ABSTRACT: AIM: Aim of our study was to assess airway and hemodynamic changes during endotracheal extubation. OBJECTIVE: To evaluate the efficacy of IV dexmedetomidine in attenuating airway reflexes and hemodynamic responses during endotracheal extubation. MATERIAL AND METHODS: 60 patients of ASA grade I, II, & III, divided into two groups, of age between 20-50 years of both sexes, scheduled for receiving general anaesthesia in Gandhi Medical College and Hamidia Hospital, Bhopal (M.P.) Group I (n= 30) (Control Group): 10 ml of normal saline was given 15 min. before the end of surgery. Group II (n= 30): Injection Dexmedetomidine 0.7µg/kg of body weight diluted in normal saline to prepare 10 ml of drug solution was given 15 min. before the end of surgery. Heart rate (HR) was recorded by ECG monitoring; Blood pressure (SBP and DBP) was recorded by non-invasive blood pressure monitoring, Respiratory rate (RR), Oxygen saturation. The pre-operative parameters were recorded before the induction of general anaesthesia. All the patients were observed for airway reflexes (coughing, breath-holding, laryngospasm and bronchospasm at extubation), Hemodynamic responses – HR, SBP DBP and arrhythmias, Respiratory monitoring – RR and SpO2.HR, SBP, and DBP were recorded just before drug administration/basal value (T₀) and 1 min. (T₁), 3 min. (T₂), and 5 min. (T₃) after drug administration, at extubation (T₄) and at 1 min. (T₅), 5 min. (T₆), 10 min. (T₇), and 30 min. (T₈), after extubation. RESULT: Analysis revealed that there was initially insignificant change in mean (±SD) HR, SBP, DBP in both the groups which became highly significant during extubation and remained significantly higher till the end of surgery. Change in the mean (±SD) respiratory rate and spo2of the both the groups were statistically insignificant throughout the study period. CONCLUSION: IV bolus dexmedetomidine is found very effective in attenuating these airway and hemodynamic reflexes without causing any un towards side effect or any other specific complication. KEYWORDS: Dexmedetomidine, attenuates the airway reflexes and hemodynamic responses during extubation, general anaesthesia.

INTRODUCTION: The pressor response, which is part of a huge spectrum of stress response, results from the increase in sympathetic and sympathoadrenal activity, as evidenced by increased plasma catecholamines concentrations in patients undergoing surgery under general anaesthesia. Various drug regimens and techniques have been used from time to time for attenuating the stress response to laryngoscopy and intubation, including opioids, barbiturates, benzodiazepines, beta blockers, calcium channel blockers, vasodilators, etc.

Emergence from general anesthesia and tracheal extubation is often accompanied with tachycardia and hypertension. These responses may produce myocardial ischemia or infarction in
susceptible patients.\cite{12} Drugs like lignocaine, verapamil,\cite{13} esmolol, nicardipine\cite{14} etc. have been proved to attenuate these responses.

Alpha-2 agonists decrease the sympathetic outflow and noradrenergic activity, thereby counteracting hemodynamic fluctuations occurring at the time of extubation due to increased sympathetic stimulation.\cite{12} Clonidine has been studied in this aspect.\cite{15} Dexmedetomidine, a newer drug in this class has been used to attenuate the stress response to intubation.\cite{16}

During tracheal extubation stimulus which affects hemodynamic changes are multifactorial like, light plane of anaesthesia, pain at surgical site, emergence from anaesthesia or tracheobronchial irritation and reflex sympathetic discharge caused by epipharyngeal & laryngopharyngeal stimulation leading to tachycardia, hypertension, with or without arrhythmias.

This transitory rise in pulse rate and blood pressure are probably of little consequence in ASA grade I or II patients but are a matter of concern in patients with cardiovascular diseases like hypertension and coronary artery diseases, because this may lead to complications like angina, myocardial infarction and left ventricular failure due to dangerous increase in myocardial oxygen demand. Sudden increase in arterial pressure may lead to increase in both cerebral blood flow and intracranial pressure which may result in either herniation of brain contents or decrease in cerebral perfusion pressure, leading to cerebral ischemia, similarly rise in intraocular pressure may be hazardous in patients operated for glaucoma. Such stress responses may induce postoperative hemorrhage and potentially fatal cervical hematoma after thyroid surgery.

Recently a potent and highly selective Alpha-2-adrenoceptor agonist (1620:1 α-2 to α-1 receptor) drug, dexmedetomidine compared to clonidine (220:1 α-2 to α-1 receptor), thus reducing unwanted side effects involving α-1 receptors. Dexmedetomidine is approved by FDA in December 1999 for sedation in intensive care units. It exhibits sedative, analgesic, anxiolytic and sympatholytic effects without respiratory depression. Apart from this reduction in plasma epinephrine and norepinephrine levels makes it useful in attenuating hemodynamic stress secondary to hyperadrenergic over reactivity and sedation with dexmedetomidine may facilitate transition to unassisted breathing in agitated patients.

**MATERIAL AND METHODE:** After obtaining informed consent and approval of the institutional ethics committee, this prospective randomized comparative study was conducted on 60 patients of ASA grade I, II, & III, divided into two groups, of age between 20-50 years of both sexes, scheduled for receiving general anaesthesia in Gandhi Medical College and Hamidia Hospital, Bhopal (M.P).

**INCLUSION CRITERIA:**
1. Males and non-pregnant females of ASA grade I, II, & III, aged 20-50 years.
2. Patients undergoing for various surgical procedures in in general surgery neurosurgery, and orthopaedic procedures under general anaesthesia were selected.

**EXCLUSION CRITERIA:**
1. Patients outside the above mentioned age group.
2. Pregnant or lactating mother.
3. Patients allergic to the drug.
4. Patients weighing >20% of the ideal body weight.
5. Patients with severe hypovolemia.
6. Patients receiving methyl Dopa, clonidine, β-blockers, Benzodiazipines, MAO inhibitors.
7. Patients with H/O Hypertension, Ischemic heart disease, Aortic stenosis, LVF, AV Conduction block.
8. Patients with H/O cerebrovascular accidents, severe hepatic and renal diseases, Asthma, COPD and diabetes.
9. All contraindications to general anaesthesia.

ASSESSMENT OF PATIENT:

- Proper pre anaesthetic check-up of all patients.
- All routine investigations like CBP, Urine (R & M), Blood urea, Blood sugar, ECG, & Relevant specific investigation to be done.

All patients were randomly divided into two groups (Group I, II) each of 30 patients.

Group I (n= 30) (Control Group): 10 ml of normal saline was given 15 min. before the end of surgery.

Group II (n= 30): Injection Dexmedetomidine 0.7µg/kg of body weight diluted in normal saline to prepare 10 ml of drug solution was given 15 min. before the end of surgery.

A thorough pre anaesthetic checkup of these patients was done with complete history, general examination, systemic examination, and essential investigations. Patients were kept nil orally for 6hrs before surgery.

PREMEDICATION: All the patients were uniformly pre medicated with injection glycopyrrolate 0.2 mg iv, inj. Ondensetron 4mg, iv, inj. Ranitidine 50mg. iv. Prior to induction.

PREPARATION OF PATIENTS: On arrival of the patients in the operating room, a 18 gauge intravenous cannula was inserted and infusion of 0.9% normal saline was started. The patient was connected to multichannel monitor to record heart rate (HR), non-invasive blood pressure (NIBP), and continuous ECG monitoring and oxygen saturation (SpO2). The base line blood pressure, heart rate, and rhythm were recorded and monitored continuously.

MONITORING: Heart rate (HR) was recorded by ECG monitoring; Blood pressure (SBP and DBP) was recorded by non-invasive blood pressure monitoring. Respiratory rate (RR), Oxygen saturation. The pre-operative parameters were recorded before the induction of general anaesthesia. In our study, observations just before study drug administration were taken as basal value for comparison.

After preparing the patient, pre-oxygenation with 100% oxygen was performed for 5 minutes, inj. Pentazocine 0.5 mg/kg was administered. Intravenous induction was carried out with inj. 2.5% thiopentone sodium 6mg/kg. Inj. Succinylcholine 2mg/kg was administered and intermittent positive pressure ventilation was carried out using 100% oxygen. After laryngoscopy, endotracheal intubation was performed with appropriate size (9 or 8.5 for males, and 7 or 7.5 for female patients, high volume-low pressure cuffed tube) of endotracheal tube.

General anaesthesia was maintained with N2O:O2 (66%:33%) + inhalational agents (halothane 0.2- 0.6%) and muscle relaxation was obtained with Atracurium besylate loading dose of 0.5 mg/kg and 0.1mg/kg of maintenance doses. 15min. before the end of surgery patients in group I
received 10ml of normal saline, and group II received inj. Dexmedetomidine 0.7µg/kg in 10ml of normal saline over 10 min in a double blind design.

Halothane and nitrous oxide were stopped at the end of surgery. Oropharyngeal suctioning was performed immediately prior to extubation and patient was reversed with Inj. Glycopyrrolate 0.01 mg/kg and inj. Neostigmine 0.05 mg/kg. Endotracheal extubation was carried out when patient was completely conscious and responded to verbal commands. After extubation 100% oxygen was given with a face mask for 15 mins.

All the patients were observed for:
1. Airway reflexes – coughing, breath-holding, laryngospasm and bronchospasm at extubation.
2. Hemodynamic responses – HR, SBP DBP and arrythmias.
3. Respiratory monitoring – RR and SpO2.

A decrease in peripheral arterial oxygen saturation >5% from baseline was defined as desaturation and holding breath for >20 seconds or more as breath holding. Any laryngospasm, bronchospasm, and desaturation were recorded. HR, SBP, and DBP were recorded just before drug administration/basal value (T0) and 1 min. (T1), 3 min. (T2), and 5 min. (T3) after drug administration, at extubation (T4) and at 1 min. (T5), 5 min. (T6), 10 min. (T7), and 30 min. (T8), after extubation. Any possible side effects of study drugs (during and after administration of drug) such as bradycardia, tachycardia, hypotension, hypertension, vomiting and dry mouth, sedation were recorded if observed any.

The observations recorded in both the groups were tabulated and statistical analysis carried out by using appropriate software. P-value < 0.05 taken as statistically significant.

RESULT AND ANALYSIS: Mostly patients from both the groups are between the age group of 30-39 yrs. (table-1)

Table-2 shows that male patients were 60% and 63%, while female patients were 40% and 37% in Group I and II respectively.

Table-3 shows that there was an abrupt rise in mean heart rate during and immediately after endotracheal extubation. The rise in mean HR was maximum in Group I as compared to Group II.

Table-4 shows that there is initially statistically insignificant change in mean (±SD) heart rate in both the groups 1 min. after administration of the study drug, which became statistically highly significant after 3 min. of drug administration and became highly significant throughout the end of study.

Table-5 shows that at the end of surgery there was slight increase in mean (±SD) SBP in both the groups. In Group II during extubation there was slight increase in SBP which drops 5min. after extubation, and comes to normal level 30 min. after extubation. While in Group I there is an abrupt increase in SBP during extubation which was significantly raised till the end of the study.

Table-6 shows that there was initially insignificant change in mean (±SD) SBP in both the groups which became highly significant during extubation and remained significantly higher till the end of surgery.

Table-7 shows that at the end of surgery there was slight increase in mean (±SD) DBP in both the groups. In Group II during extubation there was slight increase in DBP which drops 5min. after
extubation, and comes to normal level 30 min. after extubation. While in Group I there is an abrupt increase in DBP during extubation which was significantly raised till the end of the study.

Table-8 shows that there was initially insignificant change in mean (±SD) DBP in both the groups which became highly significant during extubation and remained significantly higher till the end of surgery.

As before endotracheal extubation all patients were under controlled ventilation and there respiratory rate was controlled. Table-9 shows that changes in mean (±SD) respiratory rate was statistically insignificant for both the groups after extubation.

Table-10 shows that change in the mean (±SD) respiratory rate of the both the groups were statistically insignificant throughout the study period.

Table-11 shows that the changes in mean (±SD) SpO₂ for both the groups were statistically insignificant.

Table-12 shows that in Group I, 66.66% patients did not cough during extubation while 26.67% and 6.67% patients had mild and moderate coughing respectively. In Group II, only 6.66% patients had mild cough while 93.34% patients had no cough during extubation.

Table-13 shows that all the patients in Group I were anxious and agitated (sedation score 1) after extubation throughout the study period. In Group II, 76.66% patients had sedation score 3 (responding to command only) while 16.66% patients had sedation score 2 (co-operative, orientated and tranquil) and only 6.66% patients were anxious and agitated (sedation score 1).

**DISCUSSION:** Emergence from general anaesthesia is frequently complicated with dangerous patient’s movement, hypertension, tachycardia, arrhythmias, myocardial ischemia, surgical bleeding, laryngospasm, bronchospasm, increase in intracranial pressure and intraocular pressure. The incidence of coughing on emergence from general anaesthesia in the presence of an endotracheal tube has been estimated as ranging between 38% -96%.

Strategies to circumvent these changes have been described with variable success. These includes extubation of tracheal tube while the patient is in deep plane of anaesthesia, use of topical anaesthesia and administration of drugs such as opioids lidocaine beta blockers calcium channel blockers before tracheal extubation.

The present study was carried out to assess the effectiveness of IV Dexmedetomidine in attenuation of airway reflexes and hemodynamic responses during endotracheal extubation.

In our study there was even distribution of age in the two groups with maximum number of patients in the age group of 30–39 years (table-1). As shown in Table-2, no statistical significance was observed as regard to distribution of sex between the two groups.

**MEAN HEART RATE:** Table -3 shows that while comparing mean heart rate of group I and II there was increase in mean HR at T₂ in group I but in group II decrease in mean HR was observed meanwhile at T₁ and T₃. Thereafter at T₄ an abrupt rise in mean HR was observed in both the groups which was maximum in group I this rise in HR was continued till the end of surgery in group I while in group II this raised HR during and after extubation was dropped down to the basal value at T₇ and remained little bit as such till the end of study that is at T₈.

Table -4 shows that while comparing the mean (±SD) HR of the two groups statistically insignificant (P>0.05) values were observed initially till T₁ (1 min. after drug administration, which
became statistically highly significant (P<0.01) at T_2 (3 min. after drug administration) and thereafter highly significant throughout the study period.

So from our study we found that iv bolus injection dexmedetomidine is quite effective in attenuating the raised HR during extubation. These findings are similar to those of following studies: Arpino et al[17] in their study on dexmedetomidine in facilitating extubation in intensive care unit in 25 patients found significantly lower heart rate in patients who failed previous attempts at weaning and extubation secondary to agitation.

Guler et al[18] in their study they found a significant rise in HR during extubation in dexmedetomidine group and placebo group. But these were statistically significantly raised in placebo group till the end of study. They found dexmedetomidine is effective in attenuating the tachycardia occurred during and after the extubation without affecting the emergence time. In our study lowest mean HR (70 ± 4.4) was found at 3 min after dexmedetomidine administration similar to Guler et al.

Talke et al[19] found dose dependent reduction in arterial blood pressure and HR after administration of dexmedetomidine during emergence from anaesthesia.

**MEAN SYSTOLIC BLOOD PRESSURE:** Table -5 shows that there was steady increase in SBP at the end of surgery which raised abruptly at T_4 (at extubation) in group I and remained raised throughout the study period. While in Group II towards the end of surgery a slight rise in SBP was noted which reached to its maximum at T_4,this rise is very less than that of the Group I at same time. In Group II this rise in SBP started to come down from T_5 (5 min. after extubation) reached to its basal value at T_7 (5 min. after extubation) and then even come down below baseline at T_9 (30 min. after extubation).

Table -6 shows that on comparing the mean (± SD) SBP of the two groups initially there was no statistical significance in change in SBP was found in both the group till T_3 (5 min. after drug administration. But thereafter at the time of extubation (T_4) this difference in mean (± SD) was found significantly high and remained significantly high for the rest of the period. These findings are consistent with those of the following studies carried out by:

Arpino et al in their study found similar control in hemodynamics and circulatory reflexes in facilitating extubation in agitated patients.

Guler et al[20] also found similar result in their study. They observed significantly lower mean SBP in group II (Dexmedetomidine group) from 3 min after drug administration up to the end of study.

Turan C et al[21] carried out their study on patients undergoing intracranial surgery by giving two different doses of dexmedetomidine at the time of extubation and found an optimal hemodynamic control during and after extubation.

Our results are also in accordance with Tanskanen et al. (2006)[22]who conducted a study and conclude that Dexmedetomidine blunted the tachycardic response to intubation and the hypertensive response to extubation. They concluded that Dexmedetomidine increased perioperative haemodynamic stability in patients undergoing brain tumour surgery.

Our results are also in accordance with. Dogru et al. (2007)[23] who conducted a study and conclude that Dexmedetomidine attenuated, but did not completely prevent, haemodynamic responses to tracheal intubation in these patients.
MEAN DIASTOLIC BLOOD PRESSURE: Table-7 shows that at the end of surgery there was steady increase in DBP in both the groups till T3 but this rise in DBP is greater in group I as compared to group II. Thereafter there was an abrupt rise in DBP in group I at the time of extubation (T4) which reached to its maximum at T5 (1 min. after extubation) and then drop down slowly towards the end of study but remained above the basal value at the end of study. In group II this rise in DBP was maximum at T4 and then started to decrease which was continued till end of study period it comes down near to basal level.

Table-8 shows that on comparing the mean (±SD) DBP of two groups it was found that there is no statistical significance (p>0.05) was observed in between the two groups up to 5 min. after drug administration (T3). But then at the time of extubation and thereafter till the end of study statistically highly significant (<0.01) change in mean (± SD) DBP was found between two groups. These findings are similar to those of following:

Arpino et al found similar hemodynamic stability with the use of dexmedetomidine in facilitating extubation in the intensive care unit in 25 mechanically ventilated patients.

Guler et al reported similar rise in DBP initially during extubation in both the placebo group and dexmedetomidine group. But this rise in DBP was significantly higher in placebo group. In their study they found that mean DBP was lower in dexmedetomidine group from 3 min. after drug administration till the end of surgery.

Turan G(24) et al while studying the effect of two different doses of dexmedetomidine in patients undergoing intracranial surgeries found the similar result during extubation. They observed significantly good control over hemodynamic and circulatory reflexes during extubation.

MEAN RESPIRATORY RATE: Table-9 showing that period between T3-T4 is not included in the study as at that period patients were intubated and receiving controlled ventilation. As shown in table-9 and 10 the changes in mean (± SD) respiratory rate between the two groups from T4 to T8 was found statistically insignificant (p>0.05).

Our finding is consistent with those of Siobal et al,(25) who observed that dexmedetomidine in the dose of 0.5μg/kg does not cause any significant respiratory depression.

MEAN OXYGEN SATURATION (SpO₂): As shown in table 11 the period between T6-T4 was excluded from the study as in this period patients were on controlled ventilation. From our study we found that there is no statistical significance in changes in mean (± SD) SpO₂ was observed between two groups from extubation till the end of study. Study is similar to those of Siobal et al, they concluded that dexmedetomidine does not cause any respiratory depression or desaturation following extubation.

AIRWAY REFLEXES: Table-12 showing that in Group I 26.67% had mild coughing after extubation while 6.67% had moderate degree of coughing on extubation. On the other hand in Group II only 6.66% had mild coughing after extubation while 93.34% did not cough at all after extubation. Guler et al found similar reduction in incidence of coughing in dexmedetomidine group (30%) as compared to placebo group (63%).

Our results are also in accordance with Aksu et al. (2009)(26) who conducted a study and conclude that Dexmedetomidine was more effective in attenuating airway reflex responses to
tracheal extubation and maintaining haemodynamic stability without prolonging recovery compared to Fentanyl.

SEDATION: As shown in table- 13 in Group I all the patients were had Ramsey sedation score of 1. While in Group II 76.66% patients were scored Ramsey sedation score 3 and 16.66% patients had score 2 and only 6.66% patients remained agitated and anxious after extubation. From these data we concluded that dexmedetomidine achieves a better level of arousable sedation after extubation which is helpful in preventing dangerous patient movement on emergence.

Arpino C et al on their study found similar level of sedation in 25 ICU patients after extubation. They concluded that dexmedetomidine produces sedation and anxiolysis without any respiratory depression.

Siobal et al also found similar level of sedation in their patients without any significant respiratory depression.

CONCLUSION: On the basis of present study the following conclusions are being drawn: There is abrupt rise in all hemodynamic and circulatory parameters during and after extubation was observed.

Also airway reflexes were triggered during and after extubation in the form of cough. IV bolus dexmedetomidine is found very effective in attenuating these airway and hemodynamic reflexes without causing any un towards side effect or any other specific complication.

| S. no. | Age (in years) | Group I | Group II |
|-------|----------------|---------|----------|
|       |                | N       | %        | N       | %        |
| 1     | 20-29          | 8       | 26.67    | 8       | 26.67    |
| 2     | 30-39          | 16      | 53.33    | 15      | 50.0     |
| 3     | 40-50          | 6       | 20.0     | 7       | 23.33    |

**TABLE 1: SHOWING DISTRIBUTION OF CASES ACCORDING TO AGE**

Mostly patients from both the groups are between the age group of 30-39 yrs.

| Sex    | Group I | Group II |
|--------|---------|----------|
|        | N       | %        | n        | %        |
| Male   | 18      | 60       | 19       | 63       |
| Female | 12      | 40       | 11       | 37       |
| Total  | 30      | 100      | 30       | 100      |

**TABLE 2: TABLE SHOWING DISTRIBUTION OF CASES ACCORDING TO SEX**

Table-2 shows that male patients were 60% and 63%, while female patients were 40% and 37% in Group I and II respectively.

| Time of Observation | Group I (Mean ± SD) | Group II (Mean ± SD) |
|---------------------|---------------------|---------------------|
| T₀                  | 76 ± 6.28           | 77 ± 5.4            |
| T₁                  | 77 ± 4.44           | 76 ± 5.28           |
TABLE 3: TABLE SHOWING MEAN (± SD) HEART RATE IN TWO GROUPS

| T_0  | 78 ± 5.98 | 70 ± 4.4 |
|------|-----------|----------|
| T_1  | 81 ± 3.65 | 73 ± 4.6 |
| T_2  | 106 ± 4.89| 94 ± 3.58|
| T_3  | 122 ± 3.9 | 95 ± 4.03|
| T_4  | 110 ± 3.5 | 82 ± 3.7 |
| T_5  | 101 ± 2.5 | 76 ± 3.7 |
| T_6  | 94 ± 1.98 | 75 ± 4.04|

T_0 – Basal value, just before the study drug, T_1 - 1 min. after drug administration, T_2 – 3 min. after drug, T_3 - 5 min. after drug, T_4 – during extubation, T_5 – 1 min. after extubation, T_6 – 5 min. after extubation, T_7 – 10 min. after extubation, T_8 – 30 min. after extubation.

Table-3 shows that there was an abrupt rise in mean heart rate during and immediately after endotracheal extubation. The rise in mean HR was maximum in Group I as compared to Group II.

| Time of Observation | Comparison of mean (±SD) of Group I&II |
|---------------------|----------------------------------------|
|                     | p-value | Significance |
| T_0                 | >0.05   | NS           |
| T_1                 | >0.05   | NS           |
| T_2                 | <0.01   | HS           |
| T_3                 | <0.01   | HS           |
| T_4                 | <0.01   | HS           |
| T_5                 | <0.01   | HS           |
| T_6                 | <0.01   | HS           |
| T_7                 | <0.01   | HS           |
| T_8                 | <0.01   | HS           |

TABLE 4: TABLE SHOWING STATISTICAL COMPARISON OF MEAN (± SD) HEART RATE OF THE TWO GROUPS

Table-4 shows that there is initially statistically insignificant change in mean (±SD) heart rate in both the groups 1 min. after administration of the study drug, which became statistically highly significant after 3 min. of drug administration and became highly significant throughout the end of study.

| Time of observation | Group I (Mean ± SD) | Group II (Mean ± SD) |
|---------------------|---------------------|----------------------|
| T_0                 | 114 ± 7.2           | 113.8 ± 6.99         |
| T_1                 | 116 ± 7.7           | 117 ± 8.6            |
| T_2                 | 120.6 ± 8.4         | 118 ± 8.7            |
| T_3                 | 122.8 ± 7.2         | 124 ± 7.3            |
| T_4                 | 148 ± 6.3           | 128 ± 6.2            |
| T_5                 | 146.4 ± 4.5         | 125.4 ± 4.8          |
| T_6                 | 138 ± 5.3           | 116.8 ± 5.06         |
| T_7                 | 136 ± 6.25          | 112 ± 3.9            |
Table-5 shows that at the end of surgery there was slight increase in mean (±SD) SBP in both the groups. In Group II during extubation there was slight increase in SBP which drops 5 min. after extubation, and comes to normal level 30 min. after extubation. While in Group I there is an abrupt increase in SBP during extubation which was significantly raised till the end of the study.

Table 6 shows that there was initially insignificant change in mean (±SD) SBP in both the groups which became highly significant during extubation and remained significantly higher till the end of surgery.

### Table 5: Table Showing Mean (± SD) Systolic Blood Pressure (mmHg) of the Two Groups

| Time of Observation | Comparison of Mean (±SD) of Group I&II | p-value | Significance |
|---------------------|----------------------------------------|---------|--------------|
| T₀                  | >0.05                                  | NS      |              |
| T₁                  | >0.05                                  | NS      |              |
| T₂                  | >0.05                                  | NS      |              |
| T₃                  | >0.05                                  | NS      |              |
| T₄                  | <0.01                                  | HS      |              |
| T₅                  | <0.01                                  | HS      |              |
| T₆                  | <0.01                                  | HS      |              |
| T₇                  | <0.01                                  | HS      |              |
| T₈                  | <0.01                                  | HS      |              |

### Table 6: Table Showing Statistical Comparison of Mean (±SD) Systolic Blood Pressure of the Two Groups

| Time of Observation | Group I (Mean ± SD) | Group II (Mean ± SD) |
|---------------------|---------------------|----------------------|
| T₀                  | 68 ± 4.1            | 69 ± 3.4             |
| T₁                  | 72.4 ± 3.5          | 71 ± 3.3             |
| T₂                  | 76 ± 3.5            | 76 ± 3               |
| T₃                  | 84 ± 4.6            | 82 ± 3.2             |
| T₄                  | 92 ± 5.3            | 85 ± 3.3             |
| T₅                  | 98 ± 4              | 81 ± 3.5             |
| T₆                  | 91.5 ± 3.7          | 78 ± 2.8             |
| T₇                  | 87 ± 4.7            | 74.8 ± 2.9           |
| T₈                  | 85 ± 3.7            | 73.3 ± 3.4           |

### Table 7: Table Showing Mean (±SD) Diastolic Blood Pressure (mmHg) of the Two Groups
Table-7 shows that at the end of surgery there was slight increase in mean (±SD) DBP in both the groups. In Group II during extubation there was slight increase in DBP which drops 5 min. after extubation, and comes to normal level 30 min. after extubation. While in Group I there is an abrupt increase in DBP during extubation which was significantly raised till the end of the study.

| Time of Observation | Comparison of (Mean ± SD) of Group I&II |
|---------------------|----------------------------------------|
|                     | p-value  | Significance |
| T₀                  | >0.05    | NS           |
| T₁                  | >0.05    | NS           |
| T₂                  | >0.05    | NS           |
| T₃                  | >0.05    | NS           |
| T₄                  | <0.01    | HS           |
| T₅                  | <0.01    | HS           |
| T₆                  | <0.01    | HS           |
| T₇                  | <0.01    | HS           |
| T₈                  | <0.01    | HS           |

**TABLE 8: TABLE SHOWING STATISTICAL COMPARISON OF MEAN (± SD) DIASTOLIC BLOOD PRESSURE (mmHg) OF THE TWO GROUPS**

Table-8 shows that there was initially insignificant change in mean (±SD) DBP in both the groups which became highly significant during extubation and remained significantly higher till the end of surgery.

| TIME OF OBSERVATION | GROUP I (Mean ± SD) | Group II (Mean ± SD) |
|---------------------|----------------------|----------------------|
| T₀                  | NA                   | NA                   |
| T₁                  | 16 ± 1.7             | 15.5 ± 1.6           |
| T₂                  | 18 ± 1.9             | 18 ± 1.8             |
| T₃                  | 18 ± 1.3             | 18 ± 1.5             |
| T₄                  | 16.8 ± 1.5           | 17 ± 1.8             |

**TABLE 9: TABLE SHOWING MEAN (± SD) RESPIRATORY RATE OF THE TWO GROUPS**

NA- Not applicable

As before endotracheal extubation all patients were under controlled ventilation and there respiratory rate was controlled. Table-9 shows that changes in mean (±SD) respiratory rate was statistically insignificant for both the groups after extubation.
Table-10 shows that change in the mean (±SD) respiratory rate of the both the groups were statistically insignificant throughout the study period.

Table-11 shows that the changes in mean (±SD) SpO₂ for both the groups were statistically insignificant.
Coughing was assessed by using 4-point scale:
1. No coughing.
2. Minimal coughing.
3. Moderate coughing. (3-4 times)
4. Severe coughing. (5 or more)

Table-12 shows that in Group I, 66.66% patients did not cough during extubation while 26.67% and 6.67% patients had mild and moderate coughing respectively. In Group II, only 6.66% patients had mild cough while 93.34% patients had no cough during extubation.

| Groups     | Score 1 | Score 2 | Score 3 | Score 4 | Score 5 | Score 6 |
|------------|---------|---------|---------|---------|---------|---------|
|            | n | %     | n | %     | n | %     | n | %     | n | %     |
| Group I    | 30 | 100   | 0 | 0     | 0 | 0     | 0 | 0     | 0 | 0     |
| Group II   | 2  | 6.66% | 5 | 16.66% | 23| 76.66%| 0 | 0     | 0 | 0     |

**TABLE 13: TABLE SHOWING SEDATION SCORE AFTER EXTUBATION IN THE TWO GROUPS**

Sedation was assessed by using RAMSEY SEDATION SCORING SCALE which is as follows:
1. Patient anxious and agitated or restless, or both.
2. Patient co-operative, orientated and tranquil.
3. Patient response to commands only.
4. Brisk response to a light glabellar tap or auditory stimulus.
5. Sluggish response to a light glabellar tap or auditory stimulus.
6. No response to any stimuli mentioned above.

Table-13 shows that all the patients in Group I were anxious and agitated (sedation score 1) after extubation throughout the study period. In Group II, 76.66% patients had sedation score 3 (responding to command only) while 16.66% patients had sedation score 2 (co-operative, orientated and tranquil) and only 6.66% patients were anxious and agitated (sedation score 1).

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AUTHORS:

1. Ravi Shankar Goarya
2. Ashish Mathur

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Anaesthesia, Late Shri B. R. Kashyap Medical College, Jagdalpur, C. G.
2. Former Post Graduate Student, Department of Anaesthesia, Gandhi Medical College, Bhopal, M. P.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Ashish Mathur,
# 15, Adarsh Colony,
Gola Ka Mandir,
Gwalior-474005, M. P.
Email: ashishgrmc2012@gmail.com

Date of Submission: 07/11/2014.
Date of Peer Review: 08/11/2014.
Date of Acceptance: 21/11/2014.
Date of Publishing: 25/11/2014.