

Effect of Dexmedetomidine on Perioperative Haemodynamic Fluctuations in Untreated Stage 1 Hypertensive Patients Undergoing Laparoscopic Cholecystectomy- A Randomised Controlled Trial

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ABSTRACT

Introduction: Eighth Joint National Committee (JNC 8) has reclassified earlier prehypertension {Systolic Blood Pressure (SBP) 130-139 mm of Hg and/or Diastolic Blood Pressure (DBP) 80-89 mmHg} as stage 1 HTN. These patients may be at greater risk of perioperative haemodynamic instability, more so in case of laparoscopic surgeries and alpha-2 agonists premedication may be useful in such patients.

Aim: To assess perioperative haemodynamic fluctuations in untreated stage 1 hypertension (HTN) patients and the role of Dexmedetomidine (Dexmed) in it.

Materials and Methods: This prospective, randomised, double blind study was conducted at Pandit Bhagwat Dayal Sharma Postgraduate Institute of Medical Sciences, Rohtak, Haryana, India from March 2019 to September 2020. Sixty-five patients with stage 1 HTN not on any anti-hypertensive drugs undergoing laparoscopic cholecystectomy were enrolled and randomly divided into group D (dexmedetomidine) and group NS (Normal Saline). Group D received Dexmed 1 µg/kg over 10 minutes before induction, followed by continuous infusion of Dexmed at 0.2 µg/kg/h and Group NS received similar volume of normal saline. Serial recording of pulse rate, Mean Arterial Pressure (MAP) and oxygen saturation was done. Postoperative sedation scores, extubation time and time to first rescue analgesia were also assessed. For analysis, quantitative variables were expressed

as mean±SD and compared using unpaired and paired t-test. Mann-Whitney test was used for quantitative data that did not follow a normal distribution. Qualitative variables were expressed as frequencies/percentages and compared using Chi-square test. A p-value <0.05 was considered statistically significant.

Results: A total of 60 patients (30 in group NS, mean age 38.40±10.32 years and 30 in group D, mean age 42.5±11.72 years) were analysed in the present study. In NS group, haemodynamic variations were seen at induction, Laryngoscopy and Intubation (L&I), creation and release of Pneumoperitoneum (PNP) and extubation but actual changes were within 10-12% of baseline value, whereas in group D, pulse rate and MAP remained stable and moderately lower than baseline throughout the perioperative period. An increase in extubation time was observed in group D (15.57±3.16 vs. 9.15±1.60 min). Group D also had significantly higher sedation scores postextubation. Group NS patients demanded rescue analgesia early (18.33±7.46 min vs. 43.53±8.57 min) (p=0.001).

Conclusion: It was concluded that the administration of i.v. Dexmed 1 µg/kg over 10 minutes followed by infusion at 0.2 µg/Kg/h results in haemodynamic stability during surgical stress, slightly delayed but smooth extubation, delayed demand of postoperative rescue analgesia and mild sedation of short duration. Dexmed induced effects help in improving the recovery profile of the patient and keeping the patient more comfortable in the postoperative period.

Keywords: Adrenergic α-2 receptor agonists, Hypertension, Intubation, Pneumoperitoneum

INTRODUCTION

Hypertension (HTN) is a frequent pre-existing and undetected systemic disease, seen in nearly one-third of adult patients presenting for non cardiac procedures [1]. JNC 8 has reclassified earlier prehypertension {SBP 130-139 and/or DBP blood pressure 80-89 mmHg} as stage 1 HTN. This new classification causes substantial increase in HTN prevalence but pharmacotherapy is not advocated in all [2]. These patients when present for surgery are mostly unaware of their HTN and not on any treatment. Hypertensive patients are more prone for intraoperative haemodynamic instability due to adaptive cardiovascular changes and sympathetic hyperactivity, exposing them to greater risk of adverse perioperative outcomes [1]. But no known benefit has been observed by acute initiation of anti-hypertensives in the preoperative period [3].

The PNP in laparoscopic (lap) surgeries amplify these cardiovascular changes that includes increase in Heart Rate (HR), Systemic Vascular

Resistance (SVR) and MAP, and decrease in Cardiac Output (CO) leading to decreased tissue perfusion [4]. American College of Cardiology and the American Heart Association (ACC/AHA) guidelines has been recommended using α-2 agonists, such as clonidine for perioperative HTN management [5] and many studies have evaluated the use of Dexmedetomidine (Dexmed) which is more α-2 receptors selective than clonidine, for preserving the haemodynamic stability [6,7]. It suppresses the release of catecholamines and vasopressin, thereby mitigating the perioperative haemodynamic changes [8]. But no such study has been done in the newly classified stage 1 (JNC 8) hypertensive patients.

Thus, stage 1 hypertensive patients who are usually not on any regular treatment may be at greater risk of perioperative haemodynamic instability, more so in case of lap surgeries and α-2 agonists premedication may be useful in such patients. Hence, we designed this study with the hypothesis that the use of Dexmed may prevent perioperative haemodynamic fluctuations in stage 1 hypertensive

patients during laparoscopic surgery. Primary objective was to compare haemodynamic variations at L&I, creation and release of PNP and extubation among study groups. Secondary outcomes were to observe difference in time to extubation, time for first rescue analgesic requirement and postoperative sedation score among the study groups. Incidence of adverse effects like hypotension, bradycardia and excessive postoperative sedation were also observed.

MATERIALS AND METHODS

This prospective, randomised, double blind study was conducted at Pandit Bhagwat Dayal Sharma Postgraduate Institute of Medical Sciences Rohtak, Haryana, from March 2019 to September 2020, after taking approval from the Institute's Ethical Committee. This trial is registered with Clinical Trial Registry of India (CTRI/2020/07/026594). Informed written consent was obtained from all the patients participating in the study.

Sample size calculation: It was based on a study that took mean change in MAP from baseline of 4.11 ± 5.09 mmHg decrease for Dexmed group as compared to 16.75 ± 4.96 mmHg increase for NS group at induction as statistically significant. Assuming these as reference values, at least 27 patients in each group were required at 5% level of significance and 95% power. Therefore, to compensate for any dropout or exclusion from the study, we planned to enroll 30 patients in each group [9].

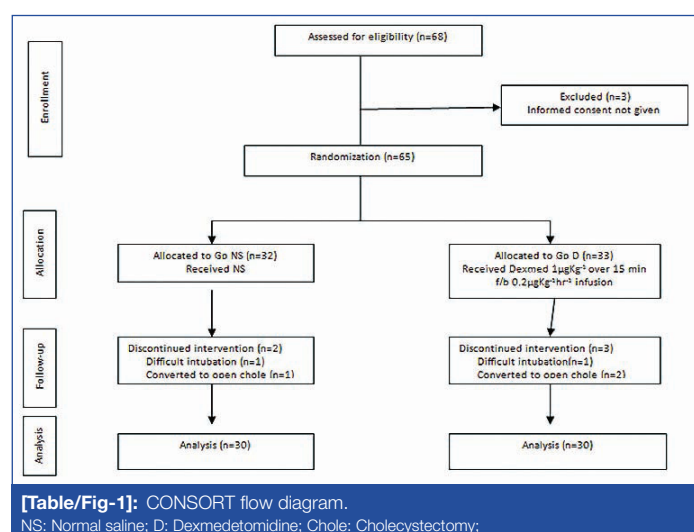
Inclusion criteria: Sixty-five patients aged between 18-65 years, of either sex with stage 1 HTN not taking any anti-hypertensive drugs undergoing laparoscopic cholecystectomy under general anaesthesia were included. Patients with BP recordings taken twice or more in the range of SBP 130-139 mm of Hg and/or DBP 80-89 mm of Hg were enrolled for the study.

Exclusion criteria: Patients with difficult airway, stage 2 HTN (SBP >140, DBP >90 mm of Hg), ASA grade 3, cardiac disease, hepatic, renal and endocrine dysfunction, pregnancy, morbid obesity (body mass index >35 kg/m²) and allergy to Dexmed were excluded.

Tablet alprazolam 0.25 mg was given as premedication at bed time and two hour prior to surgery.

Study Procedure

The patients were randomly allocated to one of the two groups of 30 patients each using sealed coded envelopes. Group D received Dexmed (Dextomid; 200 µg/two mL; Neon laboratories Ltd., Mumbai, India) 1 µg/kg over 10 min before induction, followed by continuous infusion of Dexmed at 0.2 µg/kg/h and Group NS received similar volume of normal saline [Table/Fig-1].



The study drug infusion was prepared by a fellow anaesthesiologist who was not involved in the study. To prepare the infusion, Dexmed 2 mL containing 200 µg of drug was drawn in 50 mL syringe and diluted with NS resulting in final concentration of 4 µg/mL. And in

control group, 50 mL syringe was filled with plain NS. Thus, the syringe size, volume and colour (both were colourless) of prepared solution was same for all patients, only the rate of infusion was different according to patient's weight. Thus, both the attending anaesthetist and the patient were unaware of the group allotted. In operating room, Intravenous (i.v.) access was established and routine monitoring was done. Baseline HR, non invasive SBP, DBP and MAP and oxygen saturation (SpO₂) were recorded. Depending on the actual body weight, the syringe infusion pump was set to deliver the targeted infusion rate. Fifteen minutes after starting the drug infusion, induction was done with i.v. fentanyl (2 µg/kg) and titrated dose of i.v. propofol (1-2 mg/kg) until the loss of verbal response. Endotracheal intubation was facilitated by i.v. vecuronium 0.1 mg/kg. After three minutes, patient was intubated within 30 seconds in a single attempt. Patients requiring more than one laryngoscopy attempts or more than 30 seconds were excluded from the study. Balanced anaesthesia was maintained keeping a Minimum Alveolar Concentration (MAC) of 1. PNP was created with peritoneal insufflations of CO₂ at 2 L/min. Intra-Abdominal Pressure (IAP) was maintained between 12-14 mmHg. Patients were mechanically ventilated using circle system to keep EtCO₂ between 35-45 mmHg. Infusion of the study drug and anaesthetic agents were stopped at the end of surgery and residual neuromuscular blockade was reversed by i.v. neostigmine 0.05 mg/kg and i.v. glycopyrrolate 0.01 mg/kg followed by tracheal extubation. Patients with any surgical complication were excluded from surgery. Serial recording of HR, SBP, DBP, MAP and SpO₂ was done at base line (T_{baseline}), 15 minutes postinfusion (T_{preop} 15), 1 minute postinduction (T_{induction}), 1 minutes postintubation (T_{intubation}), just after the creation of PNP (T_{PNP}), 15 minutes after creation of PNP (T_{PNP} 15), at the release of PNP (T_{PNP} end), postextubation at 1 and 60 minutes (T_{extubation} 1 and T_{extubation} 60). Postoperative pain was assessed using Visual Analogue Scale (VAS) and Ramsay Sedation Score (RSS) [10] was used for sedation assessment. Extubation time was recorded in all the patients. Time to first rescue analgesia was determined from the discontinuation of anaesthetic agents and Dexmed infusion, till patients reported VAS ≥4. Intravenous paracetamol 15 mg/Kg maximum of 1 g was used as rescue analgesic. Sedation score was assessed at 1, 15, 30 and 60 minutes postoperatively. Any adverse effects like bradycardia (HR <60 bpm), tachycardia (HR >100 bpm), hypotension and HTN (MAP <60 or >110 mmHg, respectively on two consecutive readings), excessive sedation RSS >4, respiratory depression (SpO₂ <90%) and dryness of mouth were observed throughout the study. Intraoperative episodes of tachycardia and HTN were managed by bolus of fentanyl (1-2 µg/kg) and increasing MAC from 1-1.2, and if HTN still persists, by starting nitroglycerine infusion at 0.5-1 µg/kg/min.

STATISTICAL ANALYSIS

Statistical Package for Social Sciences (SPSS) version 20.0 was used for statistical analysis. The quantitative variables were expressed as mean±SD and compared using unpaired t-test/Mann-Whitney test between groups and paired t-test within each group. The qualitative variables were expressed as frequencies/percentages and compared using Chi-square test. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 60 patients (30 in each group) were analysed. The demographic profile and surgical variables like age, gender, weight and duration of surgery depict a comparable pattern among both groups [Table/Fig-2]. Comparison of HR and MAP at various time points in both the groups is shown in [Table/Fig-3,4], respectively. Baseline HR and MAP was comparable between the two groups. In group D, a reduction in mean HR and MAP was observed 15 min post-infusion, and thereafter HR and MAP remained stable and lower than baseline throughout the perioperative period. In

group NS, a statistically significant fall in MAP was observed post-induction, and at the release of PNP, whereas decrease in HR was recorded on release of PNP and postextubation. Rise in MAP was noted postintubation, 15 min after the establishment of PNP and one minute postextubation. Similarly, an increase in HR was seen only postintubation. Difference in MAP between two groups was significant at all the times of anaesthesia except baseline [Table/Fig-4]. This difference in HR was also statistically significant between the two groups except postextubation [Table/Fig-3].

Demographic and clinical variables	Group NS (n=30)	Group D (n=30)	p-value
Age (years)	38.40±10.32	42.5±11.72	0.15
Weight (Kg)	57.40±7.731	55.27±7.343	0.27
Sex M/F	2/28	6/24	0.13
Duration of surgery (min)	93.2±10.66	92.27±12.744	0.75

[Table/Fig-2]: Demographic and surgical variables.

Values are in mean±SD or percentage; Unpaired t-test for age; weight and duration of surgery; Chi-square test for sex distribution; M: Male; F: Female; min: Minutes; NS: Normal saline; D: Dexmedetomidine

Time	Group NS (bpm) (mean±SD)	p-value (baseline vs various time points)	Group D (bpm) (mean±SD)	p-value (baseline vs various time points)	p-value (group NS vs Group D)
T _{Baseline}	83.73±10.096	NA	82.73±10.072	NA	0.702
T _{Preop 15}	83.90±10.727	0.949	77.20±7.989	0.021*	0.008*
T _{Induction}	84.73±12.451	0.733	77.17±7.216	0.017*	0.005*
T _{Intubation}	90.97±11.106	0.010*	82.47±6.750	0.906	0.001*
T _{PNP}	85.53±9.594	0.481	79.97±6.289	0.208	0.010*
T _{PNP 15}	81.77±9.669	0.445	77.00±6.023	0.009*	0.025*
T _{PNP end}	77.30±8.159	0.008*	73.40±5.952	0.0001*	0.038*
T _{Extubation 1}	75.67±8.010	0.001*	77.20±5.592	0.010*	0.394
T _{Extubation 60}	74.30±7.516	0.0001*	73.33±5.061	<0.0001*	0.559

[Table/Fig-3]: Trends showing HR (bpm) variability among study groups at various time points.

Paired t-test for intragroup comparison and Unpaired t-test for intergroup comparison; *Statistically significant p<0.05; NS: Normal saline

Time	Group NS (mmHg) (mean±SD)	p-value (baseline vs various time points)	Group D (mmHg) (mean±SD)	p-value (baseline vs various time points)	p-value
T _{Baseline}	99.20±2.340	NA	100.00±2.034	NA	0.162
T _{Preop 15}	100.33±2.171	0.057	91.53±3.060	<0.0001*	<0.0001*
T _{Induction}	92.77±3.025	<0.0001*	89.90±2.998	<0.0001*	0.0005*
T _{Intubation}	106.30±5.472	<0.0001*	94.50±3.785	<0.0001*	<0.0001*
T _{PNP}	100.03±4.650	0.386	91.37±2.512	<0.0001*	<0.0001*
T _{PNP 15}	103.10±4.483	0.0001*	93.17±3.281	<0.0001*	<0.0001*
T _{PNP end}	94.97±2.553	<0.0001*	89.60±3.081	<0.0001*	<0.0001*
T _{Extubation 1}	101.83±2.679	0.0002*	92.13±4.083	<0.0001*	<0.0001*
T _{Extubation 60}	96.93±2.638	0.0008*	88.23±3.048	<0.0001*	<0.0001*

[Table/Fig-4]: Trends showing Mean Arterial Pressure (MAP) (mmHg) variability among study groups at various time points.

Paired t-test for intragroup comparison and Unpaired t-test for intergroup comparison; *Statistically Significant p<0.05

The SpO₂ remained between 98-100% intraoperatively in all patients on Fraction of Inspired Oxygen (FiO₂) of 40% and postoperatively in post-Anaesthesia Care Unit (PACU), all patients were given oxygen via simple Face mask @5 L/min for initial half hour and all patients maintained SpO₂ between 95-100%. Before shifting to ward patients were assessed for any fall in saturation without oxygen. None of the patient in any group had respiratory depression i.e. fall in SpO₂<90%. Time to first rescue analgesia i.e. when VAS more than or equal to four noted and rescue analgesia was given at this point.

A significant increase in extubation time was observed in group D (15.57±3.16 vs.9.15±1.60 min) (p=0.001). Group D also had significantly higher sedation scores at 1, 15 and 30 min postextubation [Table/Fig-5]. Most of the patients were cooperative, oriented and calm with RSS from one to three. Only one patient in group D had RSS of four at one minute postoperatively. At 60 minutes postoperatively, all patients in both the groups were fully awake with RSS of 1. Intravenous paracetamol 15 mg/kg was used as rescue analgesic. Mean duration after which patient demanded rescue analgesia in group NS was 18.33±7.46 min which was earlier as compared to Group D (43.53±8.57 min). This difference in time period of rescue analgesia was found to be statistically significant (p=0.001). The episodes of tachycardia (HR >100 bpm) were recorded in five patients in group NS, while none of the patient in group D had any episode of tachycardia and the difference was statistically significant (p-value=0.0205). Four and three episodes of tachycardia were observed in one patient each, during the entire surgery. Out of remaining three patients, two patients had only one and one patient had two episodes of tachycardia [Table/Fig-6]. Hypertensive episodes were observed in three patients of group NS. Out of these, two patients had only one such episode while one patient had three episodes of HTN at different time points. Contrary to this, in group D, no hypertensive episode was observed and the difference between two groups was statistically insignificant (p=0.078). No episode of hypotension, bradycardia, excessive sedation or respiratory depression was observed in any group. Three patients (10%) in group D complained of excessive dryness of mouth in postoperative period [Table/Fig-6].

Time (postoperative)	Group NS (mean±SD)	Group D (mean±SD)	p-value
1 min	1.933±0.2537	2.633±0.5560	<0.0001*
15 min	1.733±0.4497	2.233±0.4302	<0.0001*
30 min	1.00±0.00	1.233±0.4302	0.005*
60 min	1.00±0.00	1.00±0.00	NA

[Table/Fig-5]: Comparison of postoperative sedation score among study groups.

Mann-Whitney test, *Statistically significant p<0.05, NA: Not applicable; NS: Normal saline; D: Dexmedetomidine; min: Minutes

Adverse effect	Group NS n=30 (%)	Group D n=30 (%)	p-value
Tachycardia	5 (16.67%)	Nil	0.0205*
Hypertension	3 (10%)	Nil	0.078
Dryness of mouth	Nil	3 (10%)	0.078

[Table/Fig-6]: Comparison of adverse events and effects among study groups.

Values are in percentage, Chi-square test, *Statistically significant p<0.05, There were no adverse effects in any subjects observed in terms of Bradycardia, Hypotension, Excessive sedation (RSS >4) and Respiratory depression (SpO₂ <90%)

DISCUSSION

Haemodynamic alterations caused by PNP have deleterious effects in patients with HTN and cardiac disease, increasing their tendency for myocardial ischaemia [11,12]. Dexmed has been used by many researchers for attenuation of haemodynamic responses in different dosages and along with various anaesthetic regimens for multiple surgeries [8,9,13-15]. There is a controversy regarding the effect of Dexmed on blood sugar levels. Hyperglycaemia may be seen with Dexmed use as it causes hypoinsulinemia but it also decreases the cortisol levels and attenuates the neuroendocrine stress response [16]. So, we assessed the haemodynamic fluctuations during laparoscopic cholecystectomy in untreated stage 1 hypertensive patients (previously classified as Pre-HTN in JNC7) and effectiveness of Dexmed to suppress it. Also, as routine blood sugar was monitored both preoperatively and in postoperative period before shifting patient to ward from PICU, no hyperglycaemia was noted. Following Dexmed infusion given over 10 minutes, a moderate fall in HR and MAP from baseline was observed in group D [Table/Fig-3,4]. No

biphasic response was observed in BP as Dexmed was administered as infusion over ten minutes [17,18]. This decrease in HR is frequently due to the combined effect of both baroreflex induced HR slowing in response to initial increase in BP and sympatholytic effect. Similar results were also observed in normotensive patients [19,20]. It was also noted that this Dexmed induced drop in BP seen amongst stage 1 hypertensive patients was not greater than that seen in normotensive patients in previous studies [19,20].

Following induction of anaesthesia, slight increase in HR along with significant decrease in BP was observed in group NS, while there was a persistent moderate decrease in HR and BP from baseline in group D at all stages of anaesthesia. As hypertensive patients have increased Systemic Vascular Resistance (SVR), these patients are more prone to anaesthetic drugs induced vasodilation and hypotension at induction of anaesthesia. However, the maximum drop in BP was approximately 10-12% of the baseline value in both the groups and no treatment in the form of vasopressors was required. Similarly, in another study, authors recorded a moderate decrease in BP but no severe hypotension requiring intervention in Dexmed group [21]. Contrary to the common assumption, that hypertensives are at higher risk of induction induced hypotension, Silva Neto WV et al., observed a smaller reduction in MAP in hypertensive patients ($18.3 \pm 14.0\%$ vs $23.0 \pm 11.4\%$). This is probably due to vascular remodeling and compensatory mechanisms induced increased sympathetic tone that may be responsible for the maintenance of an elevated SVR [22].

Sympathetic stimulation during L&I results in increase in HR and BP [20]. Similar stress hormone responses are seen during CO₂ PNP and extubation. We also noted significant increase in HR and MAP at intubation, PNP and extubation in Group NS with stable haemodynamics in Group D. Similar haemodynamic response to L&I and PNP was observed by various investigators [14,20,23,24]. Also, in another study, it was noted that the elevation in BP at L&I was less in hypertensive patients in comparison to normotensive ($8.2 \pm 16.3\%$ vs $18.2 \pm 21.2\%$ and $8.6 \pm 20.2\%$ vs $25.0 \pm 27.9\%$, respectively for DBP and SBP), as majority of patients (71.8%) in their study were chronic well controlled stage 1 hypertensive [22]. It has also been observed that greater perioperative haemodynamic instability is usually seen in patients with poorly controlled and higher BP ($>180/110$ mmHg) and in patients who have only acute control of BP (<10 days of treatment) [22].

Mean HR was consistently moderately lower in Group D in comparison to Group NS throughout the study period. Findings of the present study were comparable to the other studies, who noted that HR in Dexmed group was significantly lower as compared to baseline and control group [19,25]. Hence, Dexmed also provides smooth emergence from anaesthesia. On further analysing the results of present study, it was found that changes in BP and HR at L&I, PNP and extubation in newly diagnosed stage 1 hypertensives were although statistically significant but actual changes were within 10-12% of baseline value, far below the range seen in other studies among normotensive patients. As the recently categorised stage 1 hypertensive patients were originally normotensive in earlier studies that could be the possible reason for this response seen. Also, all other factors for increased sympathetic stimulation like prolonged and difficult laryngoscopy (cases excluded), inadequate depth of anaesthesia (MAC 1 maintained intraoperatively), normovolemia (fluid administration before PNP creation), slow insufflations of CO₂ and limiting the IAP to <15 mmHg were kept into consideration [26,27].

It has been found that intraoperative physiologic insults like an absolute fall in MAP <50 mmHg or a 40% reduction in MAP from baseline value for ≥ 10 min and tachycardia >100 bpm were associated with perioperative adverse cardiac events [28]. As, tachycardia was observed in few patients of Group NS [Table/Fig-6] with no such adverse episode in Group D, it is advisable to use Dexmed in all stage 1 hypertensive patients undergoing laparoscopic cholecystectomy.

In addition to its sympatholytic effect, Dexmed has additional beneficial effects like analgesia, anxiolysis and sedation. In the present study, an increased extubation time in Group D was observed. However, other authors did not find any significant effect of Dexmed on extubation time [19,20]. As Dexmed had anaesthetic sparing effect in a dose dependent manner and in the present study a standard MAC (1) was maintained intraoperatively in both groups that may be the probable cause of this delayed extubation in Group D [29]. Contrary to the present study, a decreased extubation time in Dexmed group was observed in another study, when Bispectral Index (BIS) guided optimal anaesthetic depth was maintained intraoperatively [15]. Similar to an earlier study, an increase in the time to receive first rescue analgesia in group D when compared to group NS was observed [20]. Although cardiovascular side effects such as bradycardia, sinus arrest and hypotension has been reported with Dexmed use [30] but none of the patients in Dexmed group had excessive sedation [Table/Fig-5] and adverse cardiac effects [Table/Fig-6]. The probable reason for not reporting these side-effects is the use of lower maintenance doses and giving loading dose over 10 minutes infusion. Dexmed causes dryness of mouth and 10% of group D patients complained of it postoperatively while no such complaint was recorded in group NS [31].

Limitation(s)

As ambulatory BP monitoring was not feasible, office BP done in Preanaesthesia Checkup (PAC) clinic and hospital wards was used to categorise patients under stage 1 HTN, and this could be a cause for staging error. However, office BP measured under optimal conditions is usually comparable to ambulatory home BP readings. Also, anaesthetic sparing effect of Dexmed could not be assessed as BIS was not used to monitor the depth of anaesthesia.

CONCLUSION(S)

To conclude, surgical stress following induction, L&I, CO₂ PNP and extubation induces haemodynamic fluctuations in stage 1 hypertensive patients but these changes are not greater than that seen in normotensive patients. A statistically significant decrease in HR and MAP was noted after Dexmed infusion, when compared with the baseline and also with the control group. Though, the significantly lower readings of vitals were noted throughout perioperative period, but this fall was not greater than 10-12% of the baseline. In addition, the dexmed induces significant grade of sedation in the postoperative period. Hence, it is inferred that the administration of i.v. Dexmed $1 \mu\text{g/kg}$ over 10 minutes followed by infusion at $0.2 \mu\text{g/kg/h}$ results in haemodynamic stability during surgical stress, slightly delayed but smooth extubation, delayed demand of postoperative rescue analgesia and mild sedation of short duration. Dexmed induced effects help in improving the recovery profile of the patient and keeping the patient more comfortable in the postoperative period.

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