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Original Research Article

A Comparative Evaluation of Hyperbaric Bupivacaine with Clonidine versus Dexmedetomidine as an Adjuvant in Lower Abdominal and Lower Limb Surgeries at a Tertiary Care Centre

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Abstract

Background: Hyperbaric bupivacaine is most commonly used in spinal anaesthesia but cause motor block with late recovery as well as hemodynamic side effects. The use of adjuvant reduces the dose of bupivacaine and thereby the side effects which mandates the search for the better adjuvant.

Objective: To evaluate the efficacy and the safety of clonidine and dexmedetomidine as an adjuvant to intrathecal hyperbaric 0.5% bupivacaine.

Materials and Methods: Ninety patients in the age group between 20 years and 60 years of either sex belonging to ASA Grade-I and Grade-II posted for elective lower abdominal surgeries and lower limb surgeries were grouped randomly into three groups (n=30) where each group received 12.5 mg of 0.5% hyperbaric bupivacaine with 0.5 ml normal saline (control group), 50µg clonidine (clonidine group) and 5µg dexmedetomidine (dexmedetomidine group).

Results: Clonidine and Dexmedetomidine group showed an early onset of both sensory and motor blockade and a higher level of sensory blockade compared to Control group and duration of sensory, motor blockade and duration of analgesia is significantly prolonged in the clonidine and dexmedetomidine group compared to the control group which was found to be statistically significant ($p<0.001$).

Conclusion: Our study concludes that intrathecal dexmedetomidine is a better neuraxial adjuvant compared to clonidine for providing early onset of sensory and motor blockade, adequate sedation and prolonged postoperative analgesia.

Keywords: Bupivacaine, Clonidine, Dexmedetomidine, Spinal Anaesthesia.

Introduction

Spinal anaesthesia is most commonly used for patients who require surgical anaesthesia for procedures of known duration and involve surgery of infra-umbilical region. Spinal anaesthesia may be useful when patients wish to remain conscious or when comorbidities such as severe respiratory disease or difficult airway increases the risk of using general anaesthesia. [1]

Bupivacaine heavy is solely used for spinal anaesthesia, the duration of action of bupivacaine in dose of 5-20 mg is 240-380 min [2], although duration is long but not long enough to produce prolonged post-operative analgesia or for longer duration of surgery, additionally it produces hemodynamic disturbances in higher doses.

A number of adjuvants to local anaesthetics for spinal anaesthesia like opioids (fentanyl 10-30mcg [3] and buprenorphine 45mcg-60mcg [4]), midazolam 1-2mg [5], magnesium sulphate 50 mg [6], ketamine and neostigmine(10-50mcg)[7] has been used. Spinal opioids prolong the duration of analgesia, but they do have drawbacks of unpredictable respiratory depression, pruritus, nausea, vomiting and urinary retention [8] which requires urinary catheterisation and constant postoperative monitoring . Neostigmine benefits, of increasing the duration of block are limited by nausea, vomiting, bradycardia and in higher doses, lower extremity weakness. [9]

Hence there is requirement of an adjuvant which can be used along with local anaesthetics which can produce prolonged analgesia without the above said side effects of adjuvants.

Alpha-2 agonists like clonidine and dexmedetomidine act on pre-junctional and post-junctional α2 receptors in the dorsal horn of the spinal cord. Activation of presynaptic receptors reduce neurotransmitter release, whereas post-junctional receptor activation results in hyperpolarization and reduction of pulse transmission. Thus it has better properties to be used as an adjuvant with bupivacaine. In this regard various authors have studied clonidine [10] and dexmedetomidine [11]. Dexmedetomidine is approximately 10-fold more α2-selective than clonidine [12]. As little as 3µg of dexmedetomidine can prolong motor and sensory block without hemodynamic compromise. [13]

On the basis of above studies it was hypothesized that both clonidine and dexmedetomidine will produce a prolonged duration of postoperative analgesia compared to the control. There will be no difference regarding the duration of analgesia between clonidine and dexmedetomidine at equipotent doses.

Hence, this study was undertaken to evaluate and compare the effect of adding clonidine versus dexmedetomidine with hyperbaric 0.5% bupivacaine in spinal anaesthesia for elective lower abdominal and lower limb surgeries.

Materials and Methods:

The study was under taken in Nalanda Medical Hospital, Patna, Bihar during the period of December 2015 to October 2017, after obtaining ethical committee clearance as well as informed consent from all patient.

Ninety patients in the age group between 20-60 years of either sex belonging to ASA Grade-I and Grade-II posted for elective lower abdominal and lower limb surgery were grouped randomly into three groups (n=30). Randomization was done using simple sealed envelope technique.

Experimental groups

Control group: received 12.5mg of 0.5% hyperbaric bupivacaine with 0.5ml normal saline. Total volume of drug 3 ml.

Clonidine group:

received 12.5mg of 0.5% hyperbaric bupivacaine with 50µg clonidine.

Clonidine (Cloneon; 150 µg/ml of Neon laboratories) is diluted to 1.5 ml with normal saline and 0.5 ml (50 µg) of it will be added to 2.5 ml of 0.5% hyperbaric bupivacaine. Total volume of drug 3 ml.

Dexmedetomidine group:

received 12.5mg o f0.5% hyperbaric bupivacaine with 5µg dexmedetomidine. Dexmedetomidine (Dexem 50 µg/0.5 ml, of Themes Laboratories) 0.5 ml is diluted to 5 ml with normal saline and 0.5 ml of this is added to 2.5 ml of 0.5% hyperbaric bupivacaine. Total volume of drug 3 ml.

Inclusion criteria

Adult patients of both sexes aged between 20-60 years, belonging to ASA grade I and II without any co-morbid diseases scheduled for elective infra-umbilical surgery.

Exclusion criteria

- Patients belonging to the following classes:
- Age group less than 20 years and more than 60 years
- Patient belong to ASA grade III, IV and V
- Pregnant females
- Patients posted for emergency surgeries
- Patients with morbid obesity
- Patients having any absolute contraindications for spinal anaesthesia like raised intracranial pressure, severe hypovolemia, bleeding diathesis and local infection.
- Patients with co-morbid diseases like diabetes, hypertension and any other are excluded from the study.

Preoperative

Preoperative assessment was done for each patient. Patients were kept nil per oral for solids 6hrs and clear fluids 2 hrs before surgery. Patients were premedicated on the night before surgery with tablet ranitidine 150mg and tablet alprazolam 0.5mg. Intravenous line obtained with 18gauge cannula and preloaded with Ringer lactate 500ml half an hour before anaesthesia. The observer and the patient are blinded for the study drug.

Parameters assessed

1. Onset of sensory blockade.
2. Maximum level of sensory blockade.
3. Time taken for maximum sensory block.
4. Onset of motor block.
5. Quality of motor blockade was assessed by Bromage scale. [14]
6. Maximum level of motor blockade attained.
7. Time taken for Maximum level of motor blockade.
8. Two segments sensory regression time will be noted.
9. Total duration of analgesia will be noted.
10. Total duration of sensory blockade
11. Total duration of motor blockade.
12. Level of sedation was assessed by a modified Wilson sedation scale [15]
13. Total duration of surgery.
14. Hemodynamic monitoring included heart rate (HR), systolic blood pressure (SBP) diastolic blood pressure (DBP), mean arterial pressure (MAP), ECG, Respiratory rate and SpO2 hourly.

Statistical Analysis: A sample size of 25 patients per group was determined through power analysis (a ¼ 0.05; b ¼ 0.80) to detect an increase of 30 min in the time of a two- dermatome sensory regression with a standard deviation of 28 min. Considering the drop outs, 30 patients were selected for each group in our study. Results are expressed as the means and

standard deviations, medians and ranges, or numbers and percentages. The comparison of normally distributed continuous variables between the groups was performed using one-way analysis of variance (ANOVA) and, if appropriate, followed by the Bonferroni test for post hoc analysis. Nominal categorical data between study groups were compared using the chi-squared test or fisher's exact test as appropriate. Ordinal categorical variables and non-normal distribution continuous variables were dexmedetomidine or clonidine for supplementation of spinal bupivacaine compared using the Mann-Whitney U-test. $P < 0.05$ was considered to be significant. All the statistical calculations were done through SPSS 16.0 (2007) for windows.

Results and Discussion:

Demographic data: Demographic data comparing age, sex, height, weight shows no statistical difference among the groups ($p>0.05$).

Table 1 shows the age distribution of the patients in all the three groups. There is no significant difference in the age of patients between the groups. All the three groups were similar with respect to age distribution ($p>0.05$).

Table 1: Age Distribution

Age in years	Groups					
	Group Saline		Group C		Group D	
	No. of Pts	%	No. of Pts	%	No. of Pts	%
21-30	16	53.3	14	46.7	18	60.0
31-40	9	30.0	6	20.0	2	6.7
41-50	5	16.7	6	20	8	26.7
51-60	0	0	4	13.3	2	6.7
Total	30	100	30	100	30	100
Mean±SD	31.17±9.752		36.60±11.082		33.07±11.585	
Minimum age in years	20		20		20	
Maximum age in years	50		59		55	

All values are expressed as Mean ± SEM, *p<0.05 compared to Control.

Table 2: Effect of various groups on sensory block characteristics.

S. No.	Sensory Parameters	Groups (n=30)		
		Control	Clonidine	Dexmedetomidine
1.	Onset of sensory blockade (in mins)	2.8±0.66	1.43±0.50**	1.17±0.37**
2.	Time taken for maximum sensory blockade (in mins)	7.4±1.10	5.9±0.80*	5.2±0.71*
3.	Maximum level of sensory blockade (T4)	2/30	8/30	12/30
4.	Time taken for regression of sensory block by two segments (in mins)	79.46±10.1	136.33±10.9*	136.33±11.59*
5.	Time taken for sensory block to regress to S1 (in mins)	203.33±42.41	365±24.6*	396.16±30.61*
6.	Duration of analgesia (in mins)	191±22.9	342.33±28.12**	369.33±34.13**

All values are expressed as Mean ± SEM, *p< 0.05 and **p<0.01 compared to Control.

Table 3: Effect of various groups on motor block characteristics.

S. No.	Motor Parameters	Groups (n=30)		
		Control	Clonidine	Dexmedetomidine
1.	Onset of motor blockade	4±0.69	1.63±0.49**	1.13±0.346**
2.	Time taken for maximum motor blockade	6.57±0.9	6.43±1.04*	5.20±0.88*
3.	Duration of motor blockade	166.16±20.95	279±24.68**	303.66±35.95**

All values are expressed as Mean ± SEM, *p< 0.05 and **p<0.01 compared to Control.

A-Sensory block characteristics

1-Onset of sensory blockade

There is a statistically highly significant (p<0.01) in the onset of sensory blockade in clonidine group and in the dexmedetomidine group compared to the control group. (Table 2) Jain et al [16] showed the onset of sensory blockade 1.13 ± 1.64 min in clonidine group (15mcg) and 1.00 ± 0.00 min in dexmedetomidine group (10 mcg) and Saikia and Das [17] showed clonidine (30 µg) group to be 2.8 ± 0.75 minutes and that in dexmedetomidine group(3 µg) to be 2.6 ± 0.68 minutes which concurs with our study while Kanazi et al [13] showed higher values 7.6 ± 4.4 min in clonidine (30µg) group and 8.6 ± 3.7 min in dexmedetomidine group(3µg), which may be due to the lower amount of drug used in their studies.

2. Time taken for maximum sensory blockade

There is a statistically significant (p<0.05) decrease in the meantime taken for the maximum sensory blockade in the clonidine group and dexmedetomidine group compared to the control group.(Table 2) Sarma et al [18] found that time to reach maximum height of sensory block (min) is 6.2±0.8 in clonidine group and 5.6±0.7 in dexmedetomidine group which concurred with our study. Suthar, Bora and Tiwari [19] found that time to reach maximum height of sensory block (min)16 ± 3.85 in bupivacaine group, 14 ± 4.11 in clonidine group and 17 ± 4.51min in dexmedetomidine group, higher value obtained compared to our study, may be due to lesser dose of drug used (clonidine 30mcg and dexmedetomidine 3 mcg). Mahendru et al [20] found that time taken for highest level of sensory block attained at 10.1 ± 3.5 in bupivacaine group, 9.5 ± 3.0 clonidine group, 10.3 ± 3.3 min in dexmedetomidine group, higher values obtained in their study compared to our study, may be due to lesser dose of drug used (clonidine 30mcg and dexmedetomidine 5mcg).

3. Maximum level of sensory blockade achieved

There is no statistical significant (p>0.05) difference in the maximum level of sensory blockade in the clonidine group and dexmedetomidine group compared to the control group. (Table 2) Sarma et al [18] found that maximum level of sensory block attained is T4, which concurs with our study. Suthar, Bora and Tiwari [19] found that maximum height of sensory block (thoracic level) 6 ± 1.52 in control group, 6 ± 1.47 in clonidine group and 6 ± 1.155 in dexmedetomidine group, lower level of block achieved may be due to lower amount of drug used (clonidine 30 mcg and dexmedetomidine 3 mcg). Mahendru et al[20] found that maximum level of block attained is T6, which is less than our study, it may be due to the lesser dose of drug used (30µg clonidine and 5 µg dexmedetomidine).

4. The time taken for regression of sensory block by two segments

There is a statistically significant (p<0.05) increase in the meantime taken for regression of sensory block by two segments in clonidine group and

dexmedetomidine group compared to the control group.(Table 2) Sarma et al [18] found that the two segments regression time was 99.4 ± 28.938 min in control group, 120.00 ± 30.436 min in clonidine group and 139.8 ± 30.655 min in dexmedetomidine group, intergroup comparison between control and both the dexmedetomidine and clonidine group was significant which concurs with our study. Partani et al [21] found that the mean time for regression of sensory block by 2 segments was highest with dexmedetomidine 139.58+14.49 as compared to clonidine 122.46+18.55 and bupivacaine 100+13.43,the mean time taken for regression of 2 segment was significantly more for both dexmedetomidine and clonidine group compared to control group, which concurs with our study and also there is significant difference between clonidine and dexmedetomidine group which is not present in our study which can be possibly due to low dose of clonidine (30µg) used as compared to our study. Kanazi et al [13] found that the time taken for regression of sensory block by two segments to be 80±28 mins in control group, 101±37 mins in clonidine group (30 µg) and 122±37 mins in dexmedetomidine group (3µg), hence there is a significant prolongation of two segment regression compared to the control group which compares with our study. But compared to our study the time duration of regression is less and it could be due to less dose of drug used.

5. The time taken for sensory block to regress to S1

There is a statistically significant ($p<0.05$) increase in the mean- time taken for regression of sensory block to S1 in clonidine group and dexmedetomidine group compared to the control group. (Table 2) Partani et al [21] found the mean-time taken for regression of sensory block to S1 202.13±26.94mins in bupivacaine group, 284.73±26.72mins in clonidine group (30µg) and 299.94±29.31 min in dexmedetomidine group (5µg) and Sarma et al [18] found 199.8 ± 32.61 min in bupivacaine group, 278.6 ± 26.208 min in clonidine group and 306.6 ± 50.567 min in dexmedetomidine group concurring with our study. Kanazi et al [13] found that the time taken for regression of sensory block to S1 to be 190±48 mins in control group, 272±38 mins in clonidine group and 303±75 mins in in dexmedetomidine group, which is less than the value in our study, this could be due to the less doses of clonidine and dexmedetomidine used (30µg clonidine and 3µg dexem).

6. Duration of analgesia

There is a statistically highly significant ($p<0.01$) increase in the duration of analgesia in dexmedetomidine and clonidine group compared to the control group. (Table 2) Sarma et al [18] the found that the time of first rescue dose requested by patient was 204.8 ± 16.81 min in bupivacaine group, 309.6 ± 50.99 min in clonidine group and 336.8 ± 55.38 min in dexmedetomidine group, which concurs with our study. Suthar, Bora and Tiwari [19] found that, duration of analgesic effect of spinal anaesthesia was 204 ± 16.9 min in bupivacaine group, 309 ± 51.5 min in clonidine (30µg) group and 336 ± 55.9 min in dexmedetomidine (3µg) group, which concurs with our study. Das, Reang and Debnath [22] found that, the analgesic effect was 147.68 ± 10.11 min in bupivacine group, 176.62 ± 10.95 in clonidine group and 197.72 ±19.78 min in dexmedetomidine group, their study also resulted in prolongation of analgesic duration by dexmedetomidine and clonidine which concurs with our study but duration of prolongation does not concurred with our study which can be probably due to less dose of clonidine used (15µg).

B Motor block characteristics

1. Onset of motor blockade

There is a statistically highly significant ($p<0.01$) decrease in the mean time for onset of motor blockade in the dexmedetomidine group and clonidine group compared to the control group.

(Table 3) Raval and Chaudhary [23] found that, onset of motor block was 1.20 ± 0.21min in clonidine group and 1.32 ± 0.21min in dexmedetomidine group, which concurs with our study. Saikia and Das [17] found that, onset of motor block was 2.9 ± 0.78 in clonidine group and 2.66 ± 0.47 in dexmedetomidine group, the duration is slightly higher due to less dose of drug used (clonidine 30µg and dexmedetomidine 3µg). Jain et al[16] found that, onset of motor block was 2.2 ± 0.55 min in dexmedetomidine group and 2.7 ± 0.63 min in clonidine group, the onset time is slightly high which can be due to less dose of clonidine used (15 µg).

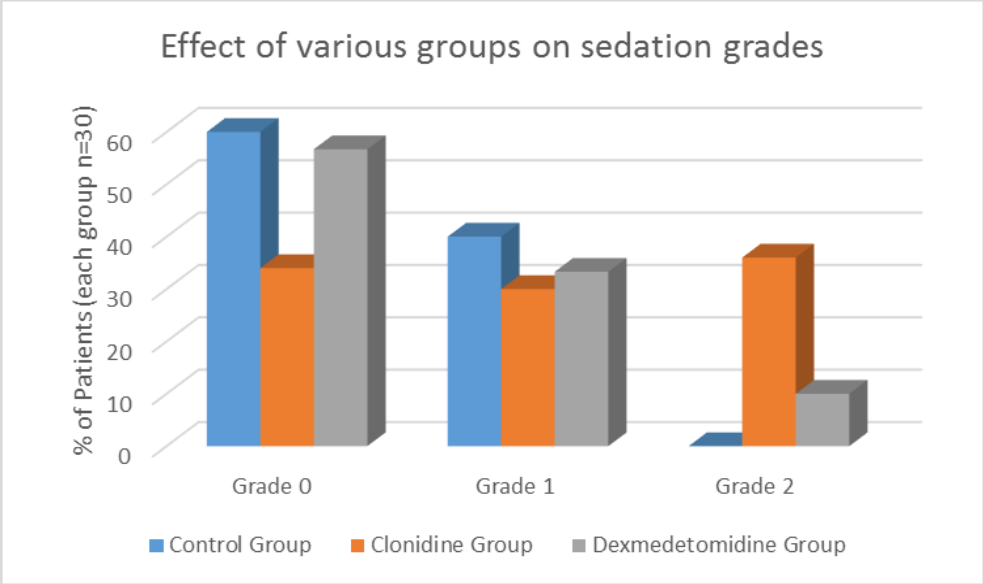
2. Time taken for maximum motor blockade

There is a statistically significant ($p<0.05$) decrease in the time taken for maximum motor blockade in dexmedetomidine and clonidine group compared to the control group. (Table 3) Das, Reang and Debnath [22] found that, the time taken for maximum motor block 7.06 ± 1.81 min in clonidine group and 6.94 ± 1.49 in dexmedetomidine group, which concurs with our study. Raval and Chaudhary [23] found that, the time taken for maximum motor block is 8.17 ± 2.12 in dexmedetomidine group and 9 ± 2.04 in clonidine group, which concurred with our study. Partani et al [21] found that, the time to reach maximum motor block was 15.23 ± 0.77 in bupivacaine group, 9.73 ± 0.47 in clonidine group and 10.38 ± 0.60 in dexmedetomidine group, the time to reach maximum motor block in their study was slightly higher probably due to lesser dose of clonidine used (30µg).

3. Duration of motor blockade

There is a statistically highly significant ($p<0.01$) increase in the duration of motor blockade in dexmedetomidine group and clonidine group compared to the control group. (Table 3) Singh and Shukla [24] found that, the regression time to bromage 0 is 172.11 ± 29.77 min for bupivacaine group, 231.93 ± 70.57 min for clonidine group and 309.93 ± 101.71 min for dexmedetomidine group, which concurs with our study. Anandani et al [25] showed the duration of motor block is 305.33±36.91 in clonidine group and 387.11±105.14 in dexmedetomidine group, which concurs with our study. Kanazietal [13] found that, the mean duration of motor blockadeis163 ± 47mins in control group, 216 ± 35minsin clonidine group and 250 ± 76mins in dexmedetomidine group which is less than the value in our study. This could be due to the less doses of clonidine and dexmedetomidine.

C. Sedation: There is a statistical significance ($p<0.05$)in mean sedation scores between control group and clonidine group and between control group and dexmedetomidine group. (Graph 1) There was no statistical significance between clonidine group and dexmedetomidine group. Sarma et al [18] found that in clonidine group 28 % has grade 1 sedation and 33 % had grade 2 sedation and in dexmedetomidine group 30 % has grade 1 sedation and 12 % has grade 2 sedation. Shukla et al[6] found that in clonidine group 31 % has grade 1 sedation and 34 % has grade 2 sedation and in dexmedetomidine group 28 % has grade 1 sedation and 15 % had grade 2 sedation. Das, Reang and Debnath [22] found in clonidine group 24 % has grade 1 sedation and 22 % has grade 2 sedation and in dexmedetomidine group 20 % has grade 1 sedation and 8% has grade 2 sedation. In our study we did not observe any evidence of respiratory depression, episodes of nausea, vomiting, shivering in any of the groups. None of the patients came back to us with backache, buttock pain or leg pain or any neurological deficit. This conformed with most of the studies.



Graph 1: Effect of various groups on sedation grades

D. Haemodynamic effects

1. Systolic blood pressure

In the control group we observed a maximum fall in mean SBP of 16.66 mmHg from mean basal SBP at 10th min, in the clonidine group it was 17.46mmHg at 40th min and in the dexmedetomidine group it was 18.66 mmHg at 20th min. There was no statistically significant difference in any of the three groups regarding fall in SBP. However it was found that there was a delay in maximum fall in SBP in the clonidine group compared to the dexmedetomidine group and the control group.

2. Diastolic blood pressure

In the control group we observed a maximum fall in mean DBP of 11.33mmHg from mean basal DBP at 20th min, in the clonidine group it was16.6 mmHg at 40th min and in the dexmedetomidine group it was 13.3 mmHg at10th min. There was no statistically significant difference in any of the three groups regarding fall in DBP. However it was found that there was a delay in maximum fall in DBP in the clonidine group compared to the dexmedetomidine group and the control group.

3. Mean arterial blood pressure

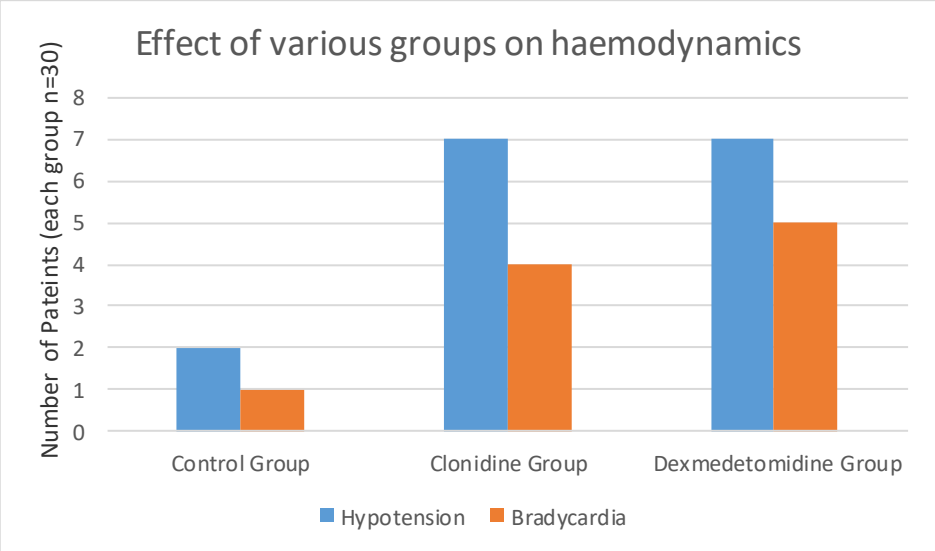
In the control group we observed a maximum fall in mean MAP of 12.2mmHg from mean basal MAP at 10thmin, in the clonidine group it was12.56mmHg at 30thmin and in the dexmedetomidine group itwas14.96mmHg at 30th min. There was no statistically significant difference in any of the three groups regarding fall in MAP. However it was found that there was a delay in maximum fall in MAP in the clonidine group and the dexmedetomidine group compared to the control group.

Two patients in control group, seven patients in clonidine group and seven patients in dexmedetomidine group developed hypotension. (Graph 2) which were easily managed with intravenous fluids and vasopressor. Anandani et al [25] found that 4 patients in dexmedetomidine group and 6 patients in clonidine group had significant hypotension which was easily treated. Chandra, Krishna and Singh [26] found that in clonidine group 9 /40 patients and 10/40 in dexmedetomidine group had hypotension. Suthar, Bora and Tiwari [19] found that hypotension in 2/25 of patients in bupivacaine group, 04 /25 in clonidine group and 2/25 in dexmedetomidine group, which concurred with our study.

4. Heart rate

In the control group we observed a maximum decrease in the mean heart rate of 7.8 bpm from basal value at 20th min, in the clonidine group it was 9.26 bpm at 30th min and in the dexmedetomidine group it was 15.33 bpm at10th min. There was no statistically significant difference ($p>0.05$) in any of the three groups regarding decrease in the mean heart rate. However it was found that there was a delay in maximum decrease in the mean heart rate in the clonidine group compared to the dexmedetomidine group and the control group.

5 in dexmedetomidine group, 4 patients in clonidine group and 1 patient in control group had bradycardia which is statistically not significant ($p>0.05$). (Graph 2) Bradycardia was easily reversed with 0.6mg intravenous atropine in all the patients. Anandani et al [25] found bradycardia in 6/30 patients in clonidine group and 3/30 patient in dexmedetomidine group. Chandra, Krishna and Singh [26] found bradycardia in 2/40 patients in clonidine and 1/40 patients in dexmedetomidine. Partani et al [21] found bradycardia in 0/50 patients in clonidine group and 5 /50 patient in dexmedetomidine group.



Graph 2: Effect of various groups on haemodynamic.

Conclusion

From the present study it can be concluded that intrathecal dexmedetomidine in the dose of 5µg or intrathecal clonidine in the dose of 50µg along with 2.5ml bupivacaine, 0.5% heavy, in patients undergoing elective lower abdominal surgeries, Decreases the onset time for sensory and motor blockade, produces higher level of sensory blockade, produces prolonged postoperative analgesia, sensory and motor blockade, produces sedation in which patients were asleep and easily arousable, and haemodynamic changes which could be easily managed. Dexmedetomidine and clonidine when used intrathecally along with bupivacaine significantly prolongs the duration of analgesia, both can be used as an adjuvant to bupivacaine but since dexmedetomidine is better in terms of onset and duration of sensory-motor block therefore dexmedetomidine is better adjuvant than clonidine.

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