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**Dr. Shashikiran DS**
Assistant Professor, Department of Anesthesia, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka, India

**Dr. Chetan B Bhat**
Consultant Intensivist and Anesthesiologist, Narayana Multispeciality Hospital, Mysore, Karnataka, India

**Corresponding Author:**
**Dr. Chetan B Bhat**
Consultant Intensivist and Anesthesiologist, Narayana Multispeciality Hospital, Mysore, Karnataka, India

# A study of hemodynamics when different doses of dexmedetomidine is used with propofol as an inducing agent

**Dr. Shashikiran DS and Dr. Chetan B Bhat**

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**Abstract**
Dexmedetomidine is a potent and highly selective α2 adrenoreceptor agonist which was approved for clinical use in 1999 and recently introduced in India. It has all the above mentioned properties and can impart significant benefits in the peri-operative use. In spite of the multiple desirable effects of dexmedetomidine, bradycardia and hypotension remain clinically significant adverse effects. High doses of dexmedetomidine can result in a decreased heart rate and cardiac output, with a biphasic dose response relation for BP. High doses of dexmedetomidine can also be a cause of systemic and pulmonary hypertension.

**Keywords:** Hemodynamics, dexmedetomidine, propofol

## Introduction
Dexmedetomidine is a potent and highly selective α2 adrenoreceptor agonist which was approved for clinical use in 1999 and recently introduced in India. It has all the above mentioned properties and can impart significant benefits in the peri-operative use. 31 In spite of the multiple desirable effects of dexmedetomidine, bradycardia and hypotension remain clinically significant adverse effects. High doses of dexmedetomidine can result in a decreased heart rate and cardiac output, with a biphasic dose response relation for BP. High doses of dexmedetomidine can also be a cause of systemic and pulmonary hypertension [1]. Mnay studies have put an effort to find an answer. Some of them were, Mi and colleagues (1998) evaluated the changes in bispectral index (BIS) and hemodynamic changes to intubation during induction with propofol 30 mg/kg/hr or propofol and 2 µg/ kg fentanyl in twenty four patients. They found that hemodynamic responses to intubation were greater with propofol rather than combination of propofol and fentanyl. Post intubation systolic blood pressure, diastolic blood pressure and heart rate increased, compared with pre induction values, more with propofol. They concluded that fentanyl, 2 µg/kg IV, blunted the hemodynamic responses to intubation, but failed to attenuate the arousal of cerebral cortical activity [2]. Bloor et al. (1992) evaluated four doses of dexmedetomidine in male volunteers. They evaluated effects of 0.25 µg/kg, 0.5 µg/kg, 1 µg/kg, 2 µg/kg infusion and compared it with placebo. They studied hemodynamic responses, cardiac output changes, plasma cathecholamine changes. Dexmedetomidine produced a maximum decrease in mean blood pressure at 60 minutes of 14%, 16%, 23%, and 27% for the 0.25, 0.5, 1.0, and 2.0 µg/kg groups, respectively ($P < .05$). At 330 minutes mean blood pressure remained below baseline by 8% and 17% at the two largest doses ($P < .05$). The two largest doses produced a transient (peak at 3 minutes, lasting less than 11 minutes) increased in mean blood pressure (16 +/- 2.5 and 24 +/- 10 mmHg, 35 respectively) and reduced heart rate (22%). Even the lowest dose decreased cathecolamine values immediately. These dexmedetomidine doses were well tolerated in the healthy volunteers [3].

## Aims and Objectives
To study the hemodynamics when different doses of dexmedetomidine is used with propofol as an inducing agent.

## Materials and Methods

This study was done in the Department of Anesthesia in Shridevi Institute of Medical Sciences and Research Hospital, Tumkur.

This study was done using 60 patients. The study was done from July 2016 to June 2017.

**They were divided into 4 groups**

Group A received 1 µg/kg of dexmedetomidine.
Group B received 0.6 µg/kg of dexmedetomidine.
Group C received 0.3 µg/kg of dexmedetomidine.
Group D received 20 ml of normal saline.

**Inclusion Criteria**

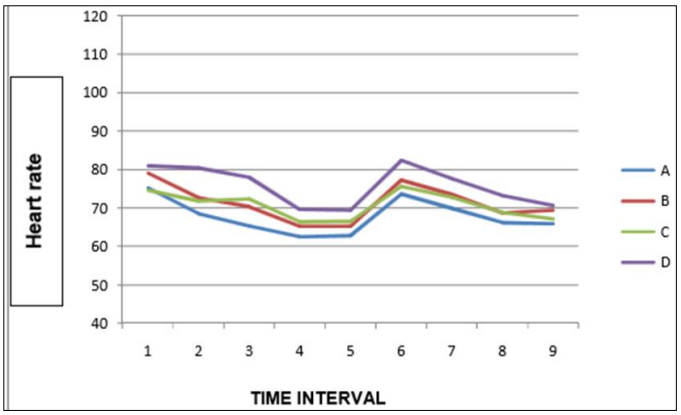
1. The patients were aged between 30-50 years
2. The patients had no co-morbidites

**Exclusion Criteria**

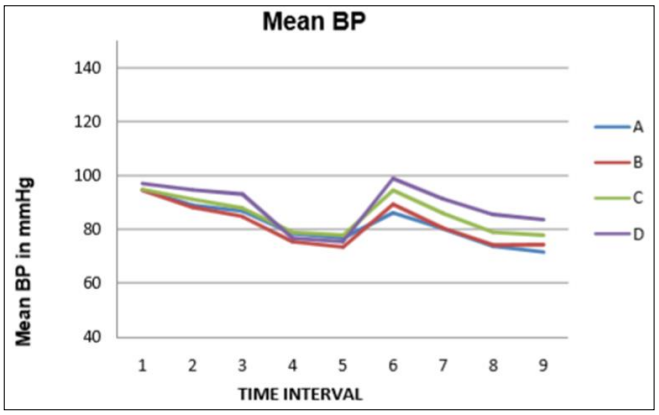
1. Aged below 30 and above 50 years
2. Patients with co-morbidities

All the statistics were done using the SPSS software 2015 (California)

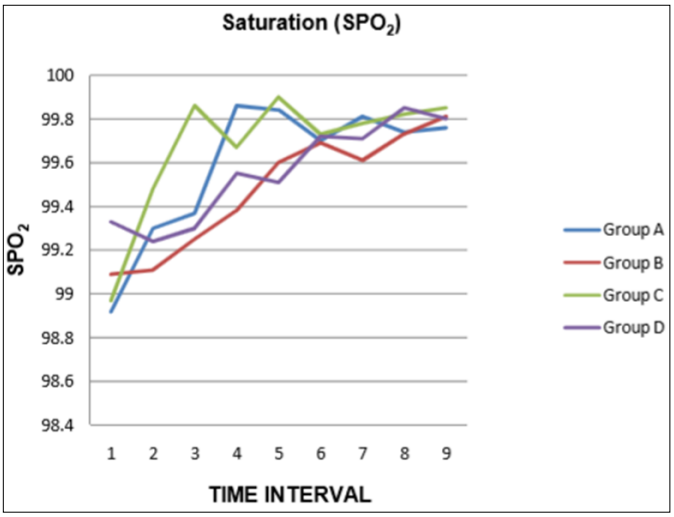
## Results




**Graph 1:** Heart Rate




**Graph 2:** Mean Blood Pressure




**Graph 3:** Saturation

**Note:** Table 1

**Key for time intervals**

1. Baseline
2. 10 min post infusion
3. 20 min post infusion
4. Post intubation
5. Before intubation
6. 1 min post intubation
7. 2 min post intubation
8. 5 min post intubation
9. 10 min post intubation

## Discussion

El-Gohary and coworkers 2010 compared the efficacy of dexmedetomidine and sodium nitroprusside (SNP) in scoliosis surgery as a hypotensive agent in forty patients. They received either dexmedetomidine 1 µg/kg over before induction followed by 0.2–0.5 µg/kg/hour infusion during maintenance or sodium nitroprusside 1–10 µg/kg/minute infusion after induction of anaesthesia. The propofol dose required for induction of anesthesia was significantly lower with dexmedetomdine (1.57 ± 0.27 mg/kg) group than in SNP group (2.28± 0.44 mg/kg). There was a statistically significant increase in the amount of blood loss and blood transfusion requirement in SNP group compared to dexmedetomdine (*P*< 0.01 Dexmedetomidine administration resulted in significant reduction in mean arterial pressure, heart rate and cardiac index. During the steady state hypotension SNP group showed significant increase in heart rate and cardiac index compared to dexmedetomidine. They concluded that dexmedetomidine is an effective and safe agent for controlled hypotension. Compared with sodium nitroprusside, it reduced blood loss. In addition, it posses inherent anaesthetic and analgesic effect [4]. Patel and co-workers (2012) evaluated the effects of intravenous infusion of dexmedetomidine on perioperative hemodynamic changes and postoperative recovery in sixty patients who recieved either fentanyl (2 µg/kg) or dexmedetomidine loading (1 µg/kg) and maintainence infusion (0.2-.0.8 µg/kg/hour). Dexmedetomidine significantly attenuated stress response to36 intubation with lesser increase in heart rate (10% vs. 17%), systolic blood pressure (6% vs. 23%) and diastolic blood pressure (7% vs. 20%). Dexmedetomidine was associated with significant sedation 2 hours post operatively as compared to the control group. They concluded that dexmedetomidine attenuates stress responses during surgery and maintains the haemodynamic stability. The sedative action of dexmedetomidine delays recovery for the first few hours post extubation [5]. Lee and associates (2012) compared the effects of dexmedetomidine and remifentanil

used in anaesthetic induction on hemodynamic changes on direct laryngoscopy and intubation in 90 patients. They received either normal saline or dexmedetomidine 1 µg/kg or remifentanil 1 µg/kg. Anaesthesia was induced with propofol. The heart rate in dexmedetomidne group was significantly lower than other groups 4 minutes after the infusion of dexmedetomidine to immediately before endotracheal intubation. After the intubation, the heart rate in control group remained significantly high compared to the other groups ($P< 0.05$). There were no significant differences between remifentanil and dexmedetomdine groups post intubation in terms of heart rate. The increase in systolic and diastolic BP due to tracheal intubation with dexmedetomidine and remifentanil was significantly lower than control group C ($P< 0.05$) [6, 7].

**Conclusion**

We conclude that 1 µg/kg and 0.6 µg/kg of dexmedetomidine offer desirable hemodynamics.

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