

Original Research Article

# To study the onset, duration and characteristics of sensory and motor blockade produced by combination of Midazolam and bupivacaine

Dr. Ankit Gupta<sup>1</sup> (Asst. Professor), Dr. Sonali Savarn<sup>2</sup> (3rd year resident),  
Dr. Saniya Khan<sup>3</sup> (2nd year Resident) & Dr. Tanya Jain<sup>4</sup> (3rd year Resident)

Department of Anaesthesiology, Rajshri Institute of Medical Sciences, Bareilly, U.P.<sup>1</sup>  
Department of Anaesthesiology, Index Medical College Hospital and Research Centre,  
Indore, M.P.<sup>2,3&4</sup>

Corresponding Author: Dr. Tanya Jain

## Abstract:

**Background & Method:** The aim of this study is to study the onset, duration and characteristics of sensory and motor blockade produced by combination of Midazolam and bupivacaine. All patients received inj. Glycopyrrolate 0.2 mg intramuscularly half an hour before the procedure. No narcotics, benzodiazepines or antiemetics were given. Preoperatively patients underwent a careful and detailed examination. On the operation table and baseline data were entered in the proforma.

**Result:** Onset of sensory blockade was in the range of 121-180 seconds in majority of patients (n=16, 53.3%) in group I and (n=19, 63.33%) in group II. The difference in mean onset of analgesia among both the groups was statistically insignificant ( $P>0.05$ ), indicating that addition of Midazolam had not shortened the onset of sensory blockade. Onset of motor blockade was in the range of 241-300 seconds in majority of patients in both groups (n=17, 56.67%) in group I and (n=18, 60%) in group II. The difference in mean onset of motor blockade among both the groups was insignificant statistically ( $P>0.05$ ) indicating that addition of Midazolam had not shortened the onset of motor blockade.

**Conclusion:** The two groups did not differ significantly as regards to duration of surgery, time of onset of analgesia, onset of motor blockade, degree of motor blockade, efficacy of analgesia intraoperatively. A detailed preoperative assessment and routine investigations were done in all patients. Patients with absolute contraindications for subarachnoid block and those who are taking pain killers, benzodiazepine, MAO inhibitors and tricyclic antidepressants were excluded from the study. Intrathecal Midazolam with Bupivacaine 0.5% (heavy) produces excellent surgical analgesia and an extended analgesic in postoperative period.

**Keywords:** onset, sensory, motor blockade, midazolam and bupivacaine.

**Study Designed:** Observational Study.

## 1. INTRODUCTION

These are drugs which reversibly blocks nerve conduction beyond the point of application when applied locally in appropriate concentration without depressing consciousness or sensation of other parts of the body and causing no structural or functional damage to nervous tissue[1].

Many drugs have been tried since the introduction of cocaine. However, Lignocaine, bupivacaine, chlorprocaine are used widely now a days. Looking to the chemistry all local anesthetic drugs have a basic formula having aromatic lipophilic group, intermediate chain (i.e. ester coo-or amide-NHCO) and hydrophilic group (i.e. secondary tertiary amine.) [2].

Most of the local anesthetics currently in use are tertiary amines. The general configuration of the local anesthetic amines comprises two key structural components[3]. One imparting lipid solubility (Lipophilic) and other water solubility (hydrophilic). The Lipophilic and hydrophilic portions are connected by an intermediate hydrocarbon chain- which is usually as ester or an amide linkage[4].

A local anesthetic amine is poorly soluble in water and rather insoluble when exposed to air. This amine is a weak base and combines readily with acids to form salts. This salt is quite soluble in water and comparatively stable[5]. This is mainly based on the pH of the solution. The salt ionizes in solution and is usually stable. The non-ionized form (the base) is lipid soluble and can easily penetrate the tissue barrier.

The proportion of the two forms of the solution depends on the PKa and Ph of the solution. All local anesthetics act by stabilizing the membrane. Alkalinization of local anesthetic solution increases the speed of onset and increases the effectiveness of the block[6].

It is suggested that the local anesthetic prevents the increase in permeability of cell membrane to sodium ion which is first event in depolarization of cell. Thus an action potential is not generated. This action affecting the process of depolarization of an impulse without affecting the resting potential is known as membrane stabilizing effect.

## 2. MATERIAL & METHOD

The present study was carried out in the Department of Anaesthesiology, Index Medical College Hospital & Research Centre, Indore (M.P.) from Nov 2021 to Oct 2022.

### Patient selection

The study comprised of 120 patients of either sex in the age group of 20 to 50 years belonging to ASA I or II, scheduled for elective and emergency lower abdominal, gynaecological, pelvic and lower limb surgeries.

### Exclusion criteria

1. Patients having contraindication for spinal anaesthesia such as severe dehydration, hypovolemia, neurological deficit, deformities of spine and local sepsis, coagulopathy or unwilling patients were not included in the study.
2. All patients with history of chronic drug intake like pain killers, benzodiazepines, anticoagulants, MAO inhibitors, tricyclic antidepressants were excluded from the study.

All patients received inj. Glycopyrrolate 0.2 mg intramuscularly half an hour before the procedure. No narcotics, benzodiazepines or antiemetics were given. Preoperatively patients underwent a careful and detailed examination. On the operation table and baseline data were entered in the proforma.

### 3. RESULTS

Table-1: Age and Sex Wise Distribution

Age (Yrs)	Group I (n=60)			Group II (n=60)			Total (%)
	Male	Female	Total (%)	Male	Female	Total (%)	
21-30	10	08	18 (30%)	08	04	12 (20%)	30 (25%)
31-40	16	14	30 (50%)	20	14	34 (57%)	64 (53%)
41-50	04	08	12 (20%)	06	08	14 (23%)	52 (22%)
<b>Total</b>	<b>30 (50%)</b>	<b>30 (50%)</b>	<b>60</b>	<b>34 (56.67)</b>	<b>26 (43.33)</b>	<b>60</b>	<b>60</b>
<b>Mean Age SD</b>	<b>34.93 ± 7.36</b>			<b>36.43 ± 7.39</b>			

Majority of patients were in age group 31-40 years in both groups. This comprised of (53%) patients out of 120 patients of this study.

Mean age in group I was  $34.93 \pm 7.36$  years and in group II, it was  $36.43 \pm 7.39$  years.

Differences between both groups were statistically insignificant ( $P > 0.05$ )

Table-2: Onset of sensory blockade

On set (seconds)	Group-I		Group-II	
	No.	%	No.	%
61 – 120	04	6.67	06	10.00
121-180	32	53.3	38	63.33
181 – 240	22	36.67	14	23.33
241 – 300	02	3.33	02	3.33
Mean	184.83 ±		172.83 ± 37.10	
Range	100 – 300		90 – 280	
T=1.24P>0.05				

Onset of sensory blockade was in the range of 121-180 seconds in majority of patients (n=16, 53.3%) in group I and (n=19, 63.33%) in group II.

The difference in mean onset of analgesia among both the groups was statistically insignificant ( $P > 0.05$ ), indicating that addition of Midazolam had not shortened the onset of sensory blockade.

Table-3: Onset of motor blockade

On set (seconds)	Group-I		Group-II	
	No.	%	No.	%
121-180	04	6.67	04	6.67
181-240	02	3.33	06	10.00
241-300	34	56.67	36	60.00
301-360	10	16.67	10	16.67
361-420	10	16.67	04	6.67
Mean	302.6 ± 57.83		289 ± 53.33	
Range	180 – 400		180 – 400	
T=0.95P > 0.05				

Onset of motor blockade was in the range of 241-300 seconds in majority of patients in both groups (n=17, 56.67%) in group I and (n=18, 60%) in group II.

The difference in mean onset of motor blockade among both the groups was insignificant statistically (P>0.05) indicating that addition of Midazolam had not shortened the onset of motor blockade.

#### 4. DISCUSSION

The mean onset of motor blockade recorded in this study was 302.6±57.83 in group I and 289±53.33 in group II. The difference was found to be statistically insignificant indicating that Midazolam has no influence when added to local anaesthetics in respect to onset of sensory and motor blockade[7].

Receptors for benzodiazepine are present in the spinal cord and GABA receptor is believed to play a role in the blockade of nociception[8]. Midazolam acts mainly in the brain and the spinal cord on GABA receptor complex, not by blocking transmission of impulse through nerve fibres.

Maximum dermatomal level of sensory anaesthesia and modified Bromage scale at 15 minutes were comparable in both groups. Mean duration of motor blockade was 179 ± 23.33 minutes in group I and 210.4 ± 30.26 minutes in group II. The difference between both the groups was statistically significant (P<0.01)[9].

Prolonged motor blockade in the intrathecal Midazolam group compared to control group(P<0.01). In this study assessment of postoperative analgesia was done by visual analogue scale. This is a simple and reliable method for assessment of pain. Most of the authors working on pain have employed this and advocated its use because of its simplicity[10].

#### 5. CONCLUSION

The two groups did not differ significantly as regards to duration of surgery, time of onset of analgesia, onset of motor blockade, degree of motor blockade, efficacy of analgesia intraoperatively. A detailed preoperative assessment and routine investigations were done in all patients. Patients with absolute contraindications for subarachnoid block and those who are taking pain killers, benzodiazepine, MAO inhibitors and tricyclic antidepressants were excluded from the study. Intrathecal Midazolam with Bupivacaine 0.5% (heavy) produces excellent surgical analgesia and an extended analgesic in postoperative period.

## 6. REFERENCES

1. Raju PK. Ultrasound-guided brachial plexus blocks. *Br J Anaesth: Contin Educ Anaesth Crit Care Pain* 2014; 14: 185-91.
2. Tiwari P, Jain M, Rastogi B, Aggarwal S, Gupta K, Singh VP. A comparative clinical study of perineural administration of 0.75% ropivacaine with and without dexmedetomidine in upper limb surgery by ultrasound guided single injection supraclavicular brachial plexus block. *Global Anesth Perioper Med* 2015; 1: 131-3.
3. Shaikh SI, Veena K. Midazolam as an adjuvant in supraclavicular brachial plexus block. *Anaesth Pain Intensive Care* 2012; 16: 7-12.
4. Duma A, Urbanek B, Sitzwohl C, Kreiger A, Zimpfer M, Kapral S. Clonidine as an adjuvant to local anaesthetic axillary brachial plexus block: a randomized, controlled study. *Br J Anaesth* 2005; 94(1): 112-6.
5. Chattopadhyay A, Maitra S, Sen S. A study to compare the analgesic efficacy of intrathecal bupivacaine alone with intrathecal bupivacaine midazolam combination in patients undergoing elective infraumbilical surgery. *Anesthesiol Res Pract.* 2013;567134:1-5.
6. Joshi C, Hosalli V, Ganeshnavar AK. A comparative study of intrathecal bupivacaine and bupivacaine with midazolam in lower abdominal and lower limb surgeries - A prospective randomised double blinded study. *Int J Clin Diagn Res.* 2015;3(6):1-8.
7. Raghu R, Indira P, Kiran M, Radharamana M. A comparative study of 0.375% bupivacaine with midazolam and 0.375% bupivacaine for brachial plexus block in upper limb surgeries. *Asian Pac J Health Sci* 2015; 2: 129-35.
8. Cairns BE, Sessle BJ, Hu JW. Activation of peripheral GABAA receptors inhibits temporomandibular joint-evoked jaw muscle activity. *J Neurophysiol* 1999; 81(4): 1966-9.
9. Gupta A, Kamat H, Kharod U. Efficacy of intrathecal midazolam in potentiating the analgesic effect of intrathecal fentanyl in patients undergoing lower limb surgery. *Anesth Essays Res.* 2015;9(3):379- 83.
10. Kurmanadh K, Srilakshmi K. A comparative study of the effects of intrathecal midazolam and fentanyl as additives to intrathecal hyperbaric bupivacaine (0.5%) for spinal anaesthesia. *J Evol Med Dent Sci.* 2017;6(38):3061-4.