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COMPARATIVE STUDY OF 0.5% BUPIVACAINE AND 0.5% BUPIVACAINE WITH CLONIDINE (30μg) FOR SPINAL ANAESTHESIA

Shilpashri A. M[1], Priodarshi Roychoudhury[2], Girish K. M[3]

ABSTRACT: In the present day practice of Anesthesiology, bupivacaine is the most commonly used drug for spinal anesthesia. To improve upon the quality of analgesia and prolong the duration of its action, many adjuvants have been tried. Intrathecal clonidine an α2 adrenceptor agonist has potent central antinoceptive properties with analgesic effect at spinal level mediated by postsynaptically situated adrenoreceptor in dorsal horn of spinal cord. Low doses of clonidine have shown effectiveness in intensifying spinal anesthesia. Hence this study was done in our institute to evaluate the efficacy of spinal anesthesia with clonidine added to hyperbaric bupivacaine in elective lower limb, lower abdominal, gynaecological and urological surgeries under spinal anaesthesia. This prospective, single center parallel group, double blind study conducted over a span of 1 year with 100 patients, was effective in proving that Clonidine potentiates bupivacaine spinal anesthesia by increasing the duration and improving the quality of analgesia without producing significant hemodynamic side effects and with mild sedation.
KEYWORDS: Alpha 2 agonist, 0.5% Bupivacaine, Clonidine as adjuvant, Effective analgesia.

INTRODUCTION: Spinal anesthesia, defined as the regional anesthesia obtained by blocking nerves in the subarachnoid space is a popular and common technique used worldwide. The advantages of an awake patient, simple to perform, offers rapid onset of action, minimal drug cost, relatively less side effects and rapid patient turnover has made this the choice of many a surgical procedure.[1]

These advantages are sometimes offset by relatively short duration of action and uncomfortable postoperative period when its action wears off. In order to extend intraoperative analgesia into postoperative period a number of spinal adjuvants like opioids, clonidine, ketamine, morphine and buprenorphine and so on have been added to prolong intrathecal bupivacaine action.

Recently clonidine which is an α2 adrenergic agonist has been tried as an adjuvant to prolong the action of local anesthetics. Intrathecal clonidine produces dose dependent analgesia and has been successfully used as a sole analgesic via the intrathecalroute.[2] hence, this study was designed to evaluate the effectiveness of adding30μg clonidine to bupivacaine for spinal anesthesia and to compare its use with that of bupivacaine.

MATERIALS AND METHODS: After institutional committee approval and written informed consent, patients with ASA grade I or II, aged 18-60 years scheduled to undergo elective lower limb, lower abdominal, gynaecological and urological surgeries under spinal anaesthesia were enrolled in this prospective, single center parallel group, double blind study. This study was conducted in JJMMC DAVANAGERE over a span of 1 yr. involving 100 patients.

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Inclusion Criteria:
1. ASA grade 1 and 2 patients.
2. Age group of 18-60 yrs.
3. Patients giving valid informed consent.
4. Those patients scheduled to undergo elective Lower abdominal, lower extremity, gynaecological or urological surgeries under subarachnoid block.

Exclusion Criteria:
1. Patient refusal.
2. Patients belonging to ASA grade 3 and grade 4.
3. Patients physically dependant on narcotics.
4. Patients with history of drug allergy.
5. Patients with gross spinal abnormality, localized skin sepsis, hemorrhagic diathesis or neurological involvement /Diseases.
6. Head injury cases.
7. Patients with cardiac, pulmonary, hepatic or renal disorders.
8. Patients with peripheral neuropathy.
9. Extremes of age.
10. Patients having inadequate subarachnoid blockade and who are later supplemented by general anaesthesia.
11. Obstetric cases for lower segment caesarean section because of drug dosage discrepancy.

The Study Population was randomly divided into two groups with 50 Patients in each group:
Group "B" Bupivacaine Group: Receiving Intrathecal Bupivacaine 12.5mg (2.5mL) +0.5mL normal saline. (Total 3mL).

Group "BC" Clonidine Group: Receiving Intrathecal Bupivacaine12.5mg (2.5mL) +30µg clonidine.
All patients were premedicated with tab diazepam 10mg and tab ranitidine 150mg orally 10:00 pm at night before surgery and at 7:00am on the morning of surgery.

PROCEDURE: Patient was shifted to the OT table; IV access was obtained on the forearm with 18 Gauge IV cannula and Lactated Ringer's solution 500mL was infused intravenously before the block. The monitors connected to the patient included noninvasive B.P, oxygen saturation using pulse oximeter. Baseline PR, BP and RR, SpO2 was recorded. Under strict aseptic precautions, lumbar puncture was performed in left lateral position or sitting position by midline approach by using disposable Quincke spinal needle (23 G) at L3-L4 intervertebral space. Patients were monitored continuously using noninvasive blood pressure, pulse oximeter and electrocardiogram. After spinal anaesthesia, Oxygen (4L/min) by facemask was given. Fluid therapy was maintained with lactated Ringer's solution (10mL/kg/hr.)

The Following Parameters were Observed and Recorded:
Vital Parameters: HR, B.P and RR, SpO2 monitored at 1,3,5,10,15,20,25,30,45,60,120,180 minutes.

Assessment of Sensory Blockade: The onset of sensory block was tested by pin-prick method using a hypodermic needle. The time of onset was taken from the time of injection of drug into subarachnoid space to loss of pin prick sensation.

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The highest level of sensory block and time required to achieve it was noted. The time for two dermatomal segments regression of sensory level was noted. The duration of sensory blockade was taken as time from onset to time of return of pinprick sensation to S1 (heel) dermatomal area.

Assessment of Motor Blockade: This was assessed by Bromage scale*. The time interval between injections of drug into subarachnoid space, to the patient's inability to lift the straight extended leg was taken as onset time (Br. 3). The duration of motor block was taken from time of injection to complete regression of motor block. (Ability to lift the extended leg) (Br 0).

Modified Bromage Scale[3,4]:
- Grade 0 - Full flexion of knees and feet.
- Grade 1 - Just able to flex knees, full flexion of feet.
- Grade 2 - Unable to flex knees, but some flexion of feet possible.
- Grade 3 - Unable to move legs or feet.

Assessment of Analgesia: Pain was assessed by visual analogue score (VAS).[5] Duration of complete analgesia was defined as the time from the intrathecal injection to VAS>0-<4 and duration of effective analgesia as the time to VAS >4. Analgesics were avoided until demanded by the patient and the time taken for the first pain medication was also noted (i.e., when VAS >6) VAS was also recorded 3, 6, 12 hours postoperatively.

Quality of intraoperative Analgesia: Was assessed on a four point modified Belzarena scale[3]:
1. Unable to tolerate pain.
2. Able to tolerate discomfort with additional analgesia.
3. Some discomfort but no additional analgesics required.
4. Completely satisfied.

Sedation scores were assessed every 15 minutes both intra and post operatively using a four point score described by B.S. Sethi.[3]
- Grade 0 – Patient wide awake.
- Grade 1 – Patient is sleeping comfortably, but responding to verbal Commands.
- Grades 2 – Deep sleep but arousable
- Grade 3 – Deep sleep, unarousable.

Post operatively, monitoring of vital signs, VAS scores and sedation scores was continued every 30 minutes until the time of regression of sensory block to L1 dermatome. The incidence of hypotension (Arterial blood pressure <20% of baseline), and was treated with Inj. Mephentermine 6mg intra-venous increments and bradycardia as pulse rate <60/min was treated by atropine 0.6mg intravenous stat. Side effects like sedation , nausea, vomiting urinary retention were monitored in the recovery room and then shifted to the ward. Neurological examination was done to rule out any neurological deficits at discharge.

Statistical Analysis: Quantitative data was analyzed by student's t test and qualitative data was analyzed by Chi-square test. All values were expressed as mean±standard deviation. P<0.05 was considered statistically significant.

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RESULTS:

ONSET OF SENSORY AND MOTOR BLOCKADE:

In our study, the mean time for onset of sensory block in group BC was 112.22 seconds (1.87min) and 137.60 seconds (2.29min) in group B. The mean time for onset of motor block in group BC was 165.1 seconds (2.75min) and in group B was 231.80 seconds (3.86min). There was statistically significant difference with regard to onset of sensory and motor block between the groups with faster onset in group BC compared to group.

	Group B	Group BC	P Value
Sensory block (sec)	137.60	112.22	<0.001
Motor block (sec)	231.80	165.1	<0.001
Table 1: Onset of Sensory and Motor Blockade			

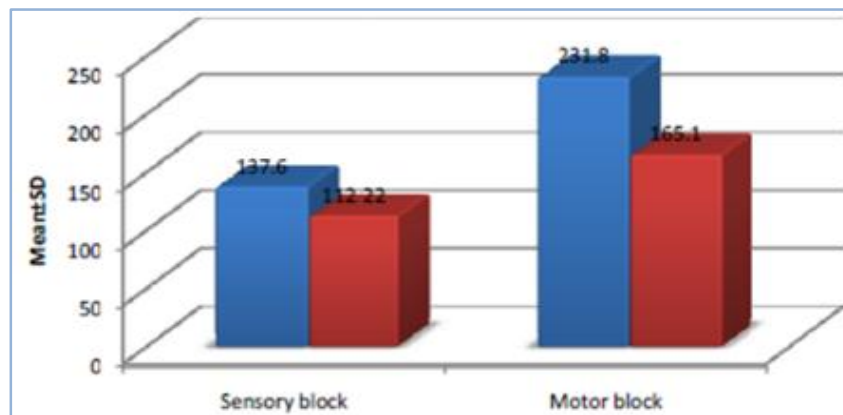Values are expressed as Mean +SD.
Students unpaired 't' test.
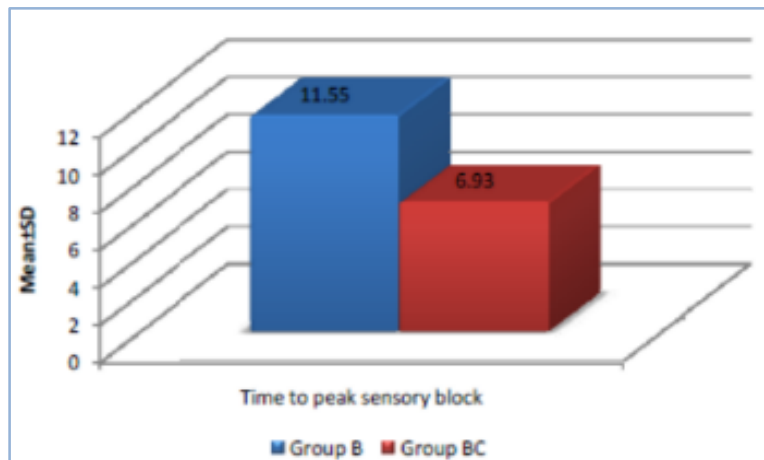

Fig. 1: Onset of Sensory and Motor Blockade

Fig. 2: Time to Peak Sensory Block

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	Group B	Group BC	P Value
Time to Peak Sensory block (in min)	11.55	6.93	<0.0001
Table 2: Time to Peak Sensory Block			

Values are expressed as Mean +SD.

	Group B (%)	Group BC (%)
T4	1(2)	7(14)
T6	10 (20)	28(56)
T8	23 (46)	13(26)
T10	16 (32)	2(4)
Table 3: Highest Level of Sensory Block		

Values are expressed as n (%).

Time for two Segment Regression:

Recovery Parameters: The time for two segment regression was considerably prolonged in group BC with 139.2 minutes and in group B it was 83.3 minutes.

Time for Complete Sensory and Motor Recovery: In our study, the time for complete sensory recovery in group BC was prolonged by about 25-30 minutes (Group B-246.8 minutes, group BC-212.1 minutes). The duration of motor block in group B was prolonged by about 10 minutes (group BC-203.5 minutes, group B-193.8 minutes). The difference was statistically significant [(p< 0.001). (Students unpaired 't' test.] Hence the duration of both sensory and motor blockade was significantly prolonged.

Duration of Analgesia: We found that the duration of complete analgesia in group BC was 240.8 min and in 165.1 min in group B. Effective analgesia was 332.64 minutes in group BC and 212.6 minutes in group B. The time for first request of rescue analgesic post-operatively was considerably delayed in group BC by 140-150 minutes compared to group B (362.84 vs. 221.4 minutes), thereby reducing the requirement of analgesics in the early postoperative period. The quality of analgesia was better as the VAS was lower in group BC than in group B.

Postoperative Analgesia: In our study also there was significant reduction in the VAS scores of the patients receiving clonidine in comparison with higher VAS scores in patients receiving bupivacaine in the first twelve hours post operatively. This implies better quality of analgesia postoperatively, and reduced need of analgesics with the use of intrathecal clonidine

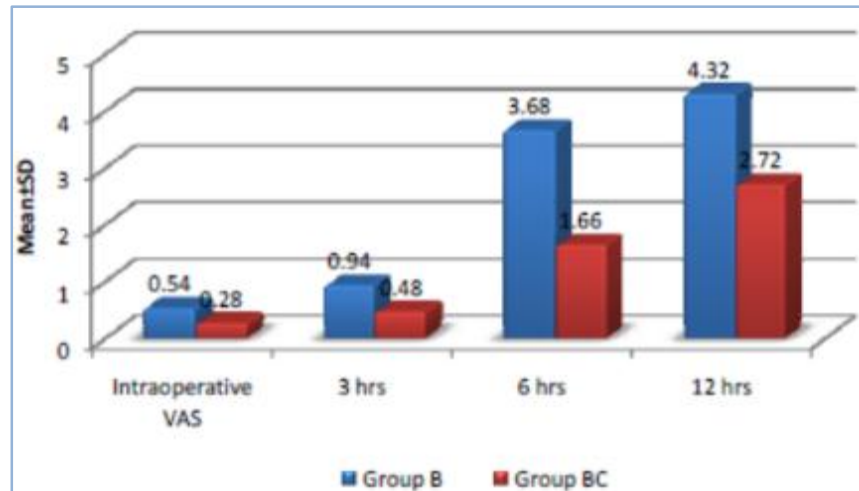| TIME	Group- B	Group - BC	P Value
Intraoperative VAS	0.54±0.05	0.28±0.05	0.008
3 hrs.	0.94	0.48	0.003
6hrs.	3.68	1.66	<0.001
12 hrs.	4.32	2.72	<0.001
Table 4: Visual Analogue Scale (VAS Scores)			

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Fig. 3: Vas Scores

VITAL PARAMETERS:

HAEMODYNAMICS: HEART RATE & BLOOD PRESSURE: In our study, the two groups did not differ significantly with respect to heart rate at any interval. There were no episodes of bradycardia in either group. The changes in mean systolic blood pressure at any time interval was statistically and clinically insignificant.

Whereas changes in mean diastolic blood pressure was statistically significant at 20 minutes, 30 minutes and one hour, but clinically insignificant. Patients in group B experienced hypotension, whereas hypotension in Clonidine group was insignificant. Hypotension was easily corrected with small doses of Inj. Mephentermine/Ephedrine.

Time Interval in (mins)	Group B	Group BC	P Value
0	79.70	79.14	0.813
5	80.14	79.46	0.983
10	80.74	78.10	0.648
20	81.22	78.32	0.390
30	81.84	77.90	0.084
60	87.94	79.04	0.101
120	81.54	78.5	0.105
150	80.82	78.8	0.258

Table 5: Heart Rate

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SBP (mins)	Group B	Group BC	P Value
Base line (0)	123	127	0.1
5	121	127	0.1
10	118	121	0.5
20	115	116	0.4
30	115	116	0.5
60	112	115	0.4
120	118	115	0.4
150	119	118	0.4

Table 6: Systolic B.P

SIDE EFFECTS: In our study, 16% of patients in BC group had grade 2 (Awake but drowsy) sedation compared to 0% of the patients in B group, Hypotension was 12%, Nausea and vomiting was 10%, bradycardia 8%, and urinary retention was 4% in group B compared to clonidine group which was 0%. Since there was mild sedation during perioperartive period, Respiratory rate was monitored to Detect respiratory depression and there was no evidence of respiratory depression in either group.

Adverse effects	Group B	Group B C
Nausea /vomiting	5 (10%)	0 (0)%
Sedation	0 (0 %)	8(16%)
Mouth Dryness	0 (0%)	0 (0%)
Bradycardia	4 (8%)	0 (0%)
Hypotension	6 (12%)	0 (0%)
Urinary Retention	2 (4%)	0 (0%)
Respiratory depression	0 (0%)	0 (0%)

Table 7: Perioperative Complications
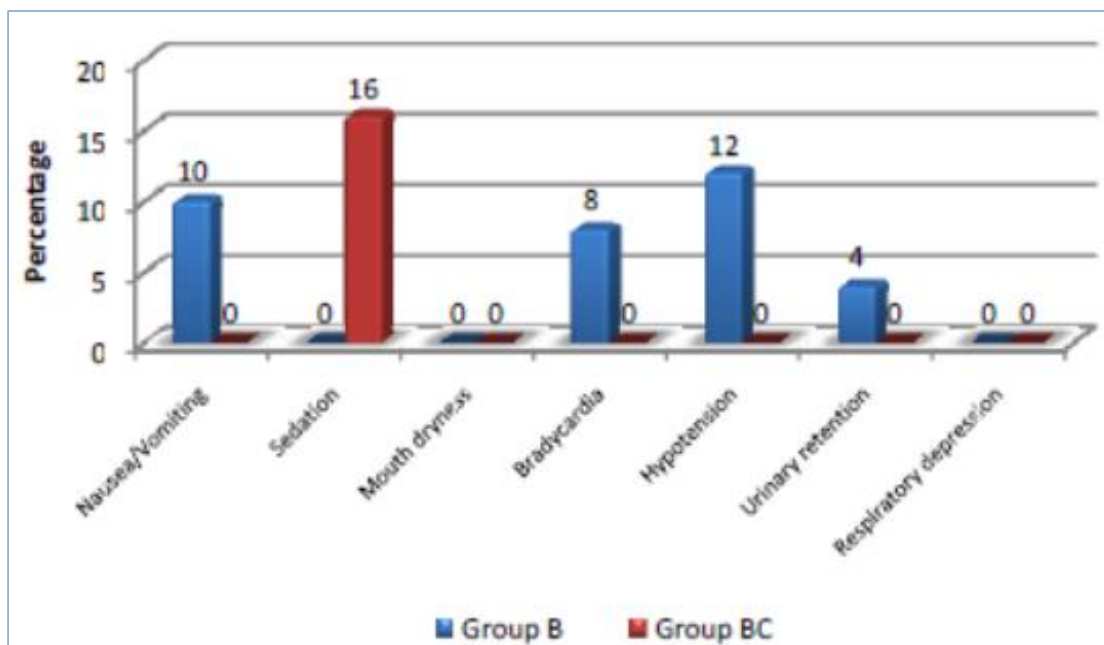
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Fig. 4: Perioperative Complications

DISCUSSION: Spinal anesthesia with hyperbaric bupivacaine 0.5% is a popular method. The duration of spinal analgesia can be prolonged by the adjuvants like vasoconstrictors, opioids, neostigmine, ketamine, midazolam, etc. Vasoconstrictors (epinephrine, ephedrine and phenylephrine) prolong the duration of action of the local anesthetic by decreasing systemic absorption but have been found to induce neurological signs and symptoms due to reduced blood supply to the spinal cord.[6] Intrathecal midazolam produces sedation, ketamine results in psychomotor symptoms and neostigmine causes excessive nausea and vomiting.

Clonidine is a selective partial agonist for α2-adrenoreceptors. It is known to increase both sensory and motor block of local anesthetics.[7] The analgesic effect following its intrathecal administration is mediated spinally through activation of postsynaptic α2 receptors in substantiagelatinosa of the spinal cord and it works by blocking the conduction of C and A delta fibers, increases potassium conductance in isolated neurons in vitro and intensifies conduction block of local anesthetic.[8] Roh et al.[7] recently suggested that one of the mechanisms for the enhanced potency of intrathecal clonidine administration in a rat model of neuropathic pain is its ability to suppress phosphorylation of NMDA receptor subunit NR 1 in spinal dorsal horn neuron of rats.

Traditionally, clonidine has been used as an antihypertensive agent since the late 1960. Its primary effect is sympatholysis and it reduces peripheral norepinephrine release by stimulation of the prejunctional inhibitory α2-adrenoceptors. Further uses based on its sedative, anxiolytic and analgesic properties are being developed. Systemic administration of clonidine improves the analgesic effects of anti-inflammatory agents and has peripheral (intraarticular, intravenous, regional) antinociceptive effects in combination with local anaesthetics, opioidsand ketamine.[2]

ONSET OF SENSORY AND MOTOR BLOCKADE: Gurudatta et al[9]: In a prospective randomized controlled study conducted on 50 patients concluded that the mean time for onset of sensory

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blockade was faster in group BC (Clonidine group) 1.62±0.85 min compared to group B (Bupivacaine group) 2.24±1.04 min which was highly significant with p value 0.000. The mean time for onset of motor blockade was faster with 1.96 ±1.55 min in BC group compared to 2.44±1.16min in group B which was significant with P value 0.017.

B. S. Sethiet al,[3]**:** In his study with 60 patients evaluated the effect of low dose 1μ/kg, intrathecal clonidine as adjuvant to bupivacaine and found that the onset of action was clinically and statistically significant with faster onset in clonidine group compared to bupivacaine groups.

Our result correlates with the above-mentioned study. Hence we conclude that addition of clonidine has a faster onset and longer duration of sensory and motor blockade.

TIME FOR PEAK SENSORY LEVEL AND HIGHEST SENSORY LEVEL BLOCKADE:
Dobrydnjov,[10]**:** In his comparative study of different doses of clonidine of 15μ (BC15) and 30μ (BC30) combined with small dose of bupivacaine during spinal anesthesia concluded that the highest level of sensory analgesia was T10 in bupivacaine group, T6 in group BC 15 and T8 in group BC30.

Which was clinically and statistically significant among the clonidine group our result correlates with the above-mentioned study hence we conclude that addition of clonidine intrathecal to hyperbaric bupivacaine results in higher level of sensory blockade and faster onset when compared to bupivacaine.

TIME FOR TWO SEGMENT REGRESSION:
Recovery Parameters: The time for two segment regression was considerably prolonged in group BC with 139.2 minutes and in group B it was 83.3 minutes.
Dobrydnjov,[10]**:** In his comparative study with different doses of clonidine, 15μ and 30μ with plain hyperbaric bupivacaine in spinal anesthesia conducted with 45 patients concluded that time to 2 segment regression was 95 min with plain bupivacaine group compared to 109 min in 15μ clonidine groupBC15 and 126 min in group BC30 which was highly significant.

B.S. Sethi et al.[3]**:** In his study concluded the mean time to regression of level of sensory analgesia by two segments was 218 min (Range 150-240min) in the clonidine group which was significantly longer than duration of 136 min (Range90-150 min) in bupivacaine group (p<0.001). Results from our study correlates with the above studies we conclude that time to regression of sensory analgesia by two segments is longer in clonidine group compared to bupivacaine group. 2 segment regression time was prolonged to almost 160% in our study, much higher percentage than in study of Dobrydnjov.[10] and can be explained by lower dose and strength of bupivacaine used by them. Moreover, the control values of the duration of sensory and motor block were lower in our study as compared to other studies.[3,10]

TIME FOR COMPLETE SENSORY AND MOTOR RECOVERY: Gurudutta et al,[9]**:** His study concluded that sensory recovery in group BC was 327 minutes and in group B was 207 minutes . The motor blockade in group BC, was 290.8minutes and in group B was 150.0 minutes.

B. S. Sethi et al,[3]**:** In his study found that the duration of complete sensory recovery in clonidine group was 614 minutes and in control group 223 minutes.

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The motor blockade was 203 minutes in clonidine group and in control group it was 136 minutes. The number of injections of diclofenac required in 24 hours was also significantly higher in control group. The prolongation of motor block in our study was comparable to studies by Strebel, et al,[11] and B. S. Sethi, et al.[3] in spite of higher volume) or higher dose of clonidine. Clonidine produced significantly longer motor blockade. Intrathecal clonidine alone even in doses Up to 450µg, does not induce motor block or weakness.

In contrast intrathecal clonidine combined with local anaesthetics significantly potentiates the intensity and duration of motor blockade. The explanation could be that alpha 2 adrenergic agonists induce cellular modification in the ventral horn of the spinal cord (motor neuron hyper polarization) and facilitates local anesthetic action. Hence we conclude that use of 30µg clonidineintrathecally results in increased intensity, prolonged sensory and motor blockade.

DURATION OF ANALGESIA: Total analgesia time was prolonged in our study similar to Strebel et al,[11] and Sethi et al[3]. It was higher than Dobrydnjov et al.[10] which is as expected considering the different doses of clonidine or bupivacaine used. We found a better quality of block in clonidine group where no supplementation with General anesthesia for 'relaxation" requests from the surgeon.

POSTOPERATIVE ANALGESIA: Gurudatta et al.[9] in this study demonstrated the duration of complete analgesia with 75µg of intrathecal clonidine was 327 min compared to 207 minutes in bupivacaine group which was highly significant. The 6 hour postoperative requirement of diclofenac injection was less in clonidine group. B. S. Sethi et al.[3] in their study found out that the duration of effective analgesia defined as the time from intrathecal injection to the time of first analgesic requirement was significantly prolonged with addition of clonidine (614 mins) and bupivacaine group (223 mins) respectively. No patient in the clonidine group required additional intraoperative analgesics compared with 17.6% in the Bupivacaine group alone. There was improved patient comfort and reduced need for intra-muscular and intravenous analgesia in the immediate postoperative period. Our results were similar to the above studies.

VITAL PARAMETERS:
HAEMODYNAMICS - HEART RATE & BLOOD PRESSURE: We did not see statistically significant hypotension in any clonidine group at any point of time compared to the control group which was in Accordance with the findings of Strebel et al,[11] and in contrast to the findings of Neimi, et al.[12] and B. S. Sethi etal,[3] who used higher doses of clonidine. Niemi, et al,[12] used 3 µg.kg-1 of clonidine added to 15mg of 0.5% bupivacaine for knee arthroscopy, Their values for MAP were significantly lower than Control group after 45 min to 8 hrs.

SIDE EFFECTS: Dobrydnjovetal,[10]**:** In his study concluded that small dose of intrathecalclonidine is not usually associated with systemic side effects such as bradycardia, hypotension or sedation.
Kaabachi et al.[13]: In his study concluded that intrathecal clonidine at 1µg/kg prolonged spinal anaesthesia without causing severe adverse effects.

B. S. Sethi et al,[3]: The results of their study showed that addition of 1µg.kg-1of clonidine to intrathecal bupivacaine is safe and likely to be as effective as higher dosages minimizing the side effects. Our results were similar to the above studies.

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CONCLUSION: Based on the present clinical comparative study, we conclude that the addition of 30μg clonidine to 0.5% hyperbaric Bupivacaine 12.5mg (2.5mL) in spinal anesthesia significantly decreases the onset time, prolongs the duration of both sensory and motor blockade. It prolongs the duration and improves the quality of postoperative analgesia with better hemodynamic stability and good sedation as compared to bupivacaine alone. It is an attractive alternative to opioids for prolonging spinal anesthesia. Clonidine will expand the scope and improve the reliability and efficacy of regional anesthesia.

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AUTHORS:
1. Shilpashri A. M.
2. Priodarshi Roychoudhury
3. Girish K. M.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Anaesthesiology, JJM Medical College, Davangere.
2. Post Graduate Student, Department of Anaesthesiology, JJM Medical College, Davangere.

FINANCIAL OR OTHER COMPETING INTERESTS: None

3. Post Graduate Student, Department of Anaesthesiology, JJM Medical College, Davangere.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Shilpashri A. M,
#1906, 2nd Cross,
S. S. Layout, 'A' Block,
Davangere-577004
E-mail: shilpashri.am@gmail.com