Medico Research Chronicles

ISSN No. 2394-3971

Original Research Article

A PROSPECTIVE, RANDOMIZED CONTROLLED STUDY OF INTRAVENOUS DEXMEDETOMIDINE ON 0.5% HYPERBARIC BUPIVACAINE USED IN SPINAL ANAESTHESIA

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Submitted on: February 2017
Accepted on: February 2017
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Abstract

Background and aims: Local anesthetics along with adjuvants like epinephrine, phenylephrine, adenosine, magnesium sulfate, sodium bicarbonate, neostigmine and alpha-2 agonists like clonidine, dexmedetomidine have been used intrathecally to prolong the duration of spinal anesthesia. Clonidine and dexmedetomidine, used intravenously are also known to prolong the duration of the spinal anesthesia.We proposed this study to evaluate the effect of intravenous dexmedetomidine on 0.5% bupivacaine spinal anesthesia.

Methods: In this prospective, double-blind, randomized controlled clinical trial, 60 patients aged 18 to 60 years, ASA grade 1 or 2 posted for planned elective surgery for more than two hours duration under spinal anesthesia.study design: The patients were randomly allocated into 2 groups, after giving spinal anesthesia with 0.5% bupivacaine. Group D received a loading dose of 1 ug/kg Dexmedetomidine and Group C received an equivalent quantity of normal saline. The duration of sensory and motor blockade, hemodynamic parameters, postoperative analgesia, time of rescue analgesia, sedation scores and side effects were studied. Results: The duration of sensory and motor block was significantly prolonged in dexmedetomidine group as compared to control group ($p<0.001$) Haemodynamic parameters like pulse rate, blood pressure was significantly lower in dexmedetomidine group. Mean time for first request of rescue analgesic in postoperative period was significantly longer in D group (5.43 hrs) as compared to C group (2.43 hrs) ($p <0.001$) Sedation scores were significantly higher in dexmedetomidine group [4.3+0.47] as compared to control group [2+0.0] ($p<0.001$) Side effects like shivering, nausea, and vomiting were less in D group Conclusion: Intravenous dexmedetomidine significantly prolongs the duration of sensory and motor block of bupivacaine spinal anaesthesia, provides excellent sedation and good postoperative analgesia

Keywords: dexmedetomidine, spinal anesthesia, intravenous, postoperative analgesia.

Introduction

Spinal anesthesia is a commonly used technique in anesthetic practice for gynecological, lower abdominal, pelvic, and lower limb surgeries. Local anesthetics along with adjuvant like epinephrine, phenylephrine, adenosine, magnesium sulfate, sodium bicarbonate, neostigmine and α-2 agonists like clonidine, dexmedetomidine have been used intrathecally[1] to prolong the duration of spinal anesthesia. Alpha- 2 agonists like clonidine and dexmedetomidine are also used intravenously to prolong the duration of the spinal anaesthesia[2,3,4,5,6] Dexmedetomidine has been used intravenously within one hour after the spinal block and it was found that it prolonged bupivacaine spinal anesthesia for approximately one hour without adverse effect[7]Dexmedetomidine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective α-2 receptor agonist activity. Few studies have shown the efficacy of intravenous dexmedetomidine in prolonging prilocaine/bupivacaine/ropivacaine spinal anesthesia in addition to providing good sedation, postoperative analgesia and prevent shivering. Our study is designed to evaluate the effect of intravenous dexmedetomidine on spinal anesthesia with 0.5% bupivacaine

Materials and Methods

With institutional ethics committee approval [IEC/IRB NO:1/2013(D1)]a randomized, double-blind, prospective study was done on 60 patients of ASA status (1 or 2) aged between 18- 60 years for planned elective surgery of more than two hours duration under spinal anesthesia. Patients were randomly allocated into 2 groups, **Group D** (Dexmedetomidine)received loading dose of 1 µg/kg of dexmedetomidine intravenously by infusion pump over 10 mins followed by maintenance dose, 0.5 ug/kg/her until the end of surgery and **Group C**(control) received equivalent quantity of normal saline as loading and maintenance intravenously by infusion pump. Patients with skin infection at the puncture site, coagulopathy, hypersensitivity to local anesthetics, asthma, cardiac, renal, hepatic or CNS disorders, and pregnant females were excluded from the study. Patients on calcium channel blockers, ACE inhibitors, alpha-2 agonists, sedative medications, opioids, and antidepressants were also excluded. The detailed pre-anaesthetic checkup was done, procedure explained, written, valid, informed consent was obtained.In the operation theater, adequate starvation status was confirmed. Monitors attached and baseline heart rate (HR), blood pressure (B.P), electrocardiogram and peripheral arterial oxygen saturation (SPO_2) were obtained. An intravenous line was secured and an infusion of ringer's lactate was started. Premedicationdone with Ondansetron(4 mg) and iv Ranitidine(50mg) Under all aseptic precautions, spinal anesthesia was given at L3-L4/L2-L3 using 25G Quincke's spinal needle with 3ml of 0.5% hyperbaric bupivacaine. Vital parameters (HR, BP, SPO_2, RR), onset and level of sensory and motor block was noted. After 20 min patients allocated to group D received a loading dose of 1 µg/kg of dexmedetomidine intravenously by infusion pump over 10 mins followed by a maintenance dose, 0.5 ug/kg/hr until the end of surgery and group C patients received an equivalent quantity of normal saline as loading and maintenance intravenously by the infusion pump. Vital parameters were recorded every 5 mins. intraoperatively and for 30 mins. postoperatively in postanaesthesia care unit (PACU), the Sensory blockade was checked and the time taken to the highest level of sensory blockade, two dermatomal regression from the maximum level and regression to S1 level was noted. Sensory blockade was assessed every 5 mins for first 10 mins and thereafter every 15 mins intraoperatively

and post-operatively till complete regression of the block.Motor blockade was assessed by Modified Bromage Scale every 5 mins after giving spinal and start of surgery and thereafter every 15 mins till the end of surgery and postoperatively.Time taken for the motor blockade to reach Modified Bromage Scale 3 and regression of motor blockade to Modified Bromage Scale 0 was noted. Fentanyl 1 µg/kg was given to patients who required additional analgesia.The level of sedation was evaluated both intro and postoperatively every 15mins using Ramsay Level of Sedation Scale*

Hypotension (SBP<100 mm Hg or > 20% fall from the baseline value) bradycardia(HR<60)and postoperative complications like nausea and vomiting were noted and treated appropriately. Number of patients requiring supplemental analgesia (1 µg/kg body wt of fentanyl) intraoperatively and time for the first request for postoperative analgesia were noted

Statistical Analysis:

At the end of the study decoding of patients, data was done and comparison between the two groups was done with all values expressed as the mean ± standard deviation (SD) or numbers and percentages. The means of the continuous variables (Age, BMI, and duration of surgery) were compared using analysis of variance ANOVA, while the demographic data for the categorical variables (sex, ASA class) were compared using chi-square test, a p value of <0.05 was considered statistically significant.

Observation and Results:

All 60 patients operated under spinal anesthesia with 0.5% hyperbaric bupivacaine completed the study protocol and were included in the analysis.The demographic data (age, gender, weight, ASA status and duration of surgery) were comparable in both the groups and there was no statistically significant difference between them

Intraoperative and postoperative hemodynamic parameters, respiratory rate, oxygen saturation, Ramsay sedation score, postoperative analgesia and side effects were compared between Dexmedetomidine Group (Group D) and Control Group (Group C)

A) Haemodynamic Parameters

1. Pulse Rate:

Mean intraoperative pulse rate was significantly lower in dexmedetomidine group (51.10) as compared to control group (68.77) (p=0.00) Lowest intraoperative pulse rate was significantly lower in D group (36) as compared to C group (44) as seen in **Table- 2** Significantly higher number of patients in D group (9/30) had transient intraoperative pulse rate <50 beats/min as compared to C group (1/30). However, 8 (26.7%) patients in D group and 1(3.3%) patient in C group required intravenous atropine for treatment of bradycardia (p=0.00) The mean postoperative pulse rate was significantly lower in D group (58.43) as compared to C group (77.9) (p= 0.00)The lowest postoperative pulse rate is lower in D group (54)as compared to C group (66)**Table- 1.**

Table No 1: Comparison of pulse rate in both groups

Study Parameters	Group D		Group C		p-value
	Mean	Std.Dev.	Mean	Std.Dev.	
PR Baseline	74.53	6.57	76.77	4.21	0.122
PR Intraop	51.10	7.14	68.77	6.89	0.000
PR Postop	58.43	2.34	77.90	5.50	0.000

*PR- Pulse Rate

2. Systolic Blood Pressure:
There was a significant difference in SBP in both the groups. Mean intraoperative SBP was lower in D group (101.37) as compared to C group (104.10) (p = 0.346) Lowest intraoperative SBP was significantly lower in D group (75) as compared to C group (82) Significantly higher number of patients in D group (17/30) has SBP <100 mm Hg intraoperatively as compared to C group (8/30)There was no significant difference in mephentermine requirement in both groups (p =0.228) Mean postoperative SBP was significantly lower in D group (113.53) as compared to C group (120.07) (p value=0.00) These findings are as per **Table-2**

Table No.2: Comparison of MSBP in both groups

Study Parameters	**Group D**		**Group C**		**p-value**
	Mean	**Std deviation**	**Mean**	**Std deviation**	
MSBP Baseline	129.57	6.72	126.67	5.81	0.079
MSBP Intraop	101.37	12.54	104.10	9.55	0.346
MSBP Postop	113.53	6.64	120.07	5.32	0.000

* MSBP – Mean Systolic Blood Pressure

3. Diastolic Blood Pressure:
Mean intraoperative DBP was lower in D group (64.73) as compared to C group (71.13) (p <0.001) Mean postoperative DBP was significantly lower in D group (63.7) as compared to C group (72.2) (p <0.001) as seen in **Table 3**

Table No 3: Comparison of MDBP in both the groups

Study Parameters	Group D		Group C		p-value
	Mean	Std.Dev.	Mean	Std.Dev.	
MDBP Baseline	80.63	5.24	82.83	3.90	0.070
MDBP Intraop	64.73	5.69	71.13	3.21	0.000
MDBP Postop	63.70	4.91	72.20	3.06	0.000

* MDBP- Mean Diastolic Blood Pressure

B) Oxygen Saturation (SPO_2) and Respiratory Rate: There was no significant difference in intraoperative and postoperative oxygen saturation(p =0.235). and respiratory rate(p =0.143)

C) Ramsay Sedation Score:
Intraoperative Ramsay sedation score was significantly higher in D group (4.3) as compared to C group (2) (p<0.001) Maximum score in D group ranged from 4 to 5 whereas in C group was 2. Mean postoperative Ramsay sedation score was significantly higher in D group (2.17) as compared to C group (2) (p=0.019) as seen in **Table 4.**

Table No 4: comparison of Ramsay sedation score in both study groups

Study Parameter	Group D		Group C		p-value
	Mean	Std.Dev.	Mean	Std.Dev.	
MRSS Baseline	2.00	0.00	2.00	0.00	
MRSS Intraop	4.30	0.47	2.00	0.00	0.000
MRSS Postop	2.17	0.38	2.00	0.00	0.019

* MRSS- Mean Ramsay Sedation Score
1: Patient anxious, agitated/ restless
2: Patient responds to commands
3: Asleep but with brisk response to light glabellar tap or loud auditory stimulus
4: Asleep, sluggish response to light glabellar tap or loud auditory stimulus
5: Asleep, no response

D) Duration of sensory and motor blockade:
The duration of sensory blockade, duration for 2 dermatomal regression of sensory blockade and the duration for motor block regression to Modified Bromage scale 0 were significantly prolonged in dexmedetomidine group as compared to control group (p<0.001) The highest level of sensory blockade was significantly higher in dexmedetomidine group (p <0.001) There was no difference in the time for attaining the highest level of sensory blockade, time is taken for the motor blockade to reach Modified Bromage Scale* 3 between both the groups. The motor and sensory blockade in both groups is summarized in **Table 5 and 6**

Table No 5: Comparison of duration of sensory and motor blockade

Study Parameters	Control D		Control C		p value
	Mean	Std.Dev.	Mean	Std.Dev.	
Time for attaining highest level(min)	11.90	0.76	11.77	0.57	0.444
Duration of sensory blockade(Mins)	268.37	16.34	173.47	7.36	0.000
Duration of 2 dermatomal regression of sensory blockade(mins)	142.37	9.89	102.43	13.23	0.000
Duration of motor blockade to reach MBS3(mins)	5.15	0.78	4.93	0.97	0.352
Duration of motor block regression to MBSMSS0(min)	218.43	14.62	131.43	7.21	0.000
Time for first request of rescue analgesic (hrs)	5.34	0.46	2.43	0.42	0.000

MBS: Modified Bromage Scale
0: Patient is able to move hip, knee & ankle
1: Unable to move hip but able to move knee & ankle
2: Unable to move hip & knee but able to move ankle
3: Unable to move hip, knee & ankle

Table 6: Comparison of highest level of sensory blockade in study groups

Highest Level Of Sensory Block		Group		Total
		C	D	
T5	COUNT	0	5	5
	PERCENT	0.0%	16.7%	8.3%
T6	COUNT	0	25	25
	PERCENT	0.0%	83.3%	41.7%
T7	COUNT	16	0	16
	PERCENT	53.3%	0.0%	26.7%
T8	COUNT	14	0	14
	PERCENT	46.7%	0.0%	23.3%
TOTAL	COUNT	30	30	60
	PERCENT	100.0%	100.0%	100.0%
CHI-SQUARE TEST	VALUE	DF	P-VALUE	ASSOCIATION IS
PEARSON CHI-SQUARE	60.000	3	0.000	SIG

E) Intraoperative fentanyl requirement:

None of the patients in group D required fentanyl intraoperatively as compared to 2 (6.7%) patients in group C (p = 0.150)

F) postoperative analgesia:

Mean time for the first request of rescue analgesia in postoperative period was significantly longer in D group (5.43 hrs) as compared to C group (2.43 hrs) (p<0.001)

G) Side effect profile:

postoperative shivering was noted in 3 (10%) patients of C group as compared to none in D group (p = 0.076), nausea and vomiting was noted in 1 (3.3%) patient of C group as compared to none in D group (p value-0.313)

Discussion

Different drugs like epinephrine, phenylephrine, adenosine, magnesium sulphate, sodium bicarbonate, neostigmine and α-2 agonists like clonidine, dexmedetomidine have been used as adjuvants to local anaesthetics to prolong the duration of spinal anaesthesia[1]Clonidine and dexmedetomidine, used intravenously are known to prolong the duration of the spinal anaesthesia[2] Recent studies have shown the efficacy of both intrathecal and intravenous dexmedetomidine in prolonging spinal anaesthesia. Dexmedetomidine is a more suitable adjuvant to spinal anesthesia compared to clonidine as it has more sedative and analgesic effects due to its more(seven to ten times) selective alpha α-2 receptor agonist activity [8] Systemic and intrathecal injection of dexmedetomidine produces analgesia by acting at the spinal level, laminae VII, and VIII of ventral horns. The drug also acts at locus ceruleous and dorsal raphe nucleus to produce sedation and analgesia. This supraspinal action explains the prolongation of spinal anesthesia after intravenous dexmedetomidine

In our study, the time for attaining highest level of sensory block is comparable in dexmedetomidine (11.90 + 0.76mins**)** and control groups (11.77 + 0.57mins**)** The median highest cephalad level of sensory block T4 [T3 – T8] was attained in 15 min in dexmedetomidine and control groups in a similar study by Whizar-Lugo et al[3]The highest level of sensory block was higher in dexmedetomidine group [T 5] compared to control group [T7] (p< 0.001) in our study. This observation is also comparable to the

study done by Kaya et al[9] They observed sensory block to be higher in dexmedetomidine group [T 4.6 + 0.6] than control group [T 6.4 +0.8] (p< 0.001) In our study time for two dermatomal regression of sensory blockade was significantly prolonged in dexmedetomidine group D[142.37 + 9.89 mins] compared to control group C[102.43 + 13.23] (p< 0.001). Significant prolongation in mean time for two dermatomal regression of sensory blockade was also reported by others [Kaya et al [9]-145 + 26 min vs 97 + 27 mins (p< 0.001), Tekin et al[5]-148.3 mins vs 122.8 mins (p< 0.001) in dexmedetomidine and control groups respectively] Similarly Hong et al [10]reported that the mean time to two-segment regression was prolonged in dexmedetomidine group [78 mins vs 39 mins for cold, 61 mins vs 41 mins for pinprick for dexmedetomidine group and control group respectively] Similar results were reported by Elcıcek et al[4] The duration of sensory blockade i.e. time for regression to S1 dermatome was significantly prolonged in dexmedetomidine group [268.37 + 16.34 mins] compared to control group [173.47 + 7.36] (p< 0.001) in our study. Significant prolongation in mean duration of sensory blockade in dexmedetomidine group was also reported by others [Al Mustafa et al[2]-261.5 ± 34.8 min vs 165.2 ± 31.5 min (p< 0.05), Whizar-Lugo et al–[3]208±43.5 mins vs 137±121.9 mins (p= 0.05) in dexmedetomidine and control groups respectively]

In our study there was no significant difference in time taken for motor blockade to reach modified Bromage Scale 3 in both the groups [5.15 + 0.78 mins in dexmedetomidine group compared to 4.93 + 0.97 mins in control group (p= 0.352)] However, the regression time to reach the modified Bromage Scale 0 was significantly prolonged in dexmedetomidine group [218.43 + 14.62 mins] compared to control group [131.43 + 7.21 mins] (p< 0.001) Delay in motor block regression to Bromage Scale 0 was also reported in previous studies [Al Mustafa et al [2]- 199 ± 42.8 min in vs 138.4 ± 31.3 min (p< 0.05), Whizar-Lugo et al [3]- 191±49.8 minsvs172±36.4 (p- not significant), Tekin et al[5]- 215 mins vs 190.8 mins (p< 0.001) for dexmedetomidine group and control group respectively] Elcıcek et al [4]and Hong et al[10]also found that complete resolution of motor blockade was significantly prolonged in dexmedetomidine group. But contrary to all the above studies, Kaya et al [9] reported no significant prolongation in the duration of motor block in dexmedetomidine group compared to control group.

In our study, intraoperative pulse rate was significantly lower in dexmedetomidine group [51.1+ 7.14] as compared to control group (68.77 + 6.89) (p<0.001) The lowest mean heart rate after subarachnoid block was significantly lower in dexmedetomidine group [36] as compared to control group (44) (p< 0.001). Significantly higher proportion of patients in dexmedetomidine group (9/30- 30%) had bradycardia (heart rate < 50) as compared to control group (1/50-2%)In the study done byTekinet al[5] the mean heart rate was significantly lower in dexmedetomidine group [70.4] as compared to control group (77.63) at 20 minutes (p= 0.02) which was comparable to our study.Higher incidence of bradycardia in dexmedetomidine group (16.66%) compared to control group (8.3%) (p= 0.46) was reported by Al Mustafa et al[2] Higher incidence of bradycardia seen can be explained by the longer duration of surgeries in our study groups (138 + 32.89)mins in control group and (136.77+ 26.43)mins in dexmedetomidine group requiring higher total dose of dexmedetomidine compared to the study done by Al Mustafa et al [2] (42.8 + 7.5)mins in control group and (45.1 + 8.3)mins in dexmedetomidine group even though the study protocol of loading and maintenance dose of dexmedetomidine were same. Whizar-Lugo et al [3] reported a higher incidence of bradycardia in the

dexmedetomidne group (32%) compared to control group (20%) Atropine requirement was more in dexmedetomidine group [8/30-26.67%] as compared to control group [1/30-3.33%] (p= 0.011) in our study. Atropine requirement was found to be significantly higher in dexmedetomidine group in other studies [Tekin et al–[5] 30%vs 6.6% (p< 0.001), Hong et al [10]-24.0% vs. 3.8% in dexmedetomidine and control groups respectively]. Similar results were reported by Elcıcek et al[4] Contrary to above studies Al Mustafa et al [2] reported no significant difference in atropine requirement between dexmedetomidine(9%) and control groups (0%) (p=0.65).

In our study intraoperative systolic blood pressure (SBP) after the spinal block was lower in dexmedetomidine group [101.37+12.54] as compared to control group [104.10 +9.55] (p=0.346). Lowest intraoperative SBP after spinal block was significantly lower in dexmedetomidine group (75) as compared to control group (82) A significantly higher number of patients in dexmedetomidine group [17/30(56.67%)] had lowest SBP >20% of baseline value as compared to control group [8/30(26.67%)]. The postoperative SBP was significantly lower in dexmedetomidine group [113.53+6.64] as compared to control group [120.07+5.32] (p<0.001). Previous studies have shown that the hypotensive effect of dexmedetomidine persists in the intraoperative as well as in the postoperative period[11,12]Eliceck et al [4] reported a significant decrease in mean arterial pressure after 20, 25, and 30 min after dexmedetomidine infusion as compared to control group. Contrary to above studies and our study, Al Mustafa et al [2] and Tekin et al [5] reported no significant difference in mean arterial pressures in dexmedetomidine and control groups. In our study, there was no significant difference in the number of patients requiring mephentermine for management of hypotension in both the groups [16.67% vs 6.67% in dexmedetomidine and control groups respectively (p= 0.228)] Similarly, Tekin et al[5] reported no significant difference between groups in the number of patients who received ephedrine to treat hypotension. No significant difference in the incidence of hypotension was reported by others [Al Mustafa et al[2]- 0% vs 20% (p = 0.15) Whizar-Lugo et al [3] - 8% vs 4% in dexmedetomidine and control groups respectively]

Despite providing good sedation, dexmedetomidine does not cause significant respiratory depression, providing wide safety margins[13]In our study, there was no significant difference in the respiratory rates between both the groups, both Intra and postoperative period comparable to similar studies by Al-Mustafa et al[2]

In our study, intraoperative Ramsay sedation scores were significantly higher in dexmedetomidine group [4.3+0.47] as compared to control group [2+0.0] (p<0.001) In dexmedetomidine group maximum sedation score was 5 and in control group was2 with a mean of 2. There was significant difference in sedation scores between the groups in the postoperative period [Dexmedetomidine group (mean -2.17) vs Control group (mean- 2) (p =0.019)] Ramsay sedation score was 2 in all patients in control group and ranged from 2 to 5 in dexmedetomidine group in the study done by Al-Mustafa et al[2] In their study the maximum score was 5 in 12% of patients, 4 in 79% of patients and 3 in 4% of patients. The maximum mean score of sedation [3.96 + 0.55] was attained 30 min after starting dexmedetomidine infusion. Hong et al [10] noted that the median sedation scores during surgery were 4 in the dexmedetomidine group and 2 in the control group (p< 0.001) A significantly higher average sedation score in dexmedetomidine group was also reported by others[4,5,8,14]

Dexmedetomidine inhibits the release of substance P from the dorsal horn of the spinal cord, leading to primary analgesic

effects[15] Dexmedetomidine was found to be effective in providing postoperative analgesia in our study. The time to first request for postoperative analgesic was significantly prolonged in dexmedetomidine group [5.34 + 0.46 hours] as compared to control group [2.43 + 0.42 hours] ($p< 0.001$) Similarly, Hong et al [10] noticed that postoperative pain intensity was lower and the mean time to first request for post-operative analgesia was longer in the dexmedetomidine group compared to the control group [6.6 hrs vs. 2.1hrs]. Kaya et al [9] in their study observed that dexmedetomidine increased the time to the first request for postoperative analgesia ($p< 0.01$) compared with midazolam and saline) and decreased analgesic requirements ($p< 0.05$). Whizar-Lugo et al [3] in their study noticed that the time to first request for postoperative analgesic in dexmedetomidine group was [220 + 30 mins] significantly prolonged as compared to control group [150 + 20 min] ($p< 0.05$)

Also, studies done by Anil Thomas, M. V. S. Satyaprakash, et al concluded that the continuous infusion of dexmedetomidine results in more advantages than just a bolus dose. They suggest using only the maintenance dose of intravenous dexmedetomidine after subarachnoid blockade for prolonging the duration and achieving sedation[16]

Similarly, studies done by SS Harsoor, D Devika Rani, Bhavana Yalamuru, K Sudheesh, and SS Nethra showed that IV supplementation of loading dose of dexmedetomidine 0.5 mcg/kg followed by infusion at 0.5 mcg/kg/h hastens the onset of sensory block and prolongs the duration of sensory block, analgesia and motor block with lesser incidence of bradycardia [17]

Clonidine and dexmedetomidine by inhibition of central thermoregulation and attenuation of the hyper adrenergic response to perioperative stress are known to prevent postoperative shivering[18] Dexmedetomidine markedly increases the range of temperatures not triggering thermoregulatory defenses. For these reasons dexmedetomidine, like clonidine, is likely to promote perioperative hypothermia and also prove to be an effective treatment for shivering[19]

In our study, none of the patients in dexmedetomidine group had postoperative shivering as compared to3 patients (10%) in control group ($p=0.076$) Similar results were reported by Tekin et al [5] (0% vs 30% in dexmedetomidine and control groups respectively).

No significant difference in the incidence of postoperative nausea and vomiting was noted between both the groups in our study [3.3% vs 0% in dexmedetomidine and control groups respectively ($p=0.313$)] Similar results were reported in previous studies[2,3]

Conclusion

IV dexmedetomidine provides excellent sedation and reduces analgesic requirement when used during general anesthesia. The aloading dose of 1 µg/kg of dexmedetomidine intravenously by infusion pump over 10 mins followed by a maintenance dose,0.5 ug/kg/hr until the end of surgery with subarachnoid block prolongs the duration of sensory block and motor block. Bradycardia and hypotension do occur, however, it is transient and responds to atropine and mephentermine. IV dexmedetomidine supplementation during SAB produces satisfactory arousable sedation without causing respiratory depression, prolongs the time of the first request of rescue analgesic and also prevents shivering, nausea, and vomiting

Acknowledgement

To respected dean madam and all the concerned staff members in the department of anesthesiology at ESI-PGIMSR, MGM Hospital, Parel, Mumbai-12

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