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Effects of Intravenous Dexmedetomidine On 5% Hyperbaric Lidocaine (Xylocaine) Spinal Anaesthesia -A Placebo Controlled Randomized Controlled Trial

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Abstract:*The objective of the study was to evaluate the effect of intravenous dexmedetomidine on the duration of subarachnoid block and sedation in patients undergoing surgeries under spinalanaesthesia with 5% lidocaie. 80 ASA physical status I/II patients undergoing elective surgeries under spinal anaesthesia were randomized into two groups of 40 each. Immediately after subarachnoid block with 2 ml of 5% hyperbaric lidocaine, group D patients received a loading dose of 1 μg/kg of dexmedetomidine intravenously by infusion pump over 10 mins followed by a maintenance dose of 0.5 μg/kg/hr till the end of surgery whereas group C received an equivalent quantity of normal saline by infusion pump. Time taken for regression to Modified Bromage Scale 0, level of sensory block, two dermatomal regression of sensory blockade, duration of sensory block and intraoperative Ramsay sedation scores were higher in group D compared to group C (p values < 0.001). In conclusion, intravenous dexmedetomidine significantly prolongs the duration of sensory and motor block of lidocaine spinal anaesthesia with good hemodynamic stability.*

Keywords:*Dexmedetomidine, Hyperbaric lidocaine, Intrathecal, Ramsay sedation scale, Spinal anaesthesia*

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I. Introduction

Subarachnoid block is a commonly used technique in anaesthetic practice for gynaecological, lower abdominal, pelvic, and lower limb surgeries. Bupivacaine is appropriate for procedures lasting for 2 to 2.5hours. If the duration of surgery prolongs it may necessitate convertion to general anaesthesia or supplementation with an intravenous anaesthetic agent. To overcome this, adjuvants like epinephrine, phenylephrine, adenosine, magnesium sulphate, sodium bicarbonate, neostigmine and alpha$_2$ agonists like clonidine, dexmedetomidinehave been used intrathecally[1]. Clonidine and dexmedetomidine are also used intravenously to prolong the duration of spinal block[2-6]. Apart from sedation and analgesia they also decrease sympathetic tone and decrease the stress responses to surgery and anaesthesia.They produce sedation and anxiolysis by binding to pre synaptic alpha$_2$ receptors in locus ceruleus[6].

Locus coeruleus is among the one having highest densities of α2 receptors which is a predominant noradrenergic nucleus in the brain and an important modulator of vigilance. Activation of α2-adrenoceptor results in hypnotic and sedative effects in this site in the CNS. The locus coeruleus site for the descending medullospinal noradrenergic pathway is an important modulator of nociceptive neurotransmission. In this site, α2-adrenergic and opioidergic systems have common effector mechanisms, which indicates, dexmedetomidine hasa supraspinal site of action [(7)] .Thus, major sedative and antinociceptive effects of dexmedetomidine are due to its stimulation of the α2 adrenoceptors in the locus coeruleus. Moreover, studies in transgenic mice have identified that the α2Aadrenoceptor subtype is responsible for relaying the sedative and analgesic properties of dexmedetomidine [(8).] Dexmedetomidine is much more effective sedative and analgesic agent than clonidine due to its improved specificity for the α2A receptor, with much less α1 effects [(7)]. It has been used safely as premedication or as a sedative agent in patients undergoing surgical procedures under regional anesthesia[(9)]

Dexmedetomdine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective alpha 2A receptor agonist activity. Few studies have shown the efficacy of intravenous dexmedetomidine in prolonging prilocaine/ bupivacaine/ ropivacaine spinal anaesthesia in addition to providing good sedation and postoperative analgesia. Present study is designed to evaluate the effect of intravenous dexmedetomidine on 5% hyperbaric xylocaine spinal anaesthesia.

II. Material And Methods:

Source of data: This study was conducted in RIMS, Ongole80 cases of ASA grade I-II undergoing lower abdominal surgerieswere included in this study.Patients were divided in to two groups each consisting of 40 patients. This study was done after obtaining informed consent from the patients

Inclusion Criteria:

1) ASA grade I-II
2) Age 20--60 years
3) No association with co morbid conditions like diabetes, hypertension, asthma.
4) Surgeries less than 1.5 hours of duration

Exclusion Criteria:

1. ASA grade III-V
2. Uncooperative patients
3. Patients with hypersensitivity to local anesthetics.
4. Infection over the site of injection.
5. Bleeding diathesis.
6. Patients receiving Ca channel blockers/ACE inhibitors / Clonidine
7. Patients on Sedative medications/ Opioids/ Antideprassants in the week prior to surgery.

After securing IV (18G) access and monitoring as per ASA standards, patients are preloaded with 20 ml/kg of Ringer's lactate solution over 10min. A baseline recording of heart rate, NIBP, RR, SP02 were recorded. After ensuring the table in horizontal position the patient turned in lateral position with neck flexed and knees drawn up as far as possible. Under strict aseptic precautions 100 mg of hyperbaric 5% lidocaine of study drug is injected in the L3-L4 interspace with 23/ 25G quinke's spinal needle. Onset of peak sensory level and motor blockade are noted.NIBP, Heart rate, Respiratory rate & oxygen saturation are recorded immediately and after 5, 10, 15, 20 min & so on.

20 mins after subarachnoid block with 100mg of 5% hyperbaric lidocaine, group D patients willreceive a loading dose of 1 µg/kg of dexmedetomidine intravenously over 20 mins followed by a maintenance dose of 0.5 µg/kg/hr till the end of surgery whereas the other group (group C) will receive an equivalent quantity of normal saline as loading and maintenance dose intravenously and serves as control.

Sensory blockade will be checked with hypodermic needle in mid axillary line and the time taken forthehighest level of sensory blockade, two dermatomal regression from the maximum level and regression to S1 level will be noted. Sensory blockade will be assessed every 2 mins for the first 10 mins and thereafter every 15 mins during surgery and postoperatively. All the durations will be calculated considering the time of spinal injection as time 0. Motor blockade will be assessed by Modified Bromage Scale. Time taken for motor blockade to reach Modified Bromage Scale 4 and regression of motor blockade to Modified Bromage Scale 0 will be noted. Motor blockade will be assessed every 2 mins before the onset of the surgery and every 15 min in PACU.Hypotension (systolic blood pressure less than 90 mm Hg or more than 20% fall from base line value then treated with inj. mephentermine) & bradycardia(heart Rate<50/min , treated with inj. atropine) and post operativecomplications like nausea and vomiting will be noted and treated appropriately

The level of sedation was evaluated both intra operatively and post operatively every 15 mins using Ramsay Level of Sedation Scale till the patient is discharged from PACU. Excessive sedation was defined as score greater than 4/6.

Table 1: Modified Bromage scale

Grade	Criteria	Degree of block
0	Able to move the hip, knee and ankle	None
1	Unable to move the hip, but is able to move the knee and ankle	Partial 33%
2	Unable to move the hip and knee, but is able to move the ankle	Partial 66%
3	Unable to move the hip, knee and ankle	Complete paralysis

The level of sedation was evaluated using Ramsay Level of Sedation Scale [9].

Table 2: Ramsay sadation score

Scale	Level of sedation
1	Patient anxious, agitated, or restless
2	Patient cooperative, oriented, and tranquil alert
3	Patient responds to commands
4	Asleep, but with brisk response to light glabellar tap or loud auditory stimulus
5	Asleep, sluggish response to light glabellar tap or loud auditory stimulus
6	Asleep, no response

III.OBSERVATIONS AND RESULTS

This study was carried out on a total number of 80 patients operated under spinal anaesthesia. Demographic data, intraoperative and postoperative hemodynamics, Respiratory rate, Ramsay sedation score and side effects were compared between

Statistical analysis

The data obtained was entered in to Microsoft excel spreadsheet. The data was expressed in terms of percentages, mean and standard deviation (SD). The data was analysed by student's unpaired t test. A probability (p) value of less than or equal to 0.05 was considered as statistically significant

.**Demographic data:**

Age:

The mean age in the Group D was 41.975 + 12.658. Yrs. as compared to 41.7±10.78 yrs in the Group C and the difference was statistically no significant (P value-0.9169). There was statistically no significant difference in age distribution in both groups

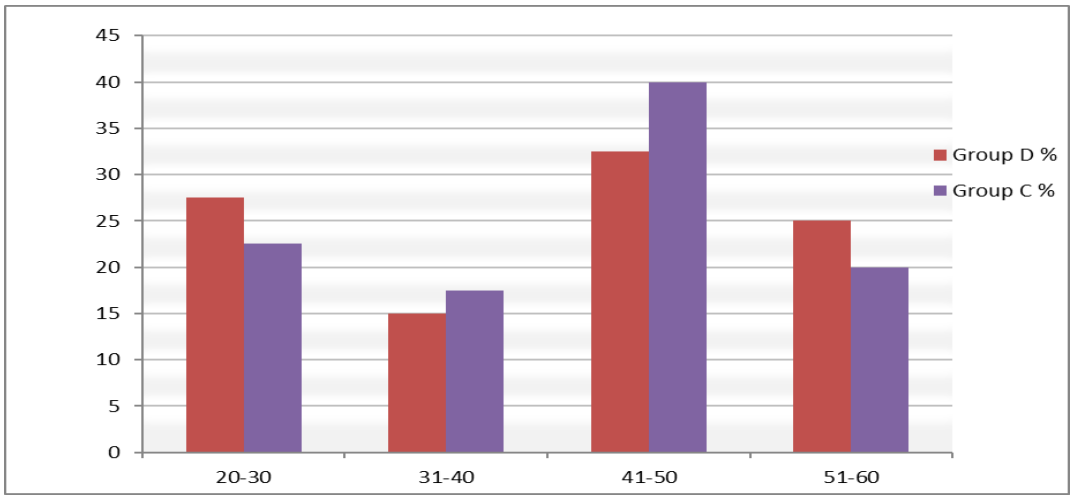

Fig1:Bar diagram showing distribution of age in both the groups

Gender:

There was no statistically significant difference between the two groups in gender distribution.

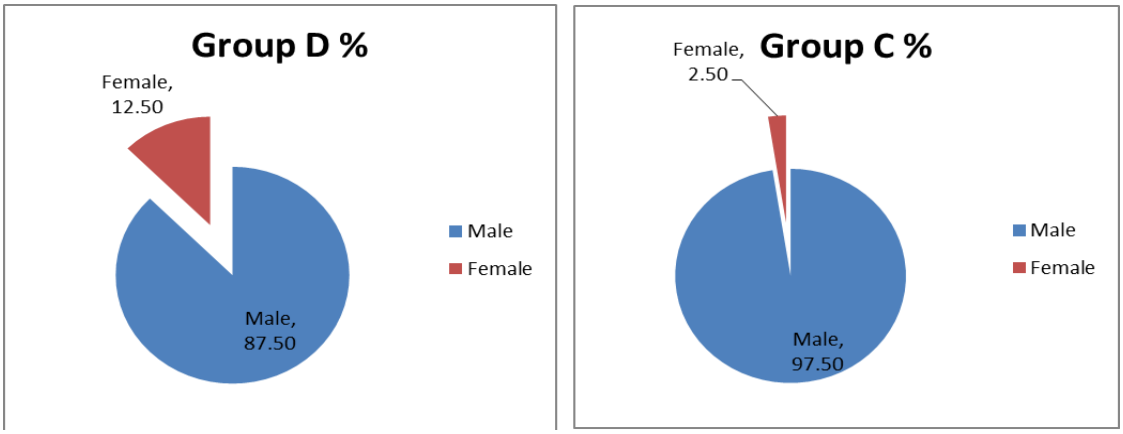

Fig2:Pie diagram showing gender distribution in both the groups

Weight:

The mean weight in the group D was 55.77±5.8kgs as compared to 55.25 ±6.35 kgs in Group C and the difference was statistically not significant (Pvalue-0.8465) .There was no statistically significantdifference in weightdistribution in both groups

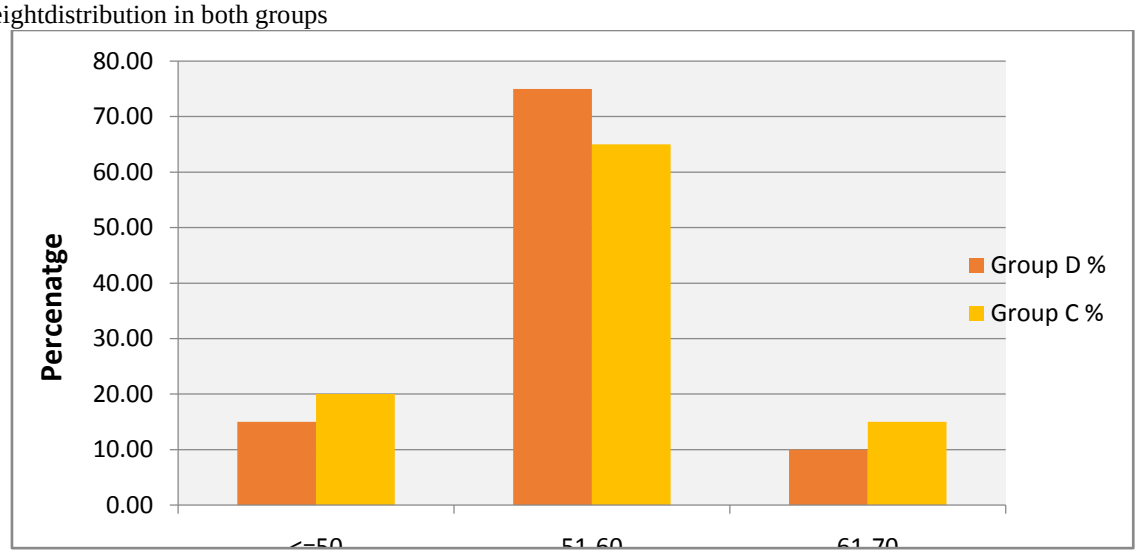

Fig3:Bar diagram showing distribution of weight in both the groups

Height:

The mean height in the group D was 159.0±4.18kgs as compared to 159.1 ±4.25 kgs in Group C and the difference was statistically not significant (Pvalue-0.9159) .There was no statistically significant difference in weight distribution in both groups

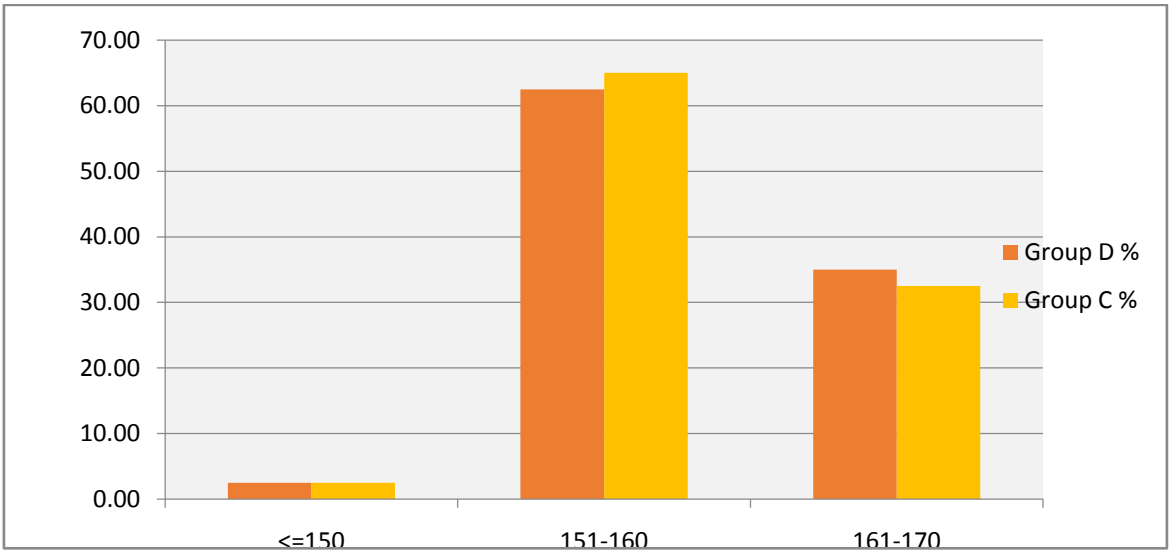

Fig4:Bar diagram showing distribution of height in both the groups

ASA: There was no statistically significant difference between the two groups in ASA grade

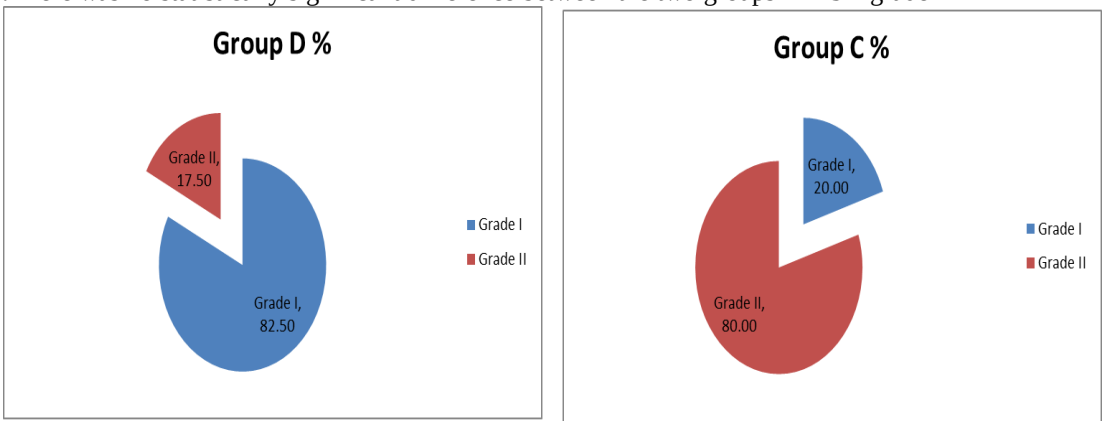

Fig5:Pie diagram showing ASA Grade in both the groups

Duration of surgery:

The mean duration of surgery in the dexmedetomidine group was 76.075±6.28 minutes as compared to 59.15 ± 13.22 minutes in control group and the difference was statistically significant (P value-0.000001). The duration of surgery in both the groups is summarized in Table

Table3: The Duration of Surgery in both groups

Duration in Min	Group D		Group C		P value
	Number	%	Number	%	
<50	0	0.00	14	35.00	
51-60	2	5.00	8	20.00	
61-70	9	22.50	13	32.50	0.000001
71-80	25	62.5	5	12.5	
81-90	4	10	0	0	
Total	40	100	40	100	
Mean+ SD	76.075+6.28		59.15+13.22		

Duration of sensory block in both the groups

The mean duration of sensory block in the group D was 139.475+3.55 minutes as compared to 123.975+4.57 minutes in group C and the difference was statistically significant (P value-0.000001). The duration of sensory block in both the groups is summarized in the table below

Table4: The Duration of Sensory blocks in both groups

Time in Min	Group D		Group C		P Value
	No	%	No	%	
110-120	0	0.00	15	37.50	
121-130	0	0.00	22	55.00	
131-140	24	60.00	3	7.50	0.000001
141-150	16	40.00	0	0.00	
Total	40	100	40	100	
Mean+_SD	139.475+_3.55		123.975+_4.57		

Duration of motor block in both the groups

The mean duration of motor block in the group D was 139.15+3.285 minutes as compared to 118.675+4.54 minutes in group C and the difference was statistically significant (P value-0.000001). The duration of motor block in both the groups is summarized in Table

Table5: The Duration of Motor blocks in both groups

Time in Min	Group D		Group C		P Value
	No	%	No	%	
110-120	0	0.00	29	72.50	
121-130	11	27.50	11	27.50	0.00001
131-140	29	72.50	0	0.00	
Total	40	100	40	100.00	
Mean+_SD	134.15+_3.285		118.675+_4.54		

Duration of two segment regression in both the groups

The mean duration of two segment regression in the group D was 81.25+3.62 minutes as compared to 69.475+3.55 minutes in group C and the difference was statistically significant (P value-0.000001). The duration of two segment regression in both the groups is summarized in Table

Table6: The Duration of two segmental regression blocks in both groups

Time in Min	Group D		Group C		P Value
	No	%	No	%	
60-70	1	2.50	30	75.00	
71-80	20	50.00	10	25.00	0.000001
81-90	19	47.50	0	0.00	
Total	40	100	40	100	
Mean+_SD	81.25+_3.62		69.475+_3.55		

IV.DISCUSSIONS

Different drugs like epinephrine, phenylephrine, adenosine, magnesium sulphate, sodium bicarbonate, neostigmine and alpha2 agonists like clonidine, dexmedetomidine have been used as adjuvants to local anaesthetics to prolong the duration of spinal anaesthesia. Among them clonindine an alpha2 agonist is widely used by oral, intrathecal and intravenous routes as an adjuvant to prolong spinal anaesthesia. Recent studies have shown the efficacy of both intrathecal and intravenous dexmedetomidine in prolonging spinal

anaesthesia. Dexmedetomdine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective alpha 2A receptor agonist activity. Systemic and intrathecal injection of dexmedetomidine produces analgesia by acting at spinal level, laminae VII and VIII of ventral horns. The drug also acts at locus ceruleus and dorsal raphe nucleus to produce sedation and analgesia. This supra spinal action explains the prolongation of spinal anaesthesia after intravenous dexmedetomidine.

Sensory blockade :

In our study mean time for two dermatomal regression of sensory blockade was significantly prolonged in dexmedetomidine group [81.25 ±3.62] compared to control group [69.475±3.55] (P value < 0.001). Significant prolongation in mean time for two dermatomal regression of sensory blockade was also reported by others [Kaya (2010) et al[10] -145 + 26 min vs 97 + 27 mins (P < 0.001), Tekin (2009) et al [6]148.3 mins vs 122.8 mins (P value < 0.001) in dexmedetomidine and control groups respectively]. Similarly Hong (2012) et al[11] reported that the mean time to two-segment regression was prolonged in dexmedetomidne 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 (2010) et al [5).Similarly SSHarsoor[12] (2013) et al reported that the time for two segment regression was prolonged in dexmedetomidine group(the time for two segment regression was 111.52±30.9 min in Group D and 53.6±18.22 min in Group C)

The duration of sensory blockade i.e. time for regression to S1 dermatome was significantly prolonged in dexmedetomidine group [139.475 ±3.55] compared to control group [123.975 ± 4.57] (P value < 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 [3](2009) 261.5 ± 34.8 min vs 165.2 ± 31.5 min (P value < 0.05), Whizar-Lugo et al [4](2007) - 208±43.5 mins vs 137±121.9 mins (P= 0.05) in dexmedetomidine and control groups respectively .

Motor blockade

The regression time to reach the modified Bromage Scale 0 was significantly prolonged in dexmedetomidine group [134.15±3.285 mins] compared to control group [118.675±4.54 min] (P value < 0.00001).Delay in motor block regression to Bromage Scale 0 was also reported in previous studies [Al Mustafa - et al [3](2009) 199 ± 42.8 min in vs138.4 ± 31.3 min (P value < 0.05), Whizar-Lugo et al[4](2007) - 191±49.8 mins vs 172±36.4 (P value- not significant), Tekin et al [6] (2009) 215 mins vs 190.8 mins (P value < 0.001) for dexmedetomidine group and control group respectively]. Elcıcek et al [5](2010) and Hong et al[11] (2012) also found that complete resolution of motor blockade was significantly prolonged in dexmedetomidine group. SS Harsoor[12] (2013) et al complete regression of motor blockade took longer time in Group D (256.44±53.10 min) compared with Group C (231.16±32.2 min), *P*>0.001. But contrary to all the above studies, Kaya et al[10] (2010) reported no significant prolongation in the duration of motor block in dexmedetomidine group compared to control group .

In our study there was no significant difference in intraopeartive and post operative systolic and diastolic blood pressure. This was similar to that reported by Mustafa and Teki in their study.AlMustafaet al [3](2009) and Tekiet al [6] (2009) reported no significant difference in mean arterial pressures in dexmedetomidine and control groups.In the present study, there was no significant difference in the number of patients requiring mephentermine for management of hypotension in both the groups [15% vs 10% in dexmedetomidine and control groups respectively . Similarly, Tekinet al[6) (2009) 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 [3](2009) - 0% vs 20% (P value- 0.15), Whizar-Lugo et al [4](2007) - 8% vs 4% in dexmedetomidine and control groups respectively].

In our study the intraoperative heart rate was lower in dexmedetomidine group than in control group.In previous studies with hyperbaric bupivacaine heart rate was significantly lower in dexmedetomidine group.The incidence of bradycardia was higher in dexmedetomidine group (27.50 %) as compared to control group (15 %) .Higher incidence of bradycardia in dexmedetomidine group [16.66%] compared to control group [8.3%] (P value 0.46) was reported by Al Mustafaet al [3](2009) .Whizar-Lugoet al [4](2007) reported higher incidence of bradycardia in dexmedetomidne group [32%] compared to control group [20%].

Effect of dexmedetomidine respiratory rate:

Despite providing good sedation, dexmedetomidine does not cause significant respiratory depression, providing wide safety margins [13]. In our study, there was no significant differencein the respiratory rates between both the groups during surgery and in the postoperative period.

Ramsay sedation scores:

In our study intraoperative Ramsay sedation scores were significantly higher in dexmedetomidine group [Mean-3.4 +0.496] as compared to control group [Mean- 2] (P value <0.001).However there was no

significant difference in sedation scores between the groups in the postoperative period.Ramsay sedation score was 2 in all patients in control group and ranged from 2-5 in dexmedetomidine group in the study done by Al Mustafa et al [(3)](2009) 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[(11)] (2012) noted that the median sedation scores during surgery were 4 in the dexmedetomidine group and 2 in the control group(Pvalue< 0.001).Tekin et al [(6)] (2009) --- noted that the average sedation score in dexmedetomidine group was significantly higher than in control group (P value < 0.001) during anesthesia.Elcicek et al[(5)](2010),Kaya et al[(10)] (2010) also reported that sedation scores during surgery were significantly higher in dexmedetomidine group than control group.

V.Conclusion

Intravenous Dexmedetomidine significantly prolongs the duration of sensory and motor block of hyperbaric Lidocaine (xylocaine) spinal anaesthesia. The incidence of bradycardia is significantly high when intravenous Dexmedetomidine is used as an adjuvant to Lidocaine (xylocaine) spinal anaesthesia. Dexmedetomidine induced bradycardia is transient and responds to atropine. The changes in blood pressure are without significant clinical impact and hypotension can be easily managed with bolus of IV fluids and mephentermine. Dexmedetomidine provides excellent sedation during surgery and sedation scores reach normal within 15 mins after stopping the drug.

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