e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(10); 509-516

Original Research Article

Effect of Intravenous Dexmedetomidine on Duration of Spinal Anaesthesia with Hyperbaric Bupivacaine in Infraumbilical Surgeries

Reshma Radhan[1], Nazima Memon[2], Vaishnavi Vishwas Kulkarni[3], Minakshi Chole[4]

[1]Junior Resident, Department of Anaesthesiology, Dr. SCGMC, Nanded
[2]Associate professor, Department of Anaesthesiology, Dr. SCGMC, Nanded
[3]Professor and Head, Department of Anaesthesiology, Dr. SCGMC, Nanded
[4]Assistant Professor, Department of Anaesthesiology, Dr. SCGMC, Nanded

Received: 15-08-2025 / Revised: 14-09-2025 / Accepted: 15-10-2025
Corresponding Author: Dr. Minakshi Chole
Conflict of interest: Nil

Abstract:

Introduction: Hyperbaric bupivacaine is routinely used for spinal anaesthesia in surgery of the lower abdomen, pelvis, orthopaedics, and urology due to its rapid onset, streamlined peak action, favourable ratio of sensory to motor blockade, haemodynamic stability, as well as safety. Notwithstanding these benefits, the duration that spinal anaesthesia is effective with hyperbaric bupivacaine is often shorter than the surgical time, particularly for surgeries longer than 90–120 minutes, or for cases where extended postoperative analgesia is needed. Intravenous dexmedetomidine appears to prolong spinal anaesthesia through several central and peripheral mechanisms. The present study was conducted to evaluate the impact of administering a single bolus dose of intravenous dexmedetomidine at 0.5 microgram/kg on the duration of spinal anaesthesia with hyperbaric bupivacaine for infraumbilical surgeries.

Methods: The present study was conducted in the department of anaesthesiology of the tertiary care center during Feb.2023 to July 2024 amongst 70 patients, categorised with ASA physical status I and II, within the age range of 18 to 65 years and were slated for infraumbilical surgeries. Patients were allocated into two groups using a non-randomised, sequential allocation method based on the order of presentation. Group D: Prior to the administration of subarachnoid block, patients received an infusion of dexmedetomidine at a rate of 0.5 micrograms per kilogram, diluted in 100 millilitres of normal saline and given intravenously over a period of 10 minutes. Group S: Prior to the administration of subarachnoid block, patients received an infusion of Normal saline, 100 ml, intravenously over a 10-minute period.

Results: The systolic blood pressure exhibited a significant reduction ($p < 0.05$) from baseline for both groups. The group-D demonstrated lower DBP values compared to the other group at all time points post baseline. Patients in the Dexmedetomidine group had a markedly quicker average time to onset of (1.10 ± 0.40 min), also significantly longer mean time to regression of the sensory block (Mean time Dexmedetomidine – 213.18 ± 21.48 minutes; Group S – 139.78 ± 18.15 minutes), $p < 0.01$ and showed statistically significant prolongation in regression time of motor block and sensory block recovery. The patients in the group -D had the first rescue analgesia time significantly prolonged in comparison to the Group S, with the Dexmedetomidine group mean being 262.52 ± 39.05 minutes and the saline group mean being 183.15 ± 26.02 minutes, $p < 0.001$.

Conclusion: Therefore, to put it succinctly, administering intravenous dexmedetomidine in conjunction with hyperbaric bupivacaine during sub-umbilical surgeries markedly prolonged both motor and sensory levels of anaesthesia.

Keywords: Dexmedetomidine, Subarachnoid Block, Infraumbilical Surgery, Rescue Analgesia, Motor Block.

Introduction

For most infraumbilical procedures, spinal anaesthesia continues to be the technique of choice as it offers dense neuraxial blockade, superior postoperative analgesia, and diminished perioperative morbidity compared to general anaesthesia.[1] Hyperbaric bupivacaine is routinely used for spinal anaesthesia in surgery of the lower abdomen, pelvis, orthopaedics, and urology due to its rapid onset, streamlined peak action, favourable ratio of sensory to motor blockade, haemodynamic stability, as well as safety. Notwithstanding these benefits, the duration that spinal anaesthesia is effective with hyperbaric bupivacaine is often shorter than the surgical time, particularly for surgeries longer than 90–120 minutes, or for cases where extended postoperative analgesia is needed.[2]

Over the last decades clinicians have sought to extend spinal anaesthesia through both intrathecal and systemic adjuvants—opioids, α_2-agonists, neostigmine, magnesium, ketamine, and corticosteroids among them. [3] Intrathecal administration of opioids is largely associated with pruritus, nausea, and respiratory depression. Conversely, intrathecal administration of α_2 agonists is linked with neurotoxicity and haemodynamic alterations, which is why these medications are mainly given via intravenous routes.[4] An ideal systemic adjunct should synergise with intrathecal local anaesthetics, enhance sedation and analgesia, preserve respiratory function, and maintain a stable haemodynamic profile. Dexmedetomidine, a α_2-adrenergic agonist, satisfies most of these criteria.

Intravenous dexmedetomidine appears to prolong spinal anaesthesia through several central and peripheral mechanisms. Dexmedetomidine has central sympatholytic action (via α2-adrenergic receptors), when given IV, it acts on locus ceruleus in the brainstem to reduce sympathetic outflow and enhance parasympathetic tone, thus potentiating spinal blockade. Inhibition of nociceptive transmission. Within the spinal cord, the dorsal horn region has the release of substance P and glutamate inhibited. This enhances the blockage of the sensory input and afferent pain signal transmission. Spinal blockade causes sympathectomy below the blockade level, reducing systemic vascular resistance and preload. When given intravenously, dexmedetomidine further suppresses central sympathetic tone while enhancing vagal activity, contributing to bradycardia and hypotension. [5] Intravenous dexmedetomidine improves overall anaesthetic quality, patients experience anxiolysis, preserved arousability and smoother emergence, producing higher satisfaction scores.[6] Dexmedetomidine's anti-shivering effect, by resetting the hypothalamic thermoregulatory threshold, is particularly valuable given the high incidence of neuraxial shivering.[7]

The present study was conducted to evaluate the impact of administering a single bolus dose of intravenous dexmedetomidine at 0.5 microgram/kg on the duration of spinal anaesthesia with hyperbaric bupivacaine for infraumbilical surgeries. Hyperbaric bupivacaine is favoured due to its faster onset of both motor and sensory blockade compared to isobaric bupivacaine.

Material and Methods

The present study was conducted in the department of anaesthesiology of the tertiary care center during Feb.2023 to July 2024 amongst 70 patients, categorised with ASA physical status I and II, within the age range of 18 to 65 years and were slated for infraumbilical surgeries. All patients were screened with a detailed history and physical examination. Patients were allocated into two groups using a non-randomised, sequential allocation method based on the order of presentation. Group D: Prior to the administration of subarachnoid block, patients received an infusion of dexmedetomidine at a rate of 0.5 micrograms per kilogram, diluted in 100 millilitres of normal saline and given intravenously over a period of 10 minutes. Group S: Prior to the administration of subarachnoid block, patients received an infusion of Normal saline, 100 ml, intravenously over a 10-minute period.

An assessment was carried out before commencing the anaesthesia. Appropriate lab tests including complete blood count, blood sugar, electrolytes, blood urea, serum creatinine, liver function tests, urinalysis, chest X-ray, and ECG were conducted. The systematic approach of the evaluation was explained to the patient using simple and relatable language.

Data collection tool: Multichannel monitors in operating theatres record the pulse rate, blood pressure (measured non-invasively), electrocardiogram (ECG), and pulse oximetry (SpO2).

Inclusion Criteria: patients with ASA grade I and II, duration of surgery less than three hours, surgical procedures performed below the umbilicus, age between 18 to 65 years of both gender, voluntary and informed participants.

Exclusion Criteria: Individuals classified as ASA grade 3 and 4, patients under the age of eighteen and over the age of sixty-five, patients unwilling to give their consent, patients with known hypersensitivity to the research drug, any contraindication to spinal anaesthesia. Procedures included inguinal herniorrhaphy, varicocelectomy, cystolithotomy, tibial nailing, ankle arthrodesis, and proximal femoral plating. Monitors for checking the pulse rate, blood pressure, ECG, and pulse oximetry, and other relevant vitals.

Methodology

The procedure included obtaining peripheral intravenous (IV) access, and standard ASA monitoring of heart rate (HR), mean arterial pressure (MAP), and peripheral oxygenation (SpO_2) blood oxygen level which was measured with ECG, BP, and pulse oximetry. Participants underwent preloading of Ringer's Lactate Solution (10 mg/kg) 15-20 minutes prior to receiving the subarachnoid block. Based on the groups, participants were either infused with dexmedetomidine 0.5 microgram/kg in 100 ml of normal saline, or were given 100 ml of normal saline, thereby maintaining concealed allocation. Spinal anaesthesia was performed after infusion of dexmedetomidine. In both arms of the study, after verifying CSF flow, 3ml (15mg) of 0.5% bupivacaine was given. The standard care was

preserved in both groups which includes the administration of bupivacaine in large quantities.

Intra-operative monitoring and management:

The vital signs of interest, heart rate (HR), mean arterial pressure (MAP) as well as pulse oximetry (SpO_2) were noted at baseline and after every five minutes for the first half an hour, and subsequently for the next thirty minutes, at fifteen-minute intervals until the patient exited the operating theatre. Evaluation of sensory blockade by the pin prick method. Grading of motor blockade is done using the modified Bromage scale.

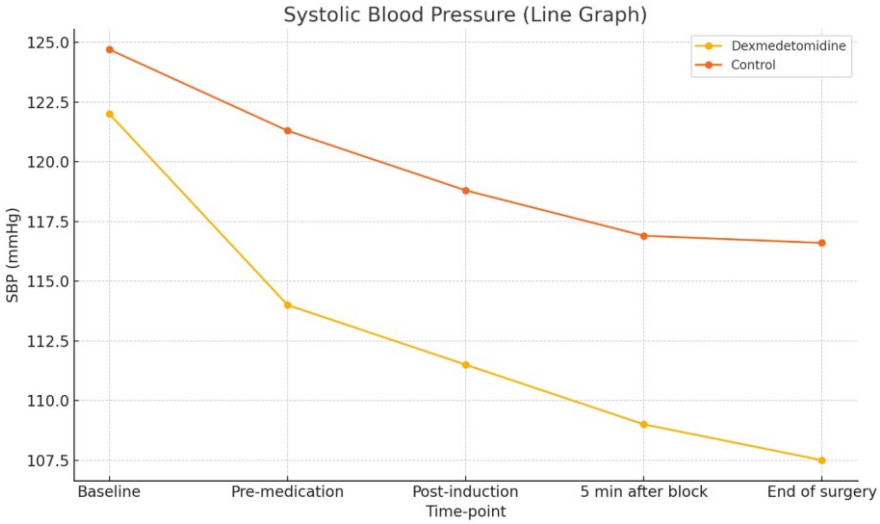
Data-collection procedures: The sensory block time interval commenced with the intrathecal injection and concluded with regression to the S1 level. The interval for the motor block commenced with the injection and concluded when the modified Bromage score of 0 was achieved. Sedation was monitored every 15 minutes using the Ramsay Sedation scale ranging from 1 to 6. The total dexmedetomidine dose and its intraoperative total administered time, the procedure time, intraoperative blood loss, and crystalloids and/or colloids administered were all noted. Shivering quantification was noted using the bedside scale for shivering on a range of 0 to 3 at the previously predetermined timeframes.

Post-operative management: Hemodynamic monitoring like HR, MAP, respiratory rate, SpO_2 and pain scores (measured through VAS 0–10) which were recorded every 15 minutes in the initial hour and consecutive measurements were taken hourly for six additional hours. Postoperative patients received first line intravenous Tramadol 100mg when their VAS score reached 4 or higher. The second line rescue medication consisted of intravenous tramadol dosage at 50 mg. The modification of Aldrete criteria served as the post anaesthesia care unit discharge assessment tool. Degree of Sensory response was assessed by pin prick (Hollmen scale). Ramsay sedation score helped evaluate levels of sedation.

Results

Table 1: Comparison of Systolic BP (mm Hg).

Time-point	Group D	Group S	P value
Baseline	122.00 ± 12.70	124.70 ± 8.90	0.8652
At the time of giving dexmedetomidine	114.00 ± 10.00	121.30 ± 9.00	0.00001
After spinal anaesthesia	111.50 ± 10.50	118.80 ± 9.10	0.00003
5 min after spinal anaesthesia	109.00 ± 10.00	116.90 ± 9.10	0.0001
End Surgery	107.50 ± 10.50	116.60 ± 9.80	0.00001

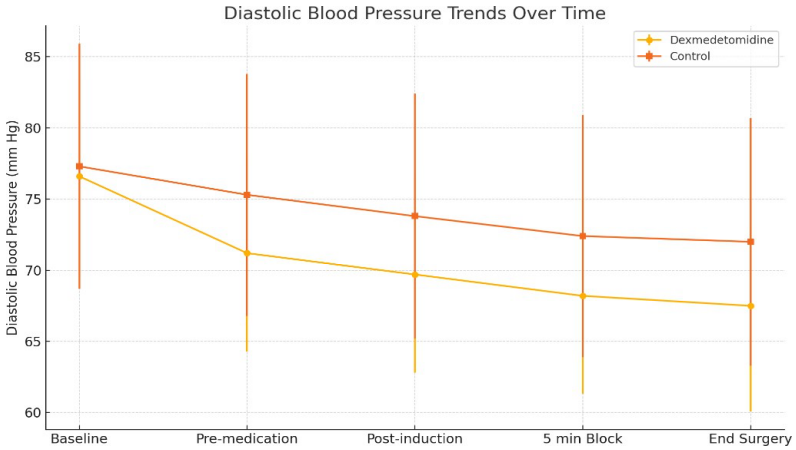


Graph 1: Comparison of Systolic BP (mm Hg)

Table no.1 and graph no.1 shows that the systolic blood pressure exhibited a significant reduction ($p < 0.05$) from baseline for both groups.

Table 2: Comparison of Diastolic BP (mm Hg).

Time-point	Group D	Group S	P value
Baseline	76.60 ± 7.10	77.30 ± 8.60	0.256
At the time of giving dexmedetomidine	71.20 ± 6.90	75.30 ± 8.50	0.2653
After spinal anaesthesia	69.70 ± 6.90	73.80 ± 8.60	0.0001
5 min after spinal anaesthesia	68.20 ± 6.90	72.40 ± 8.50	0.001
End Surgery	67.50 ± 7.40	72.00 ± 8.70	0.0005



Graph 2: Comparison of diastolic BP (mm Hg)

Table no.2 and graph no.2 shows that the both cohorts displayed a decrease in diastolic blood pressure (DBP) over time; however, the Dexmedetomidine group demonstrated lower DBP values compared to the other group at all time points post baseline. It is well documented that Dexmedetomidine is effective in lowering diastolic blood pressure significantly (p-value < 0.05) relative to the control group throughout the entire perioperative timeframe.

Table 3: Comparison of Onset Time of Sensory Block.

Parameter	Group Dexmedetomidine (n=35)	Group Saline (n=35)
Time for Onset of Sensory Block (min)	**1.10 ± 0.40**	**2.30 ± 0.60**
p-value	**<0.0001**	

Table no.3 shows that the average time taken to disarm a patient's sensation in two different groups: Saline and Dexmedetomidine. Patients in the Dexmedetomidine group had a markedly quicker average time to onset of (1.10 ± 0.40 min) compared to the Saline group's (2.30 ± 0.60 min). The intergroup difference is remarkably strong with a p value of < 0.0001.

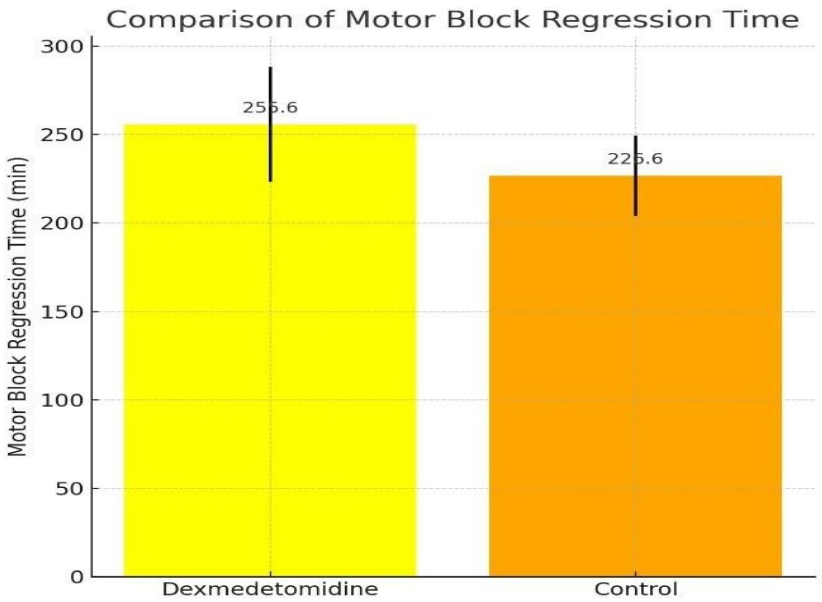
Table 4: Comparison of Sensory Block Regression Time.

Parameter	Group Dexmedetomidine (n=35)	Group S (n=35)
Sensory Block Regression Time (min)	**213.18 ± 21.48**	**39.78 ± 18.15**
p-value (t-test)	**<0.0001**	

Table no. 4 shows that the relative to the group S, the Dexmedetomidine group demonstrated a significantly longer mean time to regression of the sensory block (Mean time Dexmedetomidine – 213.18 ± 21.48 minutes; Group S – 139.78 ± 18.15 minutes), p < 0.01.

Table 5: Comparison of Motor Block Regression Time (min).

Parameter	Group D (n=35)	Group S (n=35)
Motor Block Regression Time (min)	255.62 ± 32.58	226.60 ± 22.74
p-value (t-test)	<0.001	

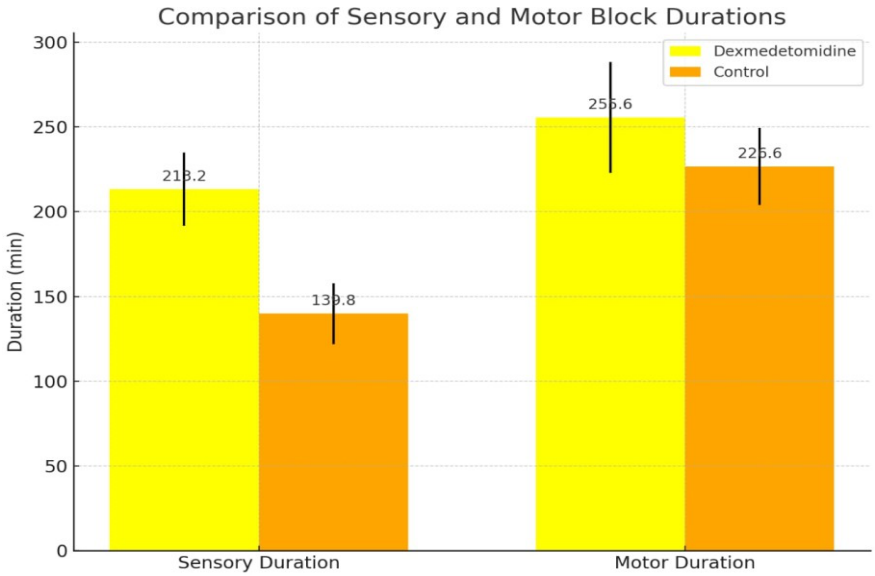


Graph 5: Comparison of Motor Block Regression Time (min).

Table no.5 and graph no.3 shows that the participants receiving dexmedetomidine showed a statistically significant prolongation in regression time of motor block recovery when compared to the Group S, dexmedetomidine (mean: 255.62 ± 32.58; Group S 226.60 ± 22.74; significant p value < 0.001).

Table 6: Comparison of Sensory & Motor Block Duration (min).

Parameter	Group D	Group S	p-value
Sensory Duration	213.20 ± 21.50	139.80 ± 18.10	<0.0001
Motor Duration	255.60 ± 32.60	226.60 ± 22.70	



Graph 6: Comparison of Sensory & Motor Block Duration (min).

Table no.6 and graph no.4 shows that the participants in the Dexmedetomidine group not only had significantly prolonged durations of sensory block to 213.2 ± 21.5 minutes and motor block to 255.6 ± 32.6 minutes, but also had better outcomes than the Group S group which recorded 139.8 ± 18.1 and 226.6 ± 22.7 minutes respectively. The differences observed were highly significant (p < 0.00001).

Table 7: Time to First Rescue Analgesia (min)

Group	Mean ± SD	p-value
GROUP D	262.52 ± 39.05	
GROUP S	183.15 ± 26.02	< 0.001

Table no.7 shows that the patients in the Dexmedetomidine group had the first rescue analgesia time significantly prolonged in comparison to the Group S, with the Dexmedetomidine group mean being 262.52 ± 39.05 minutes and the saline group mean being 183.15 ± 26.02 minutes, p < 0. 001.This result demonstrates the analgesic-sparing effect of intravenous dexmedetomidine which was able to prolong the relief of postoperative pain. This effect, in practice, may improve satisfaction and the need for analgesics shortly after surgery.

Discussion

In this case, age, height, and weight, as well as sex, were all congruent and comparable. ASA physical status, as well, was comparably in alignment with age and height metrics; the variance was minimal.

Manjunath Reddy et al,[8] performed a double-blind randomised prospective study and found no statistically significant difference considering age, height, weight, sex, and ASA physical status as demographic variables. Similar, results were observed by Fazil k [9] and Philip et al. [10]

Bupivacaine dosage: The bupivacaine dosing for both groups was 3 ml of 0.5% hyperbaric bupivacaine. Kubre et al,[11] conducted a randomised double-blinded study where participants were administered 3 ml of 0.5% hyperbaric bupivacaine and intravenous dexmedetomidine infusion for elective surgeries located beneath the umbilicus. Bharti V et al,[12] also utilised intravenous doses of dexmedetomidine and butorphanol as adjunctive sedation during the procedure; I have also employed 3 ml of 0.5% hyperbaric bupivacaine for lower abdominal surgeries.

Dexmedetomidine dosage: Manjunath Reddy et al [8] utilised dexmedetomidine at a dose of 0.5 mcg / kg IV, which was aligned with the objectives of this study. Kubre et al [11] who conducted a prospective randomised double-blind study administering IV 0.5 mcg/kg dexmedetomidine. Hence, this research administered dexmedetomidine 0.5 mcg/kg IV to evaluate its effect on subarachnoid block with hyperbaric Bupivacaine augmentation.

Time to onset of sensory block (T-10 level): According to the findings, the onset time of the sensory block to the T10 level was 1.10 ± 0.40 minutes for group D and 2.30 ± 0.60 minutes for group S. Hence group S was slower than group D. Therefore, group S experienced a slower onset time. The time difference was significant (p-value = 0.000). Manjunath Reddy et al [8] findings are comparable with the onset time of the sensory block (T10 level) which was 1.53 ± 1.27 minutes for group D and 3.15 ± 1.34 minutes for group C. There was a substantial difference between the two groups (p value 0.001). SS Harsoor et al [13] found that, 12.5 mg of hyperbaric bupivacaine indicated that dexmedetomidine provided a more rapid sensory block to the T10 level, achieving it in an average of 1.06 minutes in the dexmedetomidine group compared to 2.09 minutes in the control group. This has significance with a p value of 0.001.

Maximum level of sensory block: In this study, peak sensory block occurred at 3.90 ± 0.70 minutes for the dexmedetomidine group and 5.00 ± 0.80 minutes for the control group. Statistically significant differences were observed for both groups (p < 0.001). Manjunath Reddy et al, [8] conducted studies which showed that Group D outperformed Group C in attaining the maximum sensory block at the T6 level—Group D at 4.91 ± 2.4 mins and Group C at 5.82 ± 2.0 mins. The difference was statistically significant with a p-value of 0.044.

Sensory block duration: The present study shows that the difference in sensory block duration between groups D and S, where group D exhibited a significantly greater duration of sensory block at 213.20 ± 21.5 mins compared to group S's 139.8 ± 18.1 mins. The noted difference was statistically significant (p = 0.000). Shadab et al 2021[14] conducted research which demonstrated Group D having a significantly greater duration of the sensory block of 223.83 ± 12.64 min as opposed to the control group's 180.83 ± 17.27 min. Bhalerao et al 2019[15] conducted their study to assess the impact of intravenous dexmedetomidine on the sensory and motor block characteristics and the associated haemodynamic parameters and sedation during subarachnoid block. The total sensory duration was (260.125±9.233 min) extended in Group D compared to (200.000±9.199 min) in Group C.

Time for two dermatomal regressions: Within the dexmedetomidine group, the average duration for two dermatomal regressions of sensory blockade was 114.83 ± 12.69 minutes compared to 86.83 ± 12.28 minutes in the control group (saline). This difference was found to be significant with a P value of < 0.001. Other studies which demonstrated the same findings are: SS Harsoor et al in 2013 [13] observed that, for Group D the duration taken for two segment regression was 111.52±30.9 minutes

whereas for Group C, the duration was 53.6±18.22 minutes. Pranav Jetley et al [16] conducted a study which showed that the time to achieve two segment regression time was longer with dexmedetomidine (146.5±12.5 min) and clonidine (138.9±17.4 min) when compared to control (90.1±9.4 min; P<0.001).

Time of onset for motor block: The current study demonstrates a trend towards more rapid onset of motor block both in the dexmedetomidine group (3.20 + 0.50 min) and in the saline (control) group (4.20 + 1.0 min) p<0.01 which is significant. SS Harsoor et al [13] in their study also noted the onset of motor block as group D (3.76±2.02) and group C (4.2±2.08). Shadab et al 2021[14] concluded in their study that the onset of motor block was documented as group D (2.17±0.53), while group C had (3.87±0.52).

Duration of motor block: In the present study Group D exhibited a larger mean duration of motor block at 255.60 ± 32.60 mins compared to group S 226.60 ± 22.7 mins. This discrepancy proved to be significant with a p value of 0.000. Mi Hyeon Lee et al [17] observed that the control group in the study exhibited an average motor block duration of 98.8 ± 34.1 minutes, whereas in group D-0.5 this duration increased to 132.9 ± 43.4 minutes. Group D-0.5, as the designation implies, had received some form of intervention. This change was statistically significant as well (p < 0.05). Saravanan et al 2019[73] Participants in the control group had an average motor block duration of 98.8 ± 34.1 minutes, whereas those in group D-0.5 had an average duration of 132.9 ± 43.4 minutes. The participants ingroup D-0.5 had received some form of intervention which explains the greater average motor block time in this group. This difference was also significant with a p value less than 0.05.

Duration of analgesia: In this study, the mean duration of analgesia for group S, 183.15 ± 02 minutes, was markedly less than group D's 262.52 ± 39.05 minutes (p-value < 0.000). This thus corroborates several studies indicating that the intravenous infusion of dexmedetomidine does, indeed, prolong the duration of analgesia. Manjunath Reddy et al [8] showed that Group D had a significantly higher mean duration of analgesia as compared to Group C. Group D 218.80±11.36 mins while Group C 178.60 ± 17.96 mins. p = 0.001. Kubre et al [11] Analysis showed that group D's analgesic duration was 234.67 ± 7.64 mins while group C's was 164.17 ± 6.17 mins p value .001.

Sedation level: In the present study, the Ramsay sedation score was applied to evaluate the sedation level. It is noteworthy that Group S lagged behind Group D consistently, Group D scoring (3.6 + 0.6) and Group S scoring (2.1 + 0.3). There was a substantial difference between the two groups (p value < 0.0001). Dinesh et al [18] found that during the intraoperative period, Ramsay sedation scores were higher for group D as compared to group C. The difference noted was 0.001 significant in definitional terms. SS Harsoor et al [13] conducted a prospective randomised double-blinded study to assess the effects of intravenous dexmedetomidine on motor, sensory functions, haemodynamic parameters, and sedation levels in patients undergoing surgeries of the lower abdomen and lower limbs with subarachnoid block. This study demonstrated that the mean intraoperative Ramsay sedation score was more pronounced in group D as opposed to group C.

Hemodynamic stability: The most recent examination noted a reduction in heart rate and mean arterial blood pressure in the dexmedetomidine group after premedication. Verghese et al (2019) [19] Performed a randomised, double-blind study with 50 participants earmarked for lower limb surgeries. This study noted a considerable reduction in heart rate and blood pressure. Reddy et al (2023) [20] Performed a prospective randomised comparative study on one hundred participants undergoing infraumbilical surgeries to examine the impact of intravenous dexmedetomidine on the 0.5% hyperbaric bupivacaine spinal anaesthesia. It also noted a considerable rise in the prevalence of bradycardia which highlighted the importance of haemodynamic monitoring with dexmedetomidine as a supplement. Considering other studies and literature, during this study approximately 20% of patients in Group D and 33% of patients in Group C demonstrated bradycardic and hypotensive effects. Most other complications that developed were quite minor in nature and relatively straightforward to manage.

Adverse effects: In the present observation, episodes of bradycardia and hypotension were documented and such instances were controllable. Mi Hyeon Lee et al [17] found that in both the I-V and D groups demonstrate instances of bradycardia alongside hypotension. D-1 does not demonstrate hypotension. D-1's bradycardia exhibited the highest average value measurement. Shadab et al[14] in their study studied the effects of intravenous dexmedetomidine on the characteristics of spinal anaesthesia with hyperbaric bupivacaine. Also, they emphasise the significant reduction in heart rate and blood pressure observed in the group administered dexmedetomidine compared with the control.

Conclusion

The current study has reached the following conclusions: The addition of dexmedetomidine accelerated both sensory and motor blockade onset, two segment regression time was prolonged, motor block was more pronounced, the duration of analgesia was significantly longer and haemodynamically patients were stable, the Ramsay

sedation score was significantly elevated in the dexmedetomidine group Therefore, to put it succinctly, administering intravenous dexmedetomidine in conjunction with hyperbaric bupivacaine during sub-umbilical surgeries markedly prolonged both motor and sensory levels of anaesthesia.

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