DOI: 10.14260/jemds/2014/3802

ORIGINAL ARTICLE

COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATING HEMODYNAMIC CHANGES DURING LARYNGOSCOPY

Smitha K. S[1], Divya Shukla[2], Sathesha M[3], Raghavendra Rao[4], Nethra S. S[5], K. Sudheesh[6]

HOW TO CITE THIS ARTICLE:
Smitha K. S, Divya Shukla, Sathesha M, Raghavendra Rao, Nethra S. S, K. Sudheesh. "Comparison of two different Doses of Dexmedetomidine in attenuating Hemodynamic Changes during Laryngoscopy". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 61, November 13; Page: 13501-13508,
DOI: 10.14260/jemds/2014/3802

ABSTRACT: The sequence of induction of anaesthesia, laryngoscopy and tracheal intubation are associated with marked hemodynamic changes and autonomic reflex activity which may be a cause of concern in many high risk patients. In this study, we compare the effects of two different doses of Dexmedetomidine, 1μg/kg and 0.5μg/kg with control group with respect to hemodynamic responses such as heart rate, blood pressure, attenuation of sympathetic responses to laryngoscopy and intubation and undesirable effects. **METHODS:** 90 ASA I and II status normotensive patients scheduled for elective surgical procedure were selected and randomly divided into three groups of 30 each. Group 1 patients received 0.5μg/kg in 10 minutes, group 2 patients received 1μg/kg in 10 minutes and group 3 patients received Normal Saline. 60 secs after the infusion, patient was induced with Inj Fentanyl 1-2μg/kg, Inj. propofol and Inj Vecuronium administered. Intubation was performed. Values of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR) and O2 saturation (SpO2) were recorded at baseline (T0), 60 sec after Dexmedetomidine infusion (T1), 60 sec after induction (T2), 60 sec after intubation (T3), 5 min after intubation (T4). **RESULTS:** It was found that SBP, DBP, MAP levels were significantly lower at T3 period in group 2 than in group 1 and group 3 ($p<0.05$, $p<0.01$, $p<0.05$). Also, HR levels were significantly lower at T3 in group 2 than in group 1 and group 3($p<0.01$).There was no significant changes in SpO2 levels between the groups. **CONCLUSION:** Through this study, we empirically prove that Dexmedetomidine 1 μg.kg-1 is more effective than Dexmedetomidine 0.5 μg.kg-1 in controlling haemodynamic responses to tracheal intubation.

KEYWORDS: Dexmedetomidine, Anaesthesia, intubation, Attenuation, Pressor response, Laryngoscopy.

INTRODUCTION: Endotracheal intubation is the trans laryngeal placement of a tube into the trachea via the nose or mouth. Endotracheal intubation includes laryngoscopy & intubation. The process of laryngoscopy & intubation are noxious stimuli & therefore constitute a period of extreme hemodynamic stress and is associated with intense sympathetic activity marked by tachycardia & hypertension.

The increases in Pulse rate, Blood pressure are usually transitory, variable & unpredictable. Normal, healthy person tolerates this response, but in susceptible individuals, this transient sympathetic response can evoke life – threatening conditions.

The magnitude of the response is greater with increasing force and duration of laryngoscopy.[1] The elevation in arterial pressure typically starts within five seconds of laryngoscopy, peaks in 1-2 min and returns to control levels within 5 min. Reid and Brace in 1940 were the first to report the circulatory responses to laryngeal and tracheal stimulation in an anesthetized man.[2] A variety of

DOI: 10.14260/jemds/2014/3802

drugs have been used to control this hemodynamic response, such as vasodilators, beta blockers, calcium channel blockers, alfa$_2$ agonists and opioids. However, no modality was devoid of drawbacks and limitations.

Dexmedetomidine is a highly selective, specific & potent alpha- 2 adrenergic agonist. Compared to clonidine it is said to be 7-10 times more alpha- 2 selective & has a shorter duration of action than clonidine. Pre-treatment with dexmedetomidine attenuates hemodynamic response to tracheal intubation.[3] The present study was designed to investigate the effect of dexmedetomidine and to compare two different doses on hemodynamic responses to orotracheal intubation.

OBJECTIVE: The aim of the present study is to investigate the effects of two different doses of Dexmedetomidine infusion in controlling haemodynamic response to tracheal intubation and to look for any undesirable effects with respect to control group.

METHODOLOGY: After obtaining approval from the Ethics Committee and written informed consent, 90 ASA I or II patients aged between 18-60 years undergoing elective procedures under GA were studied.Patients undergoing various Orthopaedic, ENT and General surgical procedures were selected.

INCLUSION CRITERIA: Patients aged between 18-60yrs, Patients of either sex, Patients with ASA grade I & II, Patients scheduled for elective surgical procedure under general anaesthesia.

EXCLUSION CRITERIA: Patients with anticipated difficult airway, Hiatus hernia, GERD, Patients on antihypertensive drugs, sedatives, hypnotics & antidepressants, H/o cardiovascular, respiratory, hepatic, renal diseases, Laryngoscopy time > 20 sec, Patients with ASA grade III & above, Endocrine diseases. Pre-Anaesthetic assessment: On the day prior to surgery a thorough clinical examination of the patient was performed including General Physical Examination & systemic examination. All patients were explained about the anaesthesia technique & written informed consent taken. Patients were kept NPO according to the guidelines.

LAB INVESTIGATIONS: Routine investigations were done. {Hb %, BT, CT, Urine analysis, ECG, BUN, Serum creatinine, & Fasting blood sugar.}No specific investigations were required pertaining to the study.

PRE-MEDICATION: All patients were given tablet Alprazolam 0.5mg orally at bed time on the previous night of surgery.

TECHNIQUE OF ANAESTHESIA: 90 patients aged between 18 to 60 years belonging to ASA grade I & II were randomly divided into 3 groups, each group consisted of 30 patients. The study was conducted in a double-blind fashion. Anaesthesia machine, circuits checked, resuscitation equipment's were kept ready. On the day of surgery after confirmation of NPO status patients were shifted to the operating room & connected to multi-channel monitor. Basal systolic blood pressure (SBP), diastolic blood pressure(DBP), Mean arterial pressure (MAP), heart rate, ECG & SpO_2 were recorded (T_0).

DOI: 10.14260/jemds/2014/3802

Continuous monitoring of the vital parameters was done. An I V line was secured with an appropriate sized cannula in all patients. Dexmedetomidine infusion was prepared by adding 2ml of dexmedetomidine, i.e, 200 µg, in 48 ml of 0.9% NS in a syringe.

Hence, the preparation contained 4µg/ml of dexmedetomidine in 0.9% NS. Group 1 patients- received I V Dexmedetomidine 0.5µg per kg, infused over 10mins through a controlled infusion device. Group II patients- received IV Dexmedetomidine 1 µg/kg, infused over 10 mins through a controlled infusion device. Group III patients-received IV 0.9% Normal Saline, Infused over 10minutes through a controlled infusion device.

After 1min of stabilizing period, SBP, DBP, MAP, Heart rate, SpO_2 (T1) were recorded. Prior to induction, Inj Glycopyrrolate 0.2mg, Inj Ondansetron 4mg, & Inj Rantac 50mg was administered IV. All patients were pre-Oxygenated for 3 minutes & Anaesthesia was induced with Inj Fentanyl 1-2µg/kg & sleep dose of propofol. After successful trial ventilation, Vecuronium 0.1mg / kg was given to facilitate laryngoscopy & intubation. Oxygenation was continued through positive pressure mask ventilation using closed circuit. Maintained with O_2, N_2O, Isoflurane. At 1min after induction, SBP, DBP, MAP, Heart rate & SpO_2 were recorded (T_2).

At the onset of apnoea, using laryngoscope with a Macintosh blade, intubation was done with well lubricated, appropriate sized cuffed oral endotracheal tube. Laryngoscopy & intubation time was kept minimum (20sec). SBP, DBP, MAP, Heart rate, SpO_2 were recorded 1minute after intubation (T_3). After confirmation of the tube position, cuff was inflated, tube fixed, Connected to machine through closed circuit. Anaesthesia was maintained with N_2O, O_2, Isoflurane, controlled ventilation with appropriate fresh gas flow.

Measurements of SBP, DBP, MAP, HR and SpO_2 were performed 60 seconds after Dexmedetomidine infusion (T_1), 60 seconds after induction (T_2), 60 seconds after intubation (T_3) and 5 minutes after intubation (T_4).

Sequence	**SBP, DBP, MAP, Heart rate, SpO_2 recording**
Basal reading when the patient is shifted to OT	T_0
At 1 min after infusion of Dexmedetomidine.	T_1
At Induction (1min after sleep dose of Propofol)	T_2
At 1 min after intubation	T_3
At 5 mins after intubation	T_4

Table 1

Surgery commenced at the end of 5mins after laryngoscopy & intubation. No form of stimulus was applied during the study period. Anaesthesia was continued with N_2O (50%), O_2 (50%), Isoflurane. Vecuronium top up doses, analgesics & IV fluids administered based on the requirements. At the end of surgery, when patients had respiratory attempts, residual neuromuscular blockade was reversed with Inj Neostigmine & Glycopyrolate. Recovery was assessed & extubation was done after

DOI: 10.14260/jemds/2014/3802

thorough throat suction. After complete clinical recovery patients were shifted to post anaesthesia care unit, observed for 2 hrs for Nausea, vomiting, Bradycardia, Hypotension, & Sedation.

After assessing the Steward awakening score, patients were shifted to the ward. Post-operative follow up for 24hrs was done; side effects if any were treated & recorded.

STATISTICAL ANALYSIS: The sample size was determined by power analysis performed by a pilot study. A sample size of 18 patients per group was required to detect a 20% change in heart rate and blood pressure between baseline and intubation time, with a power of 80% at the 5% significance level. Data are expressed as the mean ± standard deviation. One way ANOVA was used to compare the study groups and the control group and also to compare the variable before and after the intervention. Chi-square test was used to analyze the categorical data and for testing the association between the variables. A P value of less than 0.05 was considered statistically significant. The package SPSS 17.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis.

RESULTS: The groups were well-matched for their demographic data. No patient was excluded from the study. Except heart rate, all other baseline hemodynamic variables were similar in all three groups. Heart rate values were statistically significantly lower in the Dexmedetomidine 1µg/kg group at all-time intervals and extremely low after intubation when compared with Dexmedetomidine 0.5µg/kg group and the control group.

Similarly the values of SBP, DBP and MAP were also statistically lower at all-time intervals especially 1 minute after intubation in Dexmedetomidine 1µg/kg group when compared to other two groups. The control group had statistically higher values of HR, SBP, DBP and MAP at all-time intervals post induction when compared to both the dexmedetomidine groups. The Dexmedetomidine 1µg/kg group had a better control of heart rate and blood pressure than Dexmedetomidine 0.5 µg/kg group and significantly better than the control group.

	DEXMED 0.5	**DEXMED 1**	**CONTROL**	**P VALUE**
AGE	41.9+10.85	42.63+11.9	39.93+12.2	0.67
SEX(M+F)	16+14	18+12	17+13	0.7£
WEIGHT	67.26+10.89	60.76+8.91	64+9.4	0.58

Table 2: Patient Characteristics

£-male sex,

	GROUPS	**T_0**	**T_1**	**T_2**	**T_3**	**T_4**
HR	1	84±9.2	77±9.2	72±7.6	85±9.1	74±9.6
	2	84±8.9	71±8.1	65±8.8	71±6.0	72±6.0
	3	94+20.1	97+13.3	91+8.3	102+12.5	100+11.3
	P value	0.025(s)	0.00020(HS)	0.04342(S)	8.5×10^{-13}(HS)	0.0270(HS)
SBP	1	132±11.8	113±7.9	104±9.3	119±7.1	110±6.2
	2	132±11.8	117±12.7	102±6.1	113±8.0	105±8.0
	3	136+14.9	132+11.7	119+11	133+11.5	132+12.5
	P value	0.7(NS)	0.06914(NS)	0.00063(HS)	0.01154(S)	0.01679(S)

DOI: 10.14260/jemds/2014/3802

DBP	1	85±9.4	78±11.5	74±9.4	92±13.7	74±8.3
	2	86±8.8	74±9.2	68±5.3	72±4.6	70±6.9
	3	86+6.8	80+6.2	75.9+7.9	80+7.7	77+7.2
	P value	0.4(NS)	0.06040(NS)	0.00350(HS)	5.59×10^{-9}(HS)	0.01906(S)
MAP	1	100±6.4	89±7.6	84±7.2	101±9.1	86±6.1
	2	102±7.0	88±7.7	79±4.4	86±4.4	82±4.9
	3	100+9.9	97+8.3	89+9.3	97+10.6	93+9.4
	P value	0.6(NS)	0.2237(NS)	0.00273(HS)	1.57×10^{-10}(HS)	0.0049(HS)

Table 3: Heart Rate & Blood Pressure

NS-not significant, HS-highly significant, S-significant, HR-heart rate, SBP-systolic blood pressure, DBP-diastolic blood pressure, MAP-mean arterial pressure GROUP 1-Dexmedetomidine 1μg/kg, GROUP 2- Dexmedetomidine 0.5μg/kg, GROUP 3-control group.

DISCUSSION: The sequence of induction of anaesthesia, laryngoscopy and intubation are associated with marked haemodynamic changes and autonomic reflex activity which may be a cause of concern in many high risk patients.[4]

Laryngoscopy and intubation is associated with a rise in heart rate, blood pressure and incidence of cardiac arrhythmias. These potentially dangerous changes disappear within 5 minutes of laryngoscopy.[5,6] Although these responses of blood pressure and heart rate are transient and short lived they may prove to be detrimental in high risk patients especially in those with cardiovascular disease, increased intracranial pressure and anomalies of the cerebral blood vessels.

Many factors influence the cardiovascular changes associated with laryngoscopy and intubation. Age, drugs, type and duration of procedures, depth of anaesthesia, hypoxia, hypercarbia, etc., influence the pressor response.

The most significant laryngoscopic factor influencing cardiovascular responses is found to be the duration of laryngoscopy.[7] A linear increase in heart rate and mean arterial pressure during the first 45 seconds has been observed. Further prolongation has little effect. As the duration of laryngoscopy is normally less than 30 seconds, the results of studies in which it takes longer than this have less clinical relevance. The force applied during laryngoscopy has only minor effect.[7] In our study the duration of laryngoscopy and intubation was limited to 20 seconds.

Variation of heart rate changes decrease with increasing age. Young patients show more extreme changes.[7] Marked fluctuations in haemodynamic response are often seen in geriatric patients.[8,9] In our study, we selected an optimal age range of 18 to 60 years.

Patients on antihypertensive drugs may exhibit a decrease in pressor response. We excluded the patients on antihypertensive medications from our study.

A variable combination of drugs used for premedication, induction, relaxation and maintenance of anaesthesia can influence the sympathetic response to laryngoscopy and intubation.
For nearly two decades, α2 adrenergic agonists have been widely used by veterinarians to achieve dose- dependent sedation, analgesia and muscle relaxation in a variety of species.

The α_2 receptors are involved in regulating the autonomic and cardiovascular systems. α_2 receptors are located on blood vessels, where they mediate vasoconstriction, and on sympathetic

DOI: 10.14260/jemds/2014/3802

terminals where they inhibit norepinephrine release. α_2 receptors are also located within the central nervous system and their activation leads to sedation, a reduction of tonic levels of sympathetic outflow and an augmentation of cardiac-vagal activity.

This can result in a decrease in heart rate and cardiac output. The use of α_2 agonists in the perioperative period has been associated with reduced anesthetic requirements and attenuated heart rate and blood pressure responses to stressful events. In addition, α_2 receptors within the spinal cord modulate pain pathways, thereby providing some degree of analgesia.[10,11,12]

It was observed that dexmedetomidine used in premedication suppresses the sympathetic activation which is due to the endotracheal intubation.[13] Güler et al. found that the increase in blood pressure and heart rate during the extubation is decreased and the quality of extubation is increased by dexmedetomidine.[14]

It was found in the study by Jaakola et al that, during the intubation blood pressure and heart rate is significantly reduced by 0.6 μg. kg^{-1} dexmedetomidine.[15] In Scheinin's study these parameters were also reduced by equal doses of dexmedetomidine.[16] In the other study which was done by Tezer et al. it is concluded that sympathetic responses during laryngoscopy and intubation were effectively reduced by dexmedetomidine 1 $\mu g.kg^{-1}h^{-1}$ and esmolol 250 μg/kg/min.[17]

Khan et al. demonstrated that heart rate, systolic and diastolic blood pressure were reduced by dexmedetomidine.[18] In another study on the patients undergoing vascular surgery, it was observed that in the recovery period dexmedetomidine infusion led to suppression of heart rate and plasma catecholamine levels.[19]

In this study dexmedetomidine 1 $\mu g.kg^{-1}$ and 0.5 $\mu g.kg^{-1}$, SAP, DAP, MAP and HR levels were significantly lower at 60 sec after induction and 5 min after intubation than baseline levels. But at 1 minute after laryngoscopy & intubation, these levels increased in all the three groups. But in 1 $\mu g.kg^{-1}$ group, the amount of increase in the vital parameter levels, 60 secs after intubation, was less when compared to 0.5 $\mu g.kg^{-1}$ and very much less than the control group was. Hence, it was found that dexmedetomidine very effective in suppressing the hemodynamic response to laryngoscopy and at dose of 1 $\mu g.kg^{-1}$ better than at dose 0.5 $\mu g.kg^{-1}$

In some studies it is observed that mean arterial pressure was decreased by low doses of dexmedetomidine (0.25-1 $\mu g.kg^{-1}$) and mean arterial pressure was increased transiently and heart rate was decreased significantly by high doses of (1-4 $\mu g.kg^{-1}$) dexmedetomidine.[10,11] Scheinin et al reported that the use of α_2 agonist leads to bradycardia.[16] Belleville et al found that dexmedetomidine which is given in two minutes the doses of 1-2 $\mu g.kg^{-1}$ causes' irregular ventilation and apnea episodes.[11] Ebert et al. didn't observe any apnea, airway obstruction and hypoxemia with bolus doses of dexmedetomidine in their study and they reported that depression of respiration may be seen due to deep sedation, for the reason that α_2 adrenergic agonists don't have active role on the respiration center.[10]

In another study in which the infusion of opioid and α_2 adrenergic agonists were compared, it was concluded that dexmedetomidine doesn't cause significant respiratory depression and it decreases the risk of apnea.[20] Hofer et al reported that dexmedetomidine seems to be a good choice in the critical patients in whom ventilation can be depressed with narcotics.[21]

In this study no hypotension or bradycardia were seen and no medical intervention was required. Also no significant respiratory depression, apnea, muscle rigidity or decrease in SpO_2 was seen in any patient.

DOI: 10.14260/jemds/2014/3802

In summary, these results suggest that to control haemodynamic responses to laryngoscopy and tracheal intubation, Dexmedetomidine is a better drug and 1 μg.kg^{-1} dose is more effective than Dexmedetomidine 0.5 μg.kg^{-1} dose without any significant side effects. The present clinical study has however following limitations:

- Adequate depth of anaesthesia and skeletal muscle relaxation was monitored only by clinical observations.
- Plasma norepinephrine levels were not measured.
- Extubation response, postoperative sedation and hemodynamic variations were not studied.
- Haemodynamic changes associated with two stages i.e. direct laryngoscopy and passage of the tracheal tube into the trachea were not studied separately.

CONCLUSION: It is concluded that pretreatment with dexmedetomidine as 10 minutes infusion prior to induction of anesthesia is a safe and effective method to attenuate the hemodynamic response to laryngoscopy and intubation. Also Dexmedetomidine at dose of 1μg/kg is more effective than 0.5 μg/kg in attenuating pressor response without any significant side effects.

REFERENCES:

1. Rose DK, Cohen MM. The airway: Problems and predictions in 18500 patients. Can J Anaesth 1991; 41: 372-83.
2. Reid LC, Brace DE. Irritation of the respiratory tract and its reflex effect upon heart. Surg Gynaec Obst 1940; 70: 157-62.
3. Textbook of pharmacology & physiology in anesthetic practice- Robert K Stoelting-4th ed: 340-45.
4. Black TE, Kay B and Healy TEJ. Reducing the hemodynamic responses to laryngoscopy and intubation. Anaesthesia 1984; 39: 883-87.
5. Russel WJ, Morris RJ, Frewin DB and Drew SE. Changes in plasma catecholamine concentrations during endotracheal intubation. Br J Anaesth 1981; 53: 837.
6. Onkar Singh, Kumar P, Swarn Kaur. Attenuation of the pressure response to laryngoscopy and tracheal intubation: Comparison of beta blockers and calcium channel blockers. Ind J Anaesth 1993; 41: 320-324.
7. Bucx MJL, Van Geel RTM, Scheck PAE and Stijnen T. Cardiovascular effects of forces applied during laryngoscopy. Anaesthesia 1995; 50: 17-22.
8. Splinter WM and Cervenko F. Hemodynamic responses to laryngoscopy and tracheal intubation in geriatric patients: effects of fentanyl, lidocaine and thiopentone. Can J Anaesth 1989; 36 (4): 370-6.
9. Ismail S, Azam SI, Khan FA. Effect of age on haemodynamic response to tracheal intubation. A Comparison of young middle aged and elderly patients. Anaesth Intensive care 2002; 30 (5): 608-614.
10. Ebert TJ et al- The effects of increasing plasma concentrations of dexmedetomidine in humans.2000; Aug; 93 (2): 382-94.
11. Bloor BC, Ward DS, Belleville JP, and Maze M. Effects of intravenous Dexmedetomidine in humans, II: Hemodynamic changes. Anesthesiology 1992; 77: 1134-42.

DOI: 10.14260/jemds/2014/3802

12. Aantaa RE, Kanto JH, Scheinin M, Kallio AMI, Scheinin H. Dexmedetomidine premedication for minor gynecological surgery. Anesth Analg 1990; 70: 407-13.
13. Erkola O, Korttila K, Aho M. Comparison of intramuscular dexmedetomidine and midazolam premedication for elective abdominal hysterectomy. Anesth Analg 1994; 79: 646-53.
14. Güler G, Akın A, Tosun Z, Eskitaşoğlu E, et al. During the extubation the effects of dexmedetomidine on cardiovascular changes and quality of extubation in the old patients undergoing cataract surgery. Turkish Journal of Anaesthesia 2005; 33: 18-23.
15. Jaakola ML, Ali-Melkkila T, Kanto J, Kallio A, Scheinin H, Scheinin M. Dexmedetomidine reduces intraocular pressure, intubation responses and anaesthetic requirements in patients undergoing ophthalmic surgery. Br J Anaesth 1992; 68: 570-575.
16. Scheinin B, Lindgren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenel responses to tracheal intubation and reduces the need for thiopentone and peroperative fentanyl. Br J Anaesth 1992; 68: 126-131.
17. Tezer E, Sarıcaoğlu F, Çelebi N, Aypar Ü. Comparison of esmolol and dexmedetomidine in induction of anesthesia about hemodynamic and anaesthetic requirements. Turkish Journal of Anesthesia 2005; 13 (4): 247-252.
18. Khan ZP, Munday IT, Jones RM, Thornton C, Mant TG, Amin D. Effects of dexmedetomidine on isoflurane requirements in healthy volunteers. 1: Pharmacodynamic and pharmacokinetic interactions. Br J Anaesth 1999; 83: 372-80.
19. Talke P, Chen R, Thomas B, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. Anesth Analg. 2000; 90 (4): 834-9.
20. Cortinez LI, Hsu YW, Sum-Ping ST, et al. Dexmedetomidine pharmacodynamics: Part I: Crossover comparison of the respiratory effects of dexmedetomidine and remifentanil in healthy volunteers. Anesthesiology 2004; 101 (5): 1066-76.
21. Hofer RE, Sprung J, Sarr MG, Wedel DJ. Anesthesia for a patient with morbid obesity using dexmedetomidine without narcotics. Can J Anaesth 2005; 52 (2): 176-80.

AUTHORS:

1. Smitha K. S.
2. Divya Shukla
3. Sathesha M.
4. Raghavendra Rao
5. Nethra S. S.
6. K. Sudheesh

PARTICULARS OF CONTRIBUTORS:

1. Senior Resident, Department of Anaesthesia, BMCRI.
2. Senior Resident, Department of Anaesthesia, BMCRI.
3. Assistant Professor, Department of Anaesthesia, BMCRI.
4. Professor & HOD, Department of Anaesthesia, BMCRI.
5. Professor, Department of Anaesthesia, BMCRI.
6. Associate Professor, Department of Anaesthesia, BMCRI.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Smitha K. S,
4061A, 1st Main,
2nd Cross, BSK 3rd Stage,
Bangalore-560085.
Email: smi9685@gmail.com

Date of Submission: 05/11/2014.
Date of Peer Review: 06/11/2014.
Date of Acceptance: 07/11/2014.
Date of Publishing: 11/11/2014.