Original Article

DOI: 10.7860/JCDR/2015/14624.6455

Comparison of Effects of Different Doses Dexmedetomidine on Inhibiting Tracheal Intubation-Evoked Haemodynamic Response in the Elderly Patients

GUAN ZHAN-YING[1], WANG CHANG-MING[2], TANG SHUAI[3],TONG LIN-LIN[4], HAN YU-FENG[5]

ABSTRACT

Background: Dexmedetomidine (DEX) is a selective α_2-adrenergic receptor agonist with anxiolytic and analgesic properties. In the present study, we aimed primarily to assess the effects of DEX on sedation, cognitive function and cardiovascular reflex responses before, during and after the tracheal intubation in the elderly patients.

Materials and Methods: Eighty patients undergoing elective abdominal surgery were randomly assigned to four Groups: Group A(saline, n=20), Group B (0.25μg/kg DEX, n=20), Group C (0.50μg/kg DEX, n=20) and Group D (1.00μg/kg DEX, n=20). With the constant speed infusion of saline and a loading different doses of DEX (diluted with saline to 50ml) for 10min respectively before induction of anaesthesia, the values of arterial pressure {systolic blood pressure (SBP), diastolic blood pressure (DBP)}, heart rate (HR) and bispectral index (BIS) at the time point of before pump DEX (T_0), at the end of infusing DEX (T_1), before tracheal intubation (T_2), at the moment of tracheal intubation (T_3) and 5min after trachea intubation (T_4) were observed, oxygen saturation (SPO_2) and the Modified Observers Assessment of Alertness/Sedation Scale (OAA/S) score were observed at the time of T_1 and T_0.

Results: Comparison among Groups, compared with Group A, SBP and DBP values in Group C at T_2 showed significant differences ($p<0.05$), SBP and DBP values in Group D at T_1, T_2 and T_4 indicated significant differences ($p<0.05$), HR values in Group D at T_1, T_2, T_3 and T_4 showed significant differences ($p<0.05$); Compared with Group A, BIS values in Group C at T_2 and T_3 indicated significant differences ($p<0.05$), BIS values in Group D at T_1, T_2, T_3 and T_4 showed significant differences ($p<0.05$); Comparison between T_3 andT_2, means of SBP, DBP and HR in Group A and in Group B showed significant differences ($p<0.05$); Group D showed significant differences in SPO_2 and (OAA/S) betweenT_1 and T_0 ($p<0.05$).

Conclusion: Comparison within Groups and between Groups in different doses DEX, the present result showed that 0.5μg/kg DEX had an effective inhibition, without respiratory depression, on tracheal intubation evoked cardiovascular response in the elderly patients.

Keywords: Cardiovascular responses, Sedation

INTRODUCTION

Dexmedetomidine (DEX), which is widely used in anaesthesia, possesses properties of sedation, anxiolysis and analgesia without the development of respiratory depression [1-3]. DEX has a relatively high ratio of α_2/α_1-activity, the α_2:α_1 binding selectivity ratio of DEX is 1620:1; therefore DEX is a highly specific and selective α_2-adrenergic receptor agonist [4-6]. Previous studies that had used intravenous boluses of DEX showed decrease in BP and cardiac output (CO) after small boluses (0.25-1μg/kg), which were associated with decrease in serum norepinephrine concentration, nevertheless the response to larger boluses (1-4 μg/kg) had been a transient increase in BP and sometimes reflex bradycardia [5,7,8]. Tracheal intubation can lead to increase in BP, HR and plasma catecholamine concentrations [9,10]. It is important for anaesthesiologists to attenuate sympathoadrenal response during tracheal intubation in patients, especially for the elderly patients, because the reserve capacity of cardiovascular structure and function, drug metabolism are increasingly impaired in the elderly patients [11]. There were few studies on how different doses DEX affect tracheal intubation-related haemodynamic response in the elderly patients. Therefore, the current study aimed to seek a more suitable bolus dose of dexmedetomidine for the elderly patients to maintain haemodynamic stability during tracheal intubation with fewer complications.

MATERIALS AND METHODS

Patients

The study was approved by the Ethical Committee of Branch Hospital of Hunnan, The General Hospital of Shen Yang Military Region on 10 May 2013. The study was conducted in accordance with the principles set forth in the Helsinki Declaration. After institutional review board approval and patients' written informed consent, eighty patients who underwent elective abdominal surgery from14 May 2013 to 1 December 2014, were randomly assigned to four Groups according to the table of random number: Group A (control Group) (saline, n=20), Group B (0.25μg/kg DEX, n=20), Group C (0.50μg/kg DEX, n=20) and Group D (1.00μg/kg DEX, n=20).

The patient characteristics of the 80 patients included in our study were summarized in [Table/Fig-1], scored as American Society of Anaesthesiologists (ASA) I–II, aged 65-78 years and weighed 50-87 kg. All patients, who were ≥65-year-old, had no history of mental illness, allergy, liver disease, bradycardia, use of α_2 agonists or antagonists and drug abuse.

Anaesthesia Procedures

All patients were premedicated to provide intramuscular injection of 0.5mg atropine and 0.1g luminal sodium 30min before surgery. Patients of Group A (n=20) were managed with the constant speed infusion of saline before induction of anaesthesia, patients of Group B (n=20), Group C (n=20) and Group D (n=20) were managed with a loading dose of 0.25μg/kg, 0.50μg/kg and 1.00μg/kg DEX (200μg DEX was diluted with saline to 50ml) respectively before tracheal intubation, the time of constant speed infusion in each Group was 10min.

Operative and anaesthesia procedures were performed in the operating theater, patients who were given a mask oxygen inhalation for 10 min before administration were monitored by cardiorespiratory monitoring which included electrocardiogram for the determination

of heart rate (HR), oscillometric blood pressure measurement {systolic blood pressure (SBP), diastolic blood pressure (DBP)}, finger tip pulse oximetry for SpO_2 monitoring simultaneously and determination of sedation/hypnosis was monitored via BIS. During the induction of general anaesthesia, sedation, analgesia and muscle relaxant consisted of the combination of propofol, fentanyl and vecuronium. After saline or different doses DEX were used, anaesthesia was induced with propofol 1.5mg.kg^{-1} over 20s followed immediately by 3.0μg.kg^{-1} fentanyl. Vecuronium 0.2mg.kg^{-1} was given 20s after completing the bolus administration of fentanyl. As soon as patient lost consciousness (unconsciousness was measured by eyelash reflex), ventilation was attempted via a mask. Then an appropriately sized tracheal tube was inserted. Anaesthesia was maintained with remifentanil (0.15μg.kg^{-1}.min^{-1}), propofol (50μg.kg^{-1}.min^{-1}) via continuous pump infusion combined with 1-2% sevoflurane.

SBP, DBP, HR and BIS at the time point of before pump DEX (T_0), at the end of infusing DEX (T_1), before tracheal intubation (T_2), at the moment of tracheal intubation (T_3) and 5min after tracheal intubation (T_4) were observed, SPO_2 and the Modified Observer's Assessment of Alertness/Sedation Scale (OAA/S) score [Table/Fig-2] [12] were observed at the time of T_0 and T_1.

STATISTICAL ANALYSIS

Data were presented as mean ± SD (mean of standard deviations). Variables were tested about normal distribution with Kolmogorov-Smirnov test and Q-Q plots. The parametric test of variance analysis in the above-mentioned subGroups of patients was assessed by Multivariate Repeated Measures ANOVA to determine dose, time effects, and interaction terms (response of each dose over time) for each measured variable. Demographic data were compared using Pearson's χ^2 test. $p<0.05$ was considered to be statistically significant.

RESULTS

The patient characteristics (gender, age and weight) of the eighty patients included in our study are summarized in [Table/Fig-1]. There were no significant differences in the patient characteristics of different subGroups.

As shown in [Table/Fig-3a-c], compared with Group A, means of SBP, DBP and HR in Group B did not show significant differences ($p>0.05$).

As shown in [Table/Fig-3a], SBP value in Group C (148.4±15.7mmHg) Vs Group A (101.2±10.8mmHg) at T_2 showed significant difference with $p<0.05$, SBP values in Group D (168.7±11.5mmHg) Vs Group A (152.6±11.7mmHg) at T_1 with $p<0.05$, and in Group D (157.8±14.5mmHg) Vs Group A (101.2±10.8mmHg) at T_2 with $p<0.05$ and in Group D (105.4±13.2mmHg) Vs Group A (120.0±10.7mmHg) at T_4 with $p<0.05$ indicated significant differences.

As shown in [Table/Fig-3b&c], DBP in Group C (73.6±15.4mmHg) Vs Group A (60.4±11.5mmHg) at T_2 showed significant difference with $p<0.05$, DBP values in Group D (90.6±11.9mmHg) Vs Group A (77.9±11.5mmHg) at T_1 with $p<0.05$, Group D (80.3±12.8mmHg) Vs Group A (60.4±11.5mmHg) at T_2 with $p<0.05$ and Group D (54.2±9.3mmHg) Vs Group A (63.7±9.8mmHg) at T_4 with $p<0.05$ indicated significant differences. HR values in Group D (57.4±9.9bpm) Vs Group A (69.1±11.2bpm) at T_1 with $p<0.05$, in Group D (55.3±11.1bpm) Vs Group A (62.3±9.8bpm) at T_2 with $p<0.05$, in Group D (59.1±10.9bpm) Vs Group A (72.1±12.4bpm) at T_3 with $p<0.05$ and in Group D (59.8±11.6bpm) Vs Group A (65.5±11.6bpm) at T_4 with $p<0.05$ showed significant differences.

Comparison between means of SBP in Group A at T_3 (155.3±11.7mmHg) Vs T_2 (101.2±10.8mmHg) with $p<0.05$, DBP at T_3 (80.1±12.3mmHg) Vs T_2 (60.4±11.5mmHg) with $p<0.05$ and HR at T_3 (72.1±12.4bpm) Vs T_2 (62.3±9.8bpm) with $p<0.05$ and in Group B means of SBP at T_3 (150.8±15.2mmHg) Vs T_2 (107.9±12.3bpm) with $p<0.05$, DBP at T_3 (76.3±10.6mmHg), Vs T_2 (66.8±14.6mmHg) with $p<0.05$ and HR at T_3 (73.3±10.8bpm) Vs T_2 (61.2±9.1bpm) with $p<0.05$ showed significant differences.

As indicated in [Table/Fig-4], when compared with Group A, BIS values at T_2 in Group C (50.3±4.3) Vs Group A (65.2±2.4) with $p<0.05$ and at T_3 in Group C (48.4±3.3) Vs Group A (60.7±2.5) with $p<0.05$, BIS values at T_1 in Group D (92.2±1.2) Vs Group A (98.7±0.8) with $p<0.05$, at T_2 in Group D (45.7±3.5) Vs Group A (65.2±2.4) with $p<0.05$, at T_3 in Group D (42.3±2.7) Vs Group A (60.7±2.5) with $p<0.05$ and at T_4 in Group D (37.7±3.7) Vs Group A (49.3±3.6) with $p<0.05$ showed significant differences.

As seen in [Table/Fig-5], SPO_2 in Group D at T_1 (90.1±2.8%) Vs T_0 (98.5±0.8%) with $p<0.05$ and OAA/S at T_1 (3.5±0.5) Vs T_0 (5.0±0.0) with $p<0.05$ showed significant differences.

	Group A	Group B	Group C	Group D	p
Age (years)	67.7±7.8	68.1±11.1	68.1±10.3	67.6±12.6	0.192
Gender (Male/Female)	12/8	11/9	10/10	12/8	0.906
Weight (kg)	65.7±10.2	66.6±14.1	66.2±9.5	66.3±11.5	0.185

[Table/Fig-1]: Control Group (Group A) and study Groups (Group B, C and D) were paired in their general characteristics ($\bar{x}$ ± s, n)

Response	Score level
Responds readily to their name spoken in a normal tone	5 (Alert)
Lethargic response to their name spoken in a normal tone	4
Response only after their name is called loudly and/or repeatedly	3
Response only after name spoken with mild prodding or shaking	2
Does not respond to mild prodding or shaking	1
Does not respond to noxious stimuli (Trapezius squeezing)	0

[Table/Fig-2]: Responsiveness Scores of the Modified Observer's Assessment of Alertness/Sedation Scale (OAA/S)

	Group A	Group B	Group C	Group D
T_0 SBP	154.7±13.2	155.8±14.1	153.9±12.6	152.1±10.7
T_1 SBP	152.6±11.7	153.3±13.6	156.7±13.4	168.7±11.5#
T_2 SBP	101.2±10.8	107.9±12.3	148.4±15.7#	157.8±14.5#
T_3 SBP	155.3±11.7*	150.8±15.2*	152.7±16.3	160.2±15.5
T_4 SBP	120.0±10.7	124.4±12.2	126.8±12.7	105.4±13.2#

[Table/Fig-3a]: Means of SBP (mmHg) were at different times in patients of each Group ($\bar{x}$ ± s).
Multivariate analysis of variance (MANOVA).SBP, systolic blood pressure.
#There was a statistical difference between the Groups at the evaluation during the procedure.
*There was a statistical difference between T_3 and T_2 in four Groups.
# $p < 0.05$, study Groups (Group B, C and D) vs. control Group (Group A).
* $p < 0.05$, T_3 vs. T_2 in the same Group

	Group A	Group B	Group C	Group D
T_0 DBP	78.2±14.3	78.9±10.2	77.8±12.4	79.2±10.6
T_1 DBP	77.9±11.5	77.6±14.8	76.7±15.4	90.6±11.9#
T_2 DBP	60.4±11.5	66.8±14.6	73.6±15.4#	80.3±12.8#
T_3 DBP	80.1±12.3*	76.3±10.6*	76.5±14.2	78.9±13.6
T_4 DBP	63.7±9.8	66.4±11.7	64.3±10.5	54.2±9.3#

[Table/Fig-3b]: Means of DBP (mmHg) were at different times in patients of each Group ($\bar{x}$ ±s).
Multivariate analysis of variance (MANOVA).DBP, diastolic blood pressure.
#There was a statistical difference between the Groups at the evaluation during the procedure.
*There was a statistical difference between T_3 and T_2 in four Groups.
# $p < 0.05$, study Groups (Group B, C and D) vs. control Group (Group A).
* $p < 0.05$, T_3 vs. T_2 in the same Group

	Group A	Group B	Group C	Group D
T_0 HR	70.5±10.4	69.7±9.6	68.1±12.2	71.2±12.6
T_1 HR	69.1±11.2	65.3±8.9	65.8±10.7	57.4±9.9#
T_2 HR	62.3 ± 9.8	61.2 ± 9.1	62.9±10.4	55.3±11.1#
T_3 HR	72.1±12.4*	73.3±10.8*	67.2±16.3	59.1±10.9#
T_4 HR	65.5±11.6	64.3±11.2	63.7±12.5	59.8±11.6#

[Table/Fig-3c]: Means of HR (bpm) were at different times in patients of each Group ($\bar{x}$±s). Multivariate analysis of variance (MANOVA). HR, indicates heart rate.
#There was a statistical difference between the Groups at the evaluation during the procedure.
*There was a statistical difference between T_3 and T_2 in four Groups.
# $p < 0.05$, study Groups (Group B, C and D) vs. control Group (Group A).
*$p < 0.05$,T_3 vs. T_2 in the same Group.

	T_0	T_1	T_2	T_3	T_4
Group A	98.4±1.1	98.7±0.8	65.2±2.4	60.7±2.5	49.3±3.6
Group B	98.5±0.8	97.5±1.3	63.2±3.5	61.3±4.9	50.4±4.3
Group C	97.9±0.5	97.1±0.7	50.3±4.3#	48.4±3.3#	47.4±2.8
Group D	98.4±0.9	92.2±1.2#	45.7±3.5#	42.3±2.7#	37.7±3.7#

[Table/Fig-4]: Means of BIS were at different times in patients of each Group ($\bar{x}$±s).
#There was a statistical difference between the Groups at the evaluation during the procedure.
# $p < 0.05$, study Groups (Group B, C and D) vs. control Group (Group A)

		Group A	Group B	Group C	Group D
T_0	SPO_2	97.9±0.7	98.2±0.5	98.0±1.2	98.5±0.8
	OAA/S	5.0±0.0	5.0±0.0	5.0±0.0	5.0±0.0
T_1	SPO_2	98.4±0.7	97.6±1.0	97.2±2.1	90.1±2.8#
	OAA/S	5.0±0.0	5.0±0.0	4.6±0.2	3.5±0.5#

[Table/Fig-5]: Means of SPO_2 (%) and OAA/S(score) were at T_1 and T_0 in patients of each Group ($\bar{x}$ ± s).
Oxygen saturation, SPO_2; the Modified Observer 's Assessment of Alertness/ Sedation Scale, OAA/S.
#There was a statistical difference between T_1 and T_0 in four Groups.
#$p < 0.05$,T_1 vs. T_0 in the same Group

DISCUSSION

Tracheal intubation is associated with increase in arterial pressure, heart rate and plasma catecholamine concentrations, which are due to intense sympathetic discharge caused by stimulation of upper respiratory tract [9,13,14]. Intubation can lead to an average increase in blood pressure by 40-50% and 20% increase in heart rate [15], although transient hypertension and tachycardia have little effect on young patients, the haemodynamic changes may be life threatening in vulnerable patients, patients who are volume depleted, vaso-constricted, or have severe heart block, such as elderly patients because aging is associated with structural and functional changes in the cardiovascular and cerebrovascular systems, which would affect myocardial and cerebral perfusion with advancing age [5]. Therefore, it is important in the elderly patients to avoid significant haemodynamic changes during tracheal intubation. Various techniques and drugs like, deepening level of anaesthesia, topical and IV Lignocaine, adrenergic blockers, vasodilators like, alpha blockers, and opioids were used to prevent tracheal intubation-related haemodynamic changes [14]. Although previous study showed that it was effective for the elderly patients in attenuating the BP and HR responses to tracheal intubation with fentanyl 1.5μg/kg to 3.0 μg /kg administered 3-4 minutes prior to tracheal intubation [14], previous study indicated that doses in excess of 5 μg /kg showed late onset of side effects and smaller doses of ≤2 μg /kg did not completely abolish the cardiovascular response [13].

DEX is a highly selective α_2 adrenergic agonist with anxiolytic, sedative, sympatholytic and analgesic sparing properties [16-18]. Some studies reported that DEX can offer better endoscopy scores, lower recall of intubation and greater patient satisfaction, with minor haemodynamic side effects during awake fiberoptic nasotracheal intubation [19,20], other studies reported that administration of DEX can improve haemodynamic stability in patients undergoing tracheal intubation [5,10,14], it meant that DEX was an elective method to prevent intubation-related responses. Intravenous use of DEX in the perioperative period can decrease serum catecholamine levels and blunt the haemodynamic response to tracheal intubation [2,7,10,21]. However, ventilatory depression, a transient increase in arterial pressure, decreases of arterial pressure, HR and cardiac output were related to varying DEX doses and infusion speed [2,10,22-24]. Few studies focused on different doses DEX on the effect of haemodynamics in the elderly patients undergoing tracheal intubation. Based on the previous studies, we decided to observe haemodynamic changes, BIS and OAA/S values in the elderly patients to seek an appropriate dose of DEX with fewer complications.

The present study indicated that 0.25μg/kg DEX did not prevent tracheal intubation-related haemodynamic changes, 0.50μg/kg DEX and 1.00μg/kg DEX attenuated this sympathoadrenal response and provided haemodynamic stability during tracheal intubation. However, 1.00μg/kg DEX led to decrease in SPO_2, a transient increase in arterial pressure and decrease in HR soon after the use of DEX, and a decrease in arterial pressure at 5min after tracheal intubation. Compared with control Group, OAA/S score indicated significant difference in Group D, many patients could be aroused to the verbal commands and then could return to sleep like state when not stimulated, which was accorded with the previous study [24,25]. Although the previous study mentioned that no patient had any end-tidal CO_2 evidence of respiratory depression during awake fiber-optic intubation [17], we believed that it was only a relative point of view, the loading dose played an important role in affecting breathing. It was a fact that the decrease in SPO_2 appeared in many elderly patients, it might mean that breathing was affected by the loading dose of 1.00μg/kg DEX. However, the adverse reaction mentioned above generally did not happen in 0.50μg/kg DEX Group. It meant that a loading dose of 0.50μg/kg DEX with the completion of 10min intravenous infusion was relatively safe and effective in preventing intubation-related responses.

CONCLUSION

In conclusion, the present study indicates that giving a loading dose of 0.25μg/kg DEX does not serve as a very useful anaesthesia adjuvant to control haemodynamic stress response to intubation in the elderly patients, 1.00 μg/kg DEX significantly suppresses the tracheal intubation-related cardiovascular responses, however, it brings a transient increase in arterial pressure, a decrease in arterial pressure subsequently, decrease in HR and a change in consciousness after administration. In comparison, we used dose of 0.50μg/kg DEX that were predicted to have few cardiovascular effects, but still be sufficient to prevent tracheal intubation-evoked haemodynamic response in the elderly patients.

REFERENCES

[1] Chattopadhyay U, Mallik S, Ghosh S, Bhattacharya S, Bisai S, Biswas H. Comparison between propofol and dexmedetomidine on depth of anaesthesia: A prospective randomizedtrial. *J Anaesthesiol Clin Pharmacol*. 2014;30(4):550-54.
[2] Venn RM, Hell J, Grounds RM. Respiratory effects of dexmedetomidine in the surgical patient requiring intensive care. *Crit Care.* 2000;4(5):302-08.
[3] Hwang W, Lee J, Park J, Joo J. Dexmedetomidine versus remifentanil in postoperative pain control after spinal surgery:a randomized controlled study. *BMC Anaesthesiol.* 2015;15:21.
[4] Hall E, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. *Anesth Analg.* 2000;90(3):699-705.
[5] Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. *Indian J Anaesth.* 2011;55(4):352-57.
[6] Virtanen R, Savola JM, Saano V, Nyman L. Characterisation of selectivity, specificity and potency of medetomidine as an α^2-receptor agonist. *Eur J Pharmacol.* 1988;150(1-2):9-14.

[7] Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenousdexmedetomidine in humans: II. Haemodynamic changes. *Anaesthesiology.*1992;77(6):1134-42.
[8] Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma concentrations of dexmedetomidine in humans.*Anaesthesiology*.2000 ;93(2):382-94.
[9] Shribman AJ. Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. *Br J Anesth.* 1987;59(3):295-99.
[10] Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. Effect of dexmedetomidine on Haemodynamic responses to laryngoscopy and intubation: perioperative haemodynamics and anaesthetic requirements. *Drugs RD.* 2006;7(1):43-52.
[11] Priebe HJ. The aged cardiovascular risk patient. *Br J Anaesth.* 2000;85(5): 763-78.
[12] Bae JY, Choi do Y, Woo CH, Kwak IS, Mun SH, Kim KM. The BIS and haemodynamic changes in major burn patients according to a slow infusion of propofol for induction. *Korean J Anaesthesiol*. 2011;60(3):161-66.
[13] Hosalli V, Es A, Hulkund SY, Joshi C. Comparative efficacy of different doses of fentanyl on cardiovascular Responses to laryngoscopy andtracheal intubation. *J Clin Diagn Res.* 2014;8(9):GC01-03.
[14] Kovac AL. Controlling the haemodynamic response to laryngoscopy and endotracheal intubation. *J Clin Anesth.* 1996;8(1):63-79.
[15] Bruder N, Ortega D, Granthil C. Consequences and prevention methods of haemodynamic changes laryngoscopy and intratracheal intubation. *Ann Fr Anesth Reanim.*1992;11(1):57-71.
[16] Manne GR, Upadhyay MR, Swadia V. Effects of low dose dexmedetomidine infusion on haemodynamic stress response, sedation and post-operative analgesia requirement in patients undergoing laparoscopic Cholecystectomy. *Indian J Anaesth.* 2014;58(6):726-31.
[17] Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: A review of clinical applications. *Curr Opin Anaesthesiol.* 2008;21(4):457-61.
[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(3):372-80.
[19] Hu R, Liu JX, Jiang H. Dexmedetomidine versus remifentanil sedation during awake fiberoptic nasotracheal intubation: a double-blinded randomized controlled trial. *J Anesth.* 2013;27(2):211-17.
[20] Scher C, Gitlin M. Dexmedetomidine and low-dose ketamine provide adequate sedation for awake fibreoptic intubation. *Can J Anesth.* 2003;50(6):607-10.
[21] Isik B, Arslan M, Özsoylar O, Akçabay M. The effects of α_2-adrenergic receptor agonist dexmedetomidine on haemodynamic response in direct laryngoscopy. *Open Otorhinolaryngol J.* 2007;107(5):5-11.
[22] Belleville JP, Ward DS, Bloor BC. Effects of intravenous dexmedetomidine in humans: I. Sedation, ventilation and metabolic rate. *Anaesthesiology.* 1992;77(6):1125-33.
[23] Pandharipane P, Ely EW, Maze M. Dexmedetomidine for sedation and perioperative management of critically ill patients. *Semin Anesth Periop Med Pain.* 2006;25(2):43-50.
[24] Haselman MA. Dexmedetomidine: A useful adjunct to consider in some high-risk situations. *AANA J.* 2008;76(5):335-39.
[25] Fernandes ML, Oliveira WM, Santos Mdo C, Gomez RS. Sedation for electroencephalography with dexmedetomidine or chloral hydrate: a comparative study on the qualitative and quantitative Electroencephalogram pattern. *J Neurosurg Anaesthesiol.* 2015;27(1):21-5.

PARTICULARS OF CONTRIBUTORS:
1. Faculty, Department of Anesthesiology, Branch Hospital of Hunnan, The GeneralHospital of Shen Yang Military Region, Shen Yang, China.
2. Faculty, Department of Anesthesiology, Liao Ning Provincial People's Hospital, Shen Yang, China.
3. Faculty, Department of Anesthesiology, An Shan Maternal and Children's Hospital, An Shan, China.
4. Faculty, Department of Anesthesiology, Branch Hospital of Hunnan, The GeneralHospital of Shen Yang Military Region, Shen Yang, China.
5. Faculty, Department of Anesthesiology, Branch Hospital of Hunnan, The GeneralHospital of Shen Yang Military Region, Shen Yang, China.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Guan Zhan-Ying,
Faculty, Department of Anesthesiology, Branch Hospital of Hunnan, The GeneralHospital of Shen Yang Military Region
Shen Yang 110016, China.
E-mail:gzhanying@yeah.net

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Apr 23, 2015**
Date of Peer Review: **Jul 03, 2015**
Date of Acceptance: **Jul 23, 2015**
Date of Publishing: **Sep 01, 2015**