significant drop in HR after a bolus dose of Dexmedetomidine. Aikarterni et al[20] noticed bradycardia and cardiac depression at a dose of 1 µg/ kg Dexmedetomidine infusion over 10 minutes. **Jayaram et al**[21] in their study observed that Dexmedetomidine in dose of with 1 µg/kg caused significant fall in HR (18 % from the baseline). In our study, mean HR was monitored every 10 minutes during pneumoperitoneum. Group D maintained a consistent HR, while Group NS experienced a gradual increase after 40 minutes. The HR difference at 40 minutes was statistically significant ($t = -4.367$, $p < 0.001$) and remained significant until the release of pneumoperitoneum. Similar findings were observed by Pyakurel et al.[22], where dexmedetomidine at 1 µg/kg provided better hemodynamic attenuation than 0.5 µg/kg for the first 40 minutes of pneumoperitoneum, with no significant attenuation afterward in either group. In our study, both groups had comparable baseline MAP, with no significant differences (Group D: 103.44 mmHg, Group NS: 103.22 mmHg). Similar to HR, both groups experienced a decrease in MAP after the loading dose, with Group D showing a slightly greater reduction. However, the difference between the groups was not statistically significant. Group D maintained a consistent MAP throughout the pneumoperitoneum, while Group NS showed a gradual rise after 60 minutes. The difference in MAP became statistically significant after 60 minutes, indicating that the infusion in Group D was more effective in maintaining stable blood pressure.Studies have shown a similar drop in MAP after a bolus dose of 1µg/kg/hr Dexmedetomidine.[23,24]

Ebert et al[25] studied the effects of increasing Plasma concentrations of Dexmedetomidine After administration of an IV bolus of Dexmedetomidine, a high (peak) plasma concentration is achieved, resulting in an increase in blood pressure combined with a marked decrease in HR. During this phase there is α2-receptor activation in the vascular smooth muscles, causing a marked increase in systemic vascular resistance and thereby hypertension. This is accompanied by a quick reductionin HR, presumably caused by the baroreceptor reflex. After a few minutes, when plasmaconcentrations of Dexmedetomidine decreases, the vasoconstriction attenuates, and since Dexmedetomidine also activates a2-receptors in the vascular endothelial cells, it results in vasodilatation together with presynaptic a2-adrenoreceptors inhibiting sympathetic release of catecholamine and increased vagal activity, this results in a hypotensive phase.

The mean MAP was monitored every 10 minutes during pneumoperitoneum. Group D exhibited consistent MAP, while Group NS showed a gradual increase after 60 minutes. The MAP difference at 60 minutes was statistically significant ($p<0.001$) and remained significant afterward. Pneumoperitoneum induces a stress response, elevating catecholamines like epinephrine and norepinephrine, which increase HR and blood pressure. IV dexmedetomidine helps mitigate this by inhibiting catecholamine release, thus maintaining hemodynamic stability. Studies show dexmedetomidine reduces plasma catecholamines by 60–80%, reflecting its effective sympatholytic effects.[25-27]

In our study, none of the patients had undue sedation. Dexmedetomidine is known to suppress noradrenergic neuronal firing of the locus coeruleus in the brain stem[28], which leads to a loss of wakefulness via activation of an endogenous sleep-promoting pathway.[29] Although patient cooperation can be achieved using other sedatives, with careful dose titration, dexmedetomidine may promote cooperative sedation more easily within the recommended dosage range.

The sedation caused by dexmedetomidine is mainly does dependent.[30]The dexmedetomidinehas similar properties of clonidine but with more affinity toward its receptor and absence of respiratory depression.[31] Hall et al[32] found that small doses of dexmedetomidine provided significant sedation that could be easily reversed with verbal or physical stimuli, and it resolved 2 hr after terminating the infusion.

Although the trauma of laparoscopic surgeries is small, postoperative pain is still the main reason that affects

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postoperative recovery and prolongs hospital stay. Previous studies have shown that dexmedetomidine could effectively relieve postoperative pain and improve the quality of postoperative recovery.[33,34] Because dexmedetomidine reduced inflammatory mediators and substance P caused by surgical trauma.[15,33] A meta-analysis showed that dexmedetomidine could relieve postoperative pain and reduce the dosage of postoperative analgesic, but the optimal dose of dexmedetomidine needs further study.[35] In our study, we found that intravenous infusion of dexmedetomidine reduced VAS scores and postoperative analgesic requirements, with significant difference between the two groups.

The mechanism of the analgesic effects of Dexmedetomidine are still not fully understood. It is usually known for its analgesic sparing effect. It acts in 2 major ways: a) preventing a nervefrom firing in the first place, and b) inhibiting the propagation of pain signals. At the level of the substantia gelatinosa of the spinal cord it acts on α-2A receptors and prevents the release of the neurotransmitters like Substance P.[36] At the level of the brain stem, it acts on the α-2 receptors within the locus coeruleus and inhibits firing of theses neurons. The locus coeruleus is the site of origin for the descending medullospinal noradrenergic pathway, an important modulator of nociception.[37]

In our study the adverse effects studied were bradycardia, hypertension, nausea & dry mouth. It was observed that 45% patients in Group D had bradycardia, compared to 26.7% in Group NS. The number of patients having bradycardia were significantly more in group D compared to Group NS. In both the groups most patients had bradycardia, after the loading dose of dexmedetomidine was given, however the HR did not drop below 50 bpm and did not require administration of atropine. As already discussed, bradycardia associated with administration of IV dexmedetomidine was explained in 2 phases. Phase 1, which is seen in young, healthy patients with high vagal tone, is due to vagal mediated reflex bradycardia in response to initially induced hypertension. Phase 2, occurred due to centrally mediated inhibition of sympathetic outflow. Cardiac arrest has been reported, after IV administration of dexmedetomidine, which has resolved after resuscitative efforts.[20,38-41] However, not all the cases should be attributed to dexmedetomidine alone, most cases had other significant comorbidities. Sinus arrests have been seen in young healthy patients as well.

It was observed that significantly higher number of patients (23.3%) in Group D had hypotension compared to 6.70% in Group NS. Dexmedetomidine is known to cause bradycardia and hypotension due to its α-2 agonism.[37,42,43] Adverse events associated with dexmedetomidine occur most frequently during or shortly after a loading dose.[44] Various methods, albeit with controversial results, have been evaluated to moderate hypotension and bradycardia associated with dexmedetomidine administration, either by omitting[45,46],decreasing[47,48] or slowing the loading infusion.

In our study, 3.3% of patients in Group D had nausea compared to none in Group NS. The incidence of nausea and vomiting after general anaesthesia has been reported to be as high as 24% and after laparoscopy it is as high as 42% due to rapid peritoneal distension[16].The etiology of PONV after laparoscopic surgery is not fully understood, and still a major drawbackof this type of surgery. Multiple factors are associated with an increased incidence of PONV, including patient, anaesthetic and surgical factors.

In a study conducted by Bakri et al[49] it showed that the incidence of nausea and vomiting significantly decreases with dexmedetomidine as effectively as dexamethasone in laparoscopic cholecystectomy. This decrease in incidence of nausea and vomiting could be due to opioid sparing effect and lesser requirement of inhaled anaesthetics[50.51]. Islam Massad et al[52] concluded that combining dexmedetomidine with other anaesthetic agents, results in more balanced anaesthesia and a significant drop in the incidence of PONV, as well as reduces the requirements of postoperative rescue anti-emetic medications after laparoscopic gynaecological surgeries.[52,53] Other theories states that dexmedetomidine decreases noradrenergic activity by acting on α2 presynaptic inhibitory adreno-receptors in the locus coeruleus, which is a major noradrenergic cell group located in the pontine brain stem and appears to have regulatory effects on extracellular dopamine. The alpha-2 adrenoreceptor agonist of dexmedetomidine, which resembles that of clonidine, decreases the noradrenergic activity as a result of binding to alpha-2 presynaptic inhibitory adrenoreceptor in the locus

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coeruleus, an inhibition that possibly results in an anti-emetic effect.

Also, it is well-known that the gastrointestinal distension stimulates vagal visceral afferents; this in turn activates the vomiting center and induces nausea and vomiting. By increasing sympathetic outflow and decreasing parasympathetic outflow from the central nervous system, dexmedetomidine may exert its effect by increasing the gastric emptying and the gastrointestinal motility, which possibly has an important effect in decreasing nausea and vomiting.

In our study, none of the patients in both the groups had dry mouth. Dexmedetomidine activates peripheral presynaptic α2-adrenergic receptor which reduces the release of catecholamines, and hence reduces sympathetic response to surgery[54].The results are consistent with the study conducted by **Parikh et al.**[55]

## 6. Conclusion:

Our study concludes that administering dexmedetomidine as a 1 µg/kg loading dose followed by a continuous infusion of 0.2 µg/kg/hr more effectively attenuates the pressor response associated with pneumoperitoneum during laparoscopic cholecystectomy compared to using only a 1 µg/kg loading dose. This regimen also reduces the need for postoperative analgesia without causing undue sedation.

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**TABLE 1: DEMOGRAPHIC DETAILS OF STUDY PARTICIPANTS:**

| PARAMETER | | GROUP D | | GROUP NS | | p VALUE |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age in years | <20 | 2 | 3.3% | 1 | 1.7% | 0.321 NS |
| | 21-30 | 12 | 20% | 6 | 10% | |
| | 31-40 | 21 | 35% | 17 | 28.3% | |
| | 41-50 | 15 | 25% | 14 | 23.3% | |
| | 51-60 | 8 | 13.3% | 21 | 35% | |
| | >60 | 2 | 3.3% | 1 | 1.7% | |
| Gender | Female | 34 | 56.7% | 35 | 58.3% | 0.853 NS |
| | Male | 26 | 43.3% | 25 | 41.7% | |
| Weight in Kg | <50 | 8 | 13.3% | 4 | 6.7% | 0.321 NS |
| | 51-60 | 31 | 51.7% | 38 | 63.3% | |
| | 61-70 | 21 | 35% | 18 | 30% | |
| ASA | 1 | 39 | 65% | 34 | 56.7% | 0.350 NS |
| | 2 | 21 | 35% | 26 | 43.3% | |

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**TABLE 2: GROUP WISE COMPARISON OF HEART RATE:**

| PARAMETER | GROUP D | | GROUP NS | | t VALUE | p VALUE |
|---|---|---|---|---|---|---|
| | N | Mean ± SD | N | Mean ± SD | | |
| Pre-Induction HR ($t_i$) | 60 | 86.98±10.88 | 60 | 87.07±7.46 | -0.049 | 0.961 |
| HR before start of Dexmedetomidine($t_{bd}$) | 60 | 80.27±7.58 | 60 | 80.65±5.22 | -0.323 | 0.748 |
| HR after loading dose of Dexmedetomidine ($t_{ad}$) | 60 | 64.92±5.55 | 60 | 65.9±4.79 | -1.039 | 0.301 |
| At 10mins HR($t_{10}$) | 60 | 64.18±4.37 | 60 | 64±4 | 0.24 | 0.811 |
| At 20mins HR($t_{20}$) | 60 | 63.93±3.44 | 60 | 65.1±3.2 | -1.923 | 0.057 |
| At 30mins HR($t_{30}$) | 60 | 64.98±2.9 | 60 | 64.7±3.08 | 0.519 | 0.605 |
| At 40mins HR($t_{40}$) | 60 | 63.85±2.49 | 60 | 66.05±3.01 | -4.367 | <0.001 |
| At 50mins HR($t_{50}$) | 60 | 63.8±2.51 | 60 | 65.85±3.16 | -3.934 | <0.001 |
| At 60mins HR($t_{60}$) | 60 | 65.43±1.86 | 60 | 66.98±2.94 | -3.452 | 0.001 |
| At 70mins HR($t_{70}$) | 60 | 65.63±2.05 | 60 | 67.62±2.82 | -4.41 | <0.001 |
| At 80mins HR($t_{80}$) | 60 | 63.37±2.82 | 60 | 68.53±2.93 | -9.835 | <0.001 |
| At 90mins HR($t_{90}$) | 52 | 64.1±3.16 | 49 | 69.67±2.84 | -9.315 | <0.001 |
| At 100mins HR($t_{100}$) | 17 | 64.65±3.18 | 16 | 68.75±3.02 | -3.794 | 0.001 |
| At 110mins HR($t_{110}$) | 9 | 63.89±3.62 | 10 | 69.5±1.51 | -4.323 | 0.001 |
| At the end of Surgery HR($t_e$) | 60 | 74.07±5.31 | 60 | 80.1±3.25 | -7.506 | <0.001 |
| Post Extubation HR ($t_{pe}$) | 60 | 94.98±5.86 | 60 | 100.15±5.68 | -4.901 | <0.001 |
| 30mins Post ExtubationHR ($t_r$) | 60 | 84.8±5.5 | 60 | 88.62±4.57 | -4.138 | <0.001 |

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**TABLE 3: GROUP WISE COMPARISON OF SYSTOLIC BLOOD PRESSURE:**

| PARAMETER | GROUP D | | GROUP NS | | t VALUE | p VALUE |
|---|---|---|---|---|---|---|
| | N | Mean ± sd | N | Mean ± sd | | |
| Pre-Induction SBP ($t_i$) | 60 | 140.05±13.61 | 60 | 141.35±11.14 | -0.573 | 0.568 |
| SBP Before Start of Dexmedetomidine ($t_{bd}$) | 60 | 122.37±12.36 | 60 | 123.65±8 | -0.675 | 0.501 |
| SBP After Bolus of Dexmedetomidine($t_{ad}$) | 60 | 106.7±11.47 | 60 | 107.07±5.33 | -0.225 | 0.823 |
| At 10mins SBP($t_{1o}$) | 60 | 105.75±10.18 | 60 | 106.07±4.67 | -0.219 | 0.827 |
| At 20mins SBP($t_{20}$) | 60 | 106.1±10.31 | 60 | 105.12±4.76 | 0.671 | 0.504 |
| At 30mins SBP($t_{30}$) | 60 | 104.93±9.97 | 60 | 106.85±4.44 | -1.36 | 0.178 |
| At 40mins SBP($t_{40}$) | 60 | 106.88±9.82 | 60 | 107.47±4.3 | -0.422 | 0.674 |
| At 50mins SBP($t_{50}$) | 60 | 107.93±7.92 | 60 | 108.87±4.92 | -0.776 | 0.44 |
| At 60mins SBP($t_{60}$) | 60 | 106.9±7.29 | 60 | 112.48±4.32 | -5.103 | <0.001 |
| At 70mins SBP($t_{70}$) | 60 | 107.07±7.38 | 60 | 112.93±4.8 | -5.159 | <0.001 |
| At 80mins SBP($t_{80}$) | 60 | 106.88±7.71 | 60 | 114.43±4.59 | -6.519 | <0.001 |
| At 90mins SBP($t_{90}$) | 52 | 107.63±8.24 | 49 | 116.24±4.68 | -6.505 | <0.001 |
| At 100mins SBP($t_{100}$) | 17 | 108.41±8.17 | 16 | 115.06±4.97 | -2.802 | 0.009 |
| At 110mins SBP($t_{110}$) | 9 | 109.11±5.6 | 10 | 116.8±5.61 | -2.984 | 0.008 |
| At the end of Surgery SBP ($t_e$) | 60 | 116.07±7.09 | 60 | 125.95±4.8 | -8.944 | <0.001 |
| Post Extubation SBP ($t_{pe}$) | 60 | 145.75±7.33 | 60 | 153.15±9.3 | -4.842 | <0.001 |
| 30mins Post Extubation SBP ($t_r$) | 60 | 126.22±9.34 | 60 | 129.23±4.93 | -2.213 | 0.029 |

**TABLE 4: GROUP WISE COMPARISON OF DIASTOLIC BLOOD PRESSURE:**

| PARAMETER | GROUP D | | GROUP NS | | t VALUE | p VALUE |
|---|---|---|---|---|---|---|
| | N | Mean ± sd | N | Mean ± sd | | |
| Pre-Induction DBP ($t_i$) | 60 | 85.13±7.91 | 60 | 84.15±7.11 | 0.716 | 0.475 |
| DBP Before Start of Dexmedetomidine ($t_{bd}$) | 60 | 78.52±7.19 | 60 | 77.5±5.97 | 0.843 | 0.401 |
| DBP After Bolus of Dexmedetomidine($t_{ad}$) | 60 | 67.93±6 | 60 | 68.33±5.46 | -0.382 | 0.703 |
| At 10mins DBP($t_{10}$) | 60 | 66.32±6.14 | 60 | 67.45±4.68 | -1.138 | 0.258 |

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| | | | | | | |
|---|---|---|---|---|---|---|
| At 20mins DBP($t_{20}$) | 60 | 65.63±5.83 | 60 | 68.13±4.36 | -2.663 | 0.009 |
| At 30mins DBP($t_{30}$) | 60 | 64.73±5.01 | 60 | 69.18±4.47 | -5.137 | <0.001 |
| At 40mins DBP($t_{40}$) | 60 | 65.77±4.75 | 60 | 70.13±4.32 | -5.269 | <0.001 |
| At 50mins DBP($t_{50}$) | 60 | 66.08±4.53 | 60 | 68.85±4.1 | -3.508 | 0.001 |
| At 60mins DBP($t_{60}$) | 60 | 65.67±4.24 | 60 | 69.55±3.4 | -5.532 | <0.001 |
| At 70mins DBP($t_{70}$) | 60 | 65.58±4.2 | 60 | 69.78±3.16 | -6.196 | <0.001 |
| At 80mins DBP($t_{80}$) | 60 | 64.95±3.99 | 60 | 70.25±2.93 | -8.287 | <0.001 |
| At 90mins DBP($t_{90}$) | 52 | 65.42±3.17 | 49 | 71.43±2.81 | -10.056 | <0.001 |
| At 100mins DBP($t_{100}$) | 17 | 65.41±2.74 | 16 | 71.44±2.97 | -6.067 | <0.001 |
| At 110mins DBP($t_{110}$) | 9 | 64.89±2.76 | 10 | 72.8±3.01 | -5.947 | <0.001 |
| At the end of Surgery DBP ($t_e$) | 60 | 71.82±3.41 | 60 | 79.02±3.43 | -11.532 | <0.001 |
| Post Extubation DBP ($t_{pe}$) | 60 | 95.9±6.72 | 60 | 98.37±3.38 | -2.54 | 0.013 |
| 30mins Post Extubation DBP ($t_r$) | 60 | 80.5±5.59 | 60 | 83±3.87 | -2.85 | 0.005 |

**TABLE 5: COMPARISON OF MEAN RAMSAY SEDATION SCORES AND MEAN VAS SCORES BETWEEN 2 GROUPS:**

| **PARAMETER** | | | **GROUP D** | **GROUP NS** | **p VALUE** |
|---|---|---|---|---|---|
| **Ramsay Sedation Score** | | Mean | 2.9 | 2.78 | 0.082NS |
| | | SD | 0.3 | 0.42 | |
| **Visual Analogue scale** | | Mean | 2.92 | 3.12 | 0.01* |
| | | SD | 0.33 | 0.49 | |
| **Rescue analgesia given** | **No** | No of Patients | 59 | 53 | 0.028* |
| | | % withingoup | 98.3% | 88.3% | |
| | **Yes** | No of Patients | 1 | 7 | |
| | | % withingoup | 1.7% | 11.7% | |

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**TABLE 6: COMPARISON OF SIDE EFFECTS AND RESCUE ANALGESIA GIVEN BETWEEN TWO GROUPS:**

| PARAMETER | | | GROUP | | TOTAL | p value |
|---|---|---|---|---|---|---|
| | | | D | NS | | |
| BRADYCARDIA | No | No of Patients | 33 | 44 | 77 | 0.036* |
| | | % within group | 55.0% | 73.3% | 64.2% | |
| | Yes | No of Patients | 27 | 16 | 43 | |
| | | % within group | 45.0% | 26.7% | 35.8% | |
| HYPOTENSION | No | No of Patients | 46 | 56 | 102 | 0.011* |
| | | % within Group | 76.7% | 93.3% | 85.0% | |
| | Yes | No of Patients | 14 | 4 | 18 | |
| | | % within Group | 23.3% | 6.7% | 15.0% | |
| NAUSEA | No | No of Patients | 58 | 60 | 118 | 0.154NS |
| | | % within Group | 96.7% | 100.0% | 98.3% | |
| | Yes | No of Patients | 2 | 0 | 2 | |
| | | % within Group | 3.3% | 0.0% | 1.7% | |
| DRY MOUTH | No | No of Patients | 60 | 60 | 120 | Cannot be computed |
| | | % within Group | 100.0% | 100.0% | 100.0% | |
| | Yes | No of Patients | 0 | 0 | 0 | |
| | | % within Group | 0% | 0% | 0% | |

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**FIGURE 1: SHOWS DISTRIBUTION OF HR AT VARIED INTERVALS**

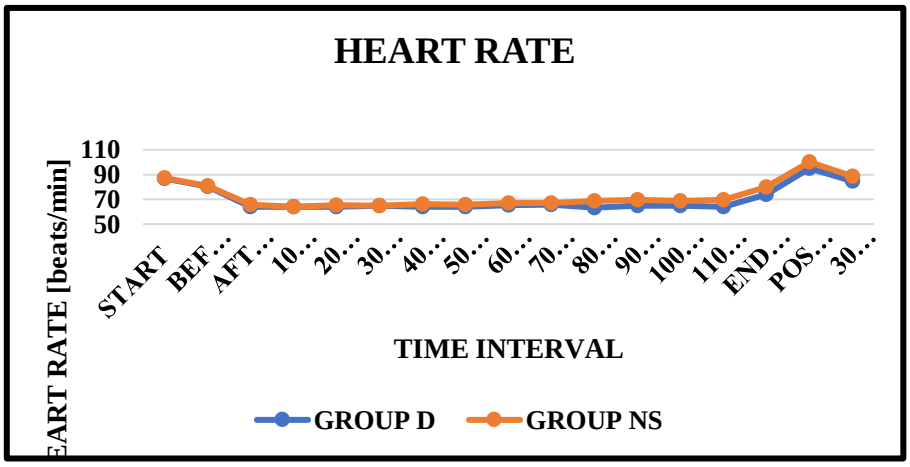


**FIGURE 2: SHOWS DISTRIBUTION OF SBP AT VARIED INTERVALS**

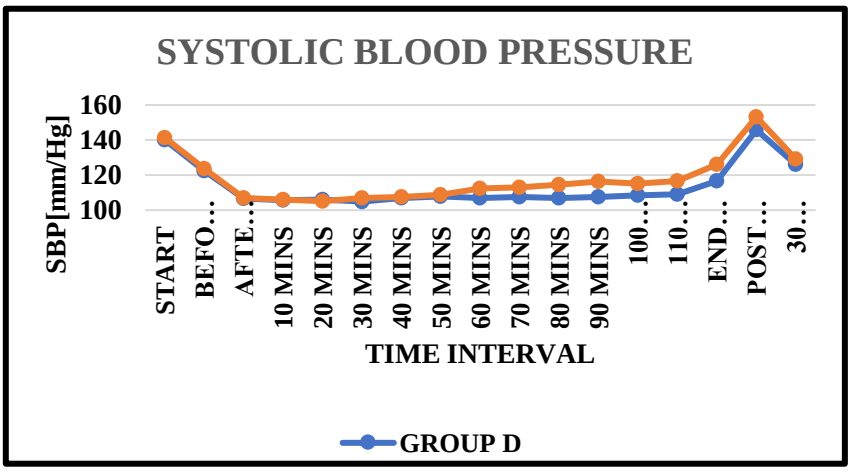


**FIGURE 3: SHOWS DISTRIBUTION OF DBP AT VARIED INTERVALS**