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Research Article

Epidural bupivacaine combined with dexmedetomidine or clonidine in infraumbilical surgeries: a comparative evaluation

Shilpi Agarwal[1], Rakesh Bahadur Singh[2]*, Dheer Singh[2], Manoj Kumar[2], Prashant Kumar Mishra[2], Bharat Bhushan Bhardwaj[2]

[1]Department of Anaesthesiology, Bheem Rao Ambedkar Institute Rotatory Cancer Hospital, AIIMS, New Delhi, India
[2]Department of Anaesthesiology, Rural Institute of Medical Sciences and Research, Saifai, Etawah, UP, India

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***Correspondence:**
Dr. Rakesh Bahadur Singh,
E-mail: drrakeshsingh27@gmail.com

ABSTRACT

Background: Alpha-2 agonist are being extensively evaluated as an alternative to neuraxial opoids, as an adjuvants in regional anaesthesia The faster onset of action of local anaesthetics, rapid establishment of both sensory and motor blockade, prolonged duration of analgesia into postoperative period, dose sparing action of local anaesthetics and stable cardiovascular parameters make these agents a very effective adjuvant in regional anaesthesia.
Methods: Our study had 45 patients, all patients belonged to ASA Grade-I or II, between 20 and 55 years of age with an average height of 150 and 170 cm and have ideal body weight requiring neuraxial blockade for lower abdominal surgeries. All the patients were randomly allocated into two groups Group-I: Epidural bupivacaine 0.5% (16 ml) + clonidine 75 µgm (1 ml) Group-II: Epidural bupivacaine 0.5 % (16 ml) + Dexmedetomidine 50 µgm (1 ml) Patients were monitored for sensory and motor blockade, hemodynamic parameters, rescue analgesia, sedation and adverse effects in perioperative period.
Results: The time of onset of sensory block at T_{10} and time to reach maximum sensory block (T6) in group-I was significantly longer as compared to group-II. The complete motor blockade (grade-3) was achieved much later and time taken for recovery to grade-0 was significantly shorter in group-I. The time for rescue analgesia in group-I was significantly shorter as compared to group-II. Hypotension was the most common side effect in both the groups. Dry mouth is a known side effect of alpha-2 agonists. Epidural dexmedetomidine produced profound sedation.
Conclusions: We conclude from this study that dexmedetomidine is a better adjuvant than clonidine for providing early onset of sensory analgesia, superior sedative properties and prolonged post-operative analgesia.

Keywords: Epidural, Bupivacaine, Clonidine, Dexmedetomidine

INTRODUCTION

For thoracic, abdominal and major orthopaedic surgery epidural anaesthesia and analgesia can provide pain relief for a longer duration and the facility of further top-ups and continuous infusion of the analgesic drugs through epidural catheter thus provides an uneventful and smooth recovery. Almost without exception epidural analgesia regardless of analgesic agent, epidural regimen, and type and time of pain assessment, provided superior postoperative analgesia compared to intravenous patient-controlled analgesia.[1]

Bupivacaine is a local anaesthetic drug belonging to the amino amide group and is indicated for nerve block, epidural and intrathecal anaesthesia. It is markedly cardiotoxic but adverse reactions are rare when it is administered correctly.[2] Epidural bupivacaine is equally safe and significantly more potent and more economical than epidural ropivacaine.

In recent years alpha-2 agonist are being extensively evaluated as an alternative[3-5] to neuraxial opioids, as an adjuvants in regional anaesthesia as neuraxial opioids may be associated with quite a few side effects, such as respiratory depression, nausea, urinary retention and pruritis. The pharmacological properties of alpha-2 agonists are being studied and have been employed clinically to achieve the desired effects in regional anaesthesia.[6-10] Epidural administration of these drugs is associated with sedation, analgesia, anxiolysis, hypnosis and sympatholysis.[11,12] The faster onset of action of local anaesthetics, rapid establishment of both sensory and motor blockade, prolonged duration of analgesia into postoperative period, dose sparing action of local anaesthetics and stable cardiovascular parameters make these agents a very effective adjuvant in regional anaesthesia.[13] They can provide pain relief by an opioid independent mechanism as it directly stimulates pre- and postsynaptic alpha-2 adrenoceptors in the dorsal horn grey matter of the spinal cord, thereby inhibiting the release of nociceptive neurotransmitters.[14]

Clonidine is an alpha-2 adrenergic agonist that has a variety of different actions including antihypertensive effects as well as the ability to potentiate the effects of local anaesthetics. It has been used as an adjuvant to epidural local anaesthetics and opioids to improve the quality of analgesia after major abdominal surgeries.[15]

Although clonidine has been used successfully over the last decade for said purpose but the introduction of dexmedetomidine has further widened the scope of alpha-2 agonists in regional anaesthesia. It is a highly selective alpha-2 agonist with an affinity of eight times greater than clonidine. Dexmedetomidine compared to clonidine is a much more selective alpha2-adrenoceptor agonist, which might permit its application in relatively high doses for sedation and analgesia without the unwanted vascular effects from activation of alpha1-receptors.In addition, dexmedetomidine is shorter-acting drug than clonidine and has a reversal drug for its sedative effect, atipamezole. These properties render dexmedetomidine suitable for sedation and analgesia during the whole perioperative period: as premedication, as an anaesthetic adjunct for general and regional anaesthesia, and as postoperative sedative and analgesic.[16]

Keeping all these pharmacological interactions in mind, we planned a double blind prospective randomized study at our institute with an aim to observe the effect of addition of these drugs to bupivacaine and to compare the analgesic and sedative effects of both these adjuvant drugs when used epidurally as an adjuvant to bupivacaine in patients undergoing infraumbilical surgeries.

METHODS

The present study on "Epidural bupivacaine combined with dexmedetomidine or clonidine in infraumbilical surgeries - A comparative evaluation" was conducted after approval by the ethical committee of the institution, a written consent was taken from the patients after explaining to them in detail about the implications of the anaesthetic and the surgical procedure. Our study had 45 patients, all patients belonged to ASA Grade-I or II, between 20 and 55 years of age with an average height of 150 and 170 cm and have ideal body weight requiring neuraxial blockade for lower abdominal surgeries. Patients having morbid obesity, pregnancy, psychiatric disease, history of drug abuse, expected duration of surgery >3 hours and any contraindication to regional anaesthesia were excluded from the study. Routine investigations were done preoperatively in all the patients.

All the patients were randomly allocated into two groups.

Group-I: Epidural bupivacaine 0.5% (16 ml) + Clonidine 75 µgm (1 ml)

Group-II: Epidural bupivacaine 0.5% (16 ml) + Dexmedetomidine 50 µgm (1 ml)

All the patients were administered premedication a night before and on the morning of the surgery which comprised tablet ranitidine 150 mg and tablet alprazolam 0.25 mg. The patients were explained about the sequence of anesthetic procedure in the pre-op room and a good IV access was secured. All the patients were preloaded with 500 ml of ringer lactate before administration of block. Thereafter, the patients were shifted to the operation theater and all monitoring devices were attached which included devices measuring Heart Rate (HR), Non-Invasive Blood Pressure (NIBP), ECG, SpO_2, and respiratory rate. Baseline hemodynamic parameters, respiratory rate, ECG and SpO_2 were recorded.

Patients were administered epidural block in a sitting position/left lateral position. The skin was infiltrated with 2% lignocaine. A 18-gauge Touhy needle was introduced by the midline approach into epidural space at L3-4 interspace, and epidural space was localized and confirmed by loss of resistance to saline technique. Epidural catheter was secured 3-5 cm into the epidural space and confirmation for correct placement was done by injecting 3 ml of 2% lignocaine HCl solution containing adrenaline 1:200000. After 4-6 min of test dose, patients in Group-I administered 0.5% bupivacaine (16 ml) + clonidine 75 µgm (1 ml). In Group-II, patients received 0.5 % bupivacaine (16 ml) + Dexmedetomidine 50 µgm (1 ml).

The bilateral pin prick method was used to evaluate and check the sensory level by 25G hypodermic needle. The onset time of block at the level of T10 was recorded and highest dermatomal level of sensory analgesia and time to achieve it was recorded. Similarly regression of sensory level block at the end of procedure was done. Time taken by highest level of block to recede to T_{10} level was recorded in all the three groups. Surgical procedures were

initiated only after the establishment of adequate surgical anesthetic effect with minimum level up to T6-7 dermatome.

Motor block and recovery was assessed by using modified Bromage scale:

Grade-0 = No block

Grade-1 = Inability to raise extended leg

Grade-2 = Inability to flex knee

Grade-3 = Inability to flex ankle and foot

And this was used to measure the motor blockade effect at 5, 10, 15, 20, 25, and 30 minute intervals after the epidural administration of the drugs.

Grading of sedation was done using five point scale:

Grade-1 = Alert and wide awake

Grade-2 = Arousable to verbal command.

Grade-3 = Arousable with gentle tactile stimulation.

Grade-4 = Arousable with vigorous shaking

Grade-5 = Unarousable

Sedation scores were recorded just before the initiation of surgery and thereafter during the surgical procedure. Hemodynamic parameters, which included HR, ECG, mean arterial pressure (MAP), SpO_2 and respiratory rate, were monitored continuously. Recordings were made every 5minutes until 30 minutes and thereafter at 10-minute intervals up to 60 minutes then at 15-minute intervals for the next hour and finally at 30 minutes in the third hour.

Hypotension (defined as systolic arterial pressure falling more than 20% mmHg) was treated with inj. mephenteramine 3-6 mg in bolus doses and bradycardia (HR<55 beats/min) was treated with 0.3 mg of inj. atropine. Intravenous fluids were given as per the body weight and operative loss requirement, with no patient requiring blood transfusion. The patients were given supplementary O_2 with the help of venturi mask.

During the surgical procedure, any adverse events like anxiety, nausea, vomiting, headache, shivering, dry mouth, respiratory depression, bradycardia, or hypotension was recorded and treated accordingly. Nausea and vomiting were treated with 4-6 mg of i.v. ondansetron.

All the patients were kept for 8 hours in recovery room. Vital parameter like pulse rate, blood pressure, respiratory rate and oxygen saturation were recorded in recovery room. All the patients were monitored for degree and duration of pain relief by pain scoring system.

Assessment of analgesia was done by using following criteria:

Analgesic score-

0 = No pain at rest or with movement

1 = No pain at rest, but pain during voluntary body movement

2 = Pain at rest but tolerable

3 = Intolerable pain

Patients in both the groups were repeatedly assessed every 30 minutes for pain and rescue analgesic in the form of 8 ml of 0.25% of bupivacaine was given when they complained intolerable pain (pain intensity score-3) which is comparable to visual analogue scale (VAS-10) or worst pain imaginable.

Comparability of the groups was analysed by Student's unpaired "t" test and chi square test. For all statistical analysis, the value of $p<0.05$ was considered significant and value of $p<0.001$ was considered as highly significant.

RESULTS

With careful appraisal of the present study, following results were found:

Table 1 shows distribution of patients according to their demographic data in group I and group II; Table 2 shows comparison of duration of surgeries in both the groups (Group I and II).

Table 1: Distribution of patients according to their demographic data.

	Group-I (Mean ± SD)	Group-II (Mean ± SD)
Age (years)	39.26 ± 12.21	39.80 ± 12.13
Height (cm)	161.72 ± 16.25	160.44 ± 5.56
Weight (kg)	56.04 ± 8.41	57.35 ± 5.95
Sex (M:F)	9:6	10:5

Table 2: Comparison of duration of surgeries in both the groups.

Groups	Total No. of patients	Mean duration	SD	't' value	'p' value
Group-I	15	108.6	35.47	0.327	>0.05
Group-II	15	104.66	30.26		

1) The time of onset of sensory block at T_{10} in group-I was significantly longer (10.53 ± 2.38 min) as compared to group-II (8.4 ± 2.92 min) (Table 3).

Table 3: Time of onset of sensory block at T_{10} level in both groups.

	Mean ± SD	't' value	'p' value
Group I	10.53 ± 2.38	2.19	<0.05
Group II	8.4 ± 2.92		

2) The time to reach maximum sensory block (T_6) in group-I was significantly longer (15.66 ± 2.38 min) as compared to group-II (13.4 ± 3.01 min) (Table 4).

Table 4: Time to reach maximum sensory block in both groups.

	Mean ± SD	't' value	'p' value
Group I	15.66 ± 2.38	2.28	<0.05
Group II	13.4 ± 3.01		

3) The complete motor blockade (grade-3) was achieved much later in group-I (21.53 ± 3.46 min) as compared to group-II (18.8 ± 3.37 min) (Table 5).

Table 5: Time taken for complete block (Grade-3) in both groups.

	Mean ± SD	't' value	'p' value
Group I	21.53 ± 3.46	2.18	<0.05
Group II	18.8 ± 3.37		

4) The time taken for recovery of motor block to grade-0 was significantly shorter in group-I (282.6 ± 37.5 min) as compared to group-II (314 ± 38.78 min) (Table 6).

Table 6: Time taken for recovery of motor block to grade-0 in both groups.

	Mean ± SD	't' value	'p' value
Group I	282.6 ± 37.5	2.25	<0.05
Group II	314 ± 38.78		

5) The time for regression of the sensory block to T_{10} was significantly shorter in group-I (360 ± 29.27 min) as compared to group-II (390.33 ± 41.07 min) (Table 7).

6) The time for rescue analgesia in the group-I was significantly shorter (399.33 ± 32.8 min) as compared to the group-II (434.33 ± 50.83 min) (Table 8).

7) Epidural dexmedetomidine produced profound sedation in 46.6% patients who were arousable by gentle tactile stimulation (grade-3) compared to achievement of similar sedation level in 13.3% in clonidine group, and statistically this difference was found to be significant ($p<0.05$) (Table 9).

Table 7: Time taken for regression of sensory block to T10 in both groups.

	Mean ± SD	't' value	'p' value
Group I	360 ± 29.27	2.30	<0.05
Group II	390.33 ± 41.07		

Table 8: Time taken for first rescue top up in both groups.

	Mean ± SD	't' value	'p' value
Group I	399.33 ± 32.8	2.24	<0.05
Group II	434.33 ± 50.83		

Table 9: Comparison of intraoperative sedation scores in both groups.

Grade	Group-I (%)	Group-II (%)	'p' value
1	05 (33.3)	02 (13.3)	<0.05
2	08 (53.3)	05 (33.3)	<0.05
3	02 (13.3)	07 (46.6)	<0.05
4	00 (00.00)	01 (06.00)	-
5	00 (00.00)	00 (00.00)	-

8) Haemodynamically, group-II showed a highly significant fall in pulse rate and blood pressure as compared to group-I which showed a significant fall following epidural anaesthesia (Table 10).

9) All patients in group-I had no significant change in their oxygen saturation and respiratory rate while in group-II there was no change in respiratory rate but there was significant change in oxygen saturation as compared to pre- epidural value (Table 11 & 12).

10) Hypotension was the most common side effect in both the groups. Dry mouth is a known side effect of alpha-2 agonists. It was observed in our study with 26.6% patients reporting with dry mouth in group-I and 20 % in group-II (Table 13).

Table 10: Comparison of hemodynamic parameters in both groups at different time interval.

Time interval	Pulse rate						Mean arterial blood pressure					
	Group I			Group II			Group I			Group II		
	Mean ± SD	't' value	'p' value	Mean ± SD	't' value	'p' value	Mean ± SD	't' value	'p' value	Mean ± SD	't' value	'p' value
15 minutes	94.46 ±18.47	1.46	>0.05!	81.13 ± 12.36	0.81	>0.05!	90.04 ± 10.50	2.24	<0.05*	90.40 ± 8.1	3.4	<0.05*
30 minutes	80.40 ±12.01	1.51	>0.05!	71.73 ± 10.93	3.55	<0.05*	87.00 ± 10.10	3.02	<0.05*	86.00 ± 7.8	5.02	<0.001**
60 minutes	82.53 ±7.69	1.22	>0.05!	76.00 ± 10.84	2.35	<0.05*	90.30 ± 09.10	2.3	<0.05*	89.80 ± 5.7	4.2	<0.001**
120 minutes	80.00± 06.90	2.08	>0.05!	75.00 ± 11.10	2.6	<0.05*	92.40 ± 08.03	1.8	<0.05*	90.62 ± 5.4	3.9	<0.05*

!- Insignificant, *****- Significant, **- Highly significant

Table 11: Comparison of mean SPO_2 in both groups at different time interval.

Time interval	SPO_2					
	Group I			Group II		
	Mean ± SD	't' value	'p' value	Mean ± SD	't' value	'p' value
15 minutes	99.60 ± 0.63	0.65	>0.05!	98.93 ± 0.96	3.78	<0.05*
30 minutes	99.40 ± 0.63	1.65	>0.05!	98.40 ± 1.50	3.8	<0.05*
60 minutes	99.80 ± 0.41	0.44	>0.05!	98.40 ± 1.40	4.08	<0.05*
120 minutes	99.73 ± 0.45	0.00	>0.05!	98.60 ± 1.20	4.1	<0.05*

!-Insignificant, *- Significant

Table 12: Comparison of mean respiratory rates in both groups at different time interval.

Time interval	SPO_2					
	Group I			Group II		
	Mean ± SD	't' value	'p' value	Mean ± SD	't' value	'p' value
15 minutes	14.40 ±1.35	0.12	>0.05!	15.20 ± 1.47	0.24	>0.05!
30 minutes	14.60 ±1.35	0.27	>0.05!	15.20 ± 1.47	0.24	>0.05!
60 minutes	14.30 ±1.29	0.33	>0.05!	15.20 ± 1.26	0.26	>0.05!
120 minutes	14.06±1.33	0.81	>0.05!	15.20 ± 1.26	0.26	>0.05!

!-Insignificant

Table 13: Comparison of side effects in both groups.

Side effects	Group-I	Group-II
Nausea/vomiting	2 (13.3)	3 (20.0)
Hypotension	4 (26.6)	6(40.0)
Bradycardia	1 (06,6)	2 (13.3)
Shivering	2 (13.3)	1 (06.6)
Dry mouth	4 (26.6)	3 (20.0)
Pruritis	0 (00.0)	0 (00.0)
Headache	1 (06.6)	0 (00.0)

DISCUSSION

Sukhminder Jit Singh Bajwa et al. (2010) did a study to find a better adjuvant in regional anaesthesia. Aims and objectives were to compare the efficacy and clinical profile of two alpha-2 agonists, dexmedetomidine and clonidine. Group RD was administered 17 ml of 0.75% epidural ropivacaine and 1.5 µg/kg of dexmedetomidine, while group RC received admixture of 17 ml of 0.75% ropivacaine and 2 µg/kg of clonidine. Addition of dexmedetomidine to ropivacaine as an adjuvant resulted in an earlier onset (8.52 ± 2.36 min) of sensory analgesia at T_{10} as compared to addition of clonidine (9.72 ± 3.44 min). Dexmedetomidine not only provided a higher dermatomal spread but also helped in achieving the maximum sensory anaesthetic level in a shorter period (13.14 ± 3.96 min) compared to clonidine (15.80 ± 4.86 min). All these initial block characteristics turned out to be statistically significant ($p<0.05$) on comparison.[17]

Thus the results of our study were comparable to the above study (Table 3 & Table 4).

They also found that modified Bromage Grade-3 was achieved earlier (17.24 ± 5.16 min) in patients who were administered dexmedetomidine as adjuvant as compared to clonidine (19.52 ± 4.06 min). The initial block characteristics were found to be statistically significant ($p<0.05$) on comparison.

In our study also we found a statistically significant ($p<0.05$) difference in time for complete motor block in Group-I (21.53 ± 3.46) and Group-II (18.8 ± 3.37). Thus the results were comparable to the above study (Table 5).

They also clearly indicated in their study that dexmedetomidine produced profound sedation in 36% of the patients who were arousable by gentle tactile stimulation compared to similar sedation levels in just 16% of patients.

Thus the results of our study were comparable to the above study (Table 9).

Bajwa et al. (2011) in another study compared the hemodynamic, sedative, and analgesia potentiating effects of epidurally administered fentanyl and dexmedetomidine when combined with ropivacaine. Motor blockade was assessed using modified Bromage scale and complete motor blockade was achieved significantly earlier (18.16 ± 4.52) in the patients who were administered Dexmedetomidine as compared to fentanyl group (22.98 ± 4.78).[18]

In our study we also found that Group-3 achieved motor blockade quiet early (18.8 ± 3.37). Thus the results were comparable to above study (Table 5).

Swami SS et al. (2012) compared clonidine and dexmedetomidine as an adjuvant to local anaesthetic agent in supraclavicular brachial plexus block with respect to onset and duration of sensory and motor block and duration of analgesia. Duration of sensory block and motor block was 227.00 ± 48.36 and 292.67 ± 59.13 min respectively, in Group-C, while it was 413.97 ± 87.13 and 472.24 ± 90.06 min respectively, in Group-D. This difference was statistically significant ($p=0.001$).[19]

The results of our study were comparable to above study because we also found significant prolongation of sensory and motor blockade in Group-II as compared to Group-I (Table 6, Table 7 & Table 8).

F Salgado et al. (2008) observed the clinical effects and the potential synergism between dexmedetomidine, a highly selective α_2-adrenergic agonist, and ropivacaine in patients undergoing epidural anaesthesia. Epidural dexmedetomidine (1 µg.kg-1) did not affect onset time nor upper level of anaesthesia ($p>0.05$) but prolonged sensory and motor block duration ($p<0.05$) and postoperative analgesia ($p<0.05$).[20]

The results of our study could be compared to above study as Group-II in our study had prolonged sensory and motor block duration as compared to group-I (Table 6, Table 7 & Table 8).

They also found that heart rate, systolic and diastolic arterial pressure were lower for DEX ($p<0.05$) but hypotension (30% reduction of initial systolic arterial pressure) and bradycardia (heart rate ≤45 bpm) were not significant ($p>0.05$).

The results of our study were comparable to above study as in our study also Group-II showed lower pulse rate and blood pressure as compared to Group-I (Table 10).

They also concluded that sedation and no respiratory depression is an advantage of association between ropivacaine and dexmedotomidine.

Thus the results of our study were comparable to the above study as there was no significant (>0.05) respiratory depression in Group-I & Group-II (Table 12).

AM El Hennaway (2009) compared the analgesic effects and side-effects of dexmedetomidine and clonidine added to bupivacaine in paediatric patients undergoing lower

abdominal surgeries. Addition of dexmedetomidine or clonidine to caudal bupivacaine significantly prolonged analgesia time [median (95% confidence interval, CI): 16 (14-18) and 12 (3-21) h, respectively] than the use of bupivacaine alone [median (95% CI): 5 (4-6) h] with p<0.001.[21]

Thus the results of our study were comparable as Group-I had significantly shorter analgesia time (p<0.05) as compared to Group-II (Table 8).

DM Elhakim et al. (2010) compared the effects of a dexmedetomidine-bupivacaine mixture with plain bupivacaine for thoracic epidural anaesthesia on intraoperative awareness and analgesic benefits, when combined with light isoflurane anaesthesia (<0.05 maximum alveolar concentration) in patients undergoing thoracic surgery with One-Lung Ventilation (OLV). They concluded that in thoracic surgery with OLV, the use of epidural dexmedetomidine decreases the anaesthetic requirements significantly, prevents awareness during anaesthesia and improves intraoperative oxygenation and post-operative analgesia.[22]

Jain et al. (2012) designed a study to evaluate the perioperative effect of epidural dexmedetomidine, in conjunction with intrathecal bupivacaine. In this trial, 60 male patients of American Society of Anesthesiologists grades I and II, between 20-50 years of age, and posted for elective lower limb orthopaedic surgery, were selected. There was a highly significant fall in the pulse rate and mean arterial pressure (p<0.001) five minutes following epidural dexmedetomidine in Group II patients, which lasted throughout the study period. The conclusion was that the addition of 2 µg/kg dexmedetomidine epidurally to 2.5 ml of intrathecal bupivacaine prolongs the duration of analgesia with a significant fall in pulse rate and mean arterial pressure.[23]

Thus the results of our study which showed a lower mean pulse rate and lower mean blood pressure for Group-II as compared to Group-I were comparable to above study (Table 10).

Shobna Gupta et al. (2010) compared epidural bupivacaine and bupivacaine and clonidine in knee replacement surgeries and found that there was no significant difference in oxygen saturation and respiratory rate in any of the groups.[24]

Thus the results of our study were comparable to the above study as in Group-1 and in Group-2 we also found no significant difference (p>0.05) in oxygen saturation and respiratory rate compared to their pre-epidural values (Table-12).

Maarouf explored the effect of epidural dexmedetomidine on the incidence of postoperative shivering. He found that patients who received dexmedetomidine at a dose of 100 microgram added to 0.5% bupivacaine (20 ml) had a lower incidence of postoperative shivering when compared to patients who received bupivacaine alone.[25]

Thus the results of our study were comparable to above study as in Group-II, the incidence of shivering was less than in Group-I (Table 13).

CONCLUSION

So this study re-established the fact, that alpha-2 agonists when added as adjuvants to epidural bupivacaine provide a faster onset of action with rapid establishment of sensory and motor block, sedation, and prolonged duration of analgesia in the postoperative period.

We conclude from this study that dexmedetomidine is a better adjuvant than clonidine for providing early onset of sensory analgesia, superior sedative properties and prolonged post-operative analgesia.

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