

# Addition of Butorphanol to Lidocaine prolongs duration of the Axillary Brachial Plexus Block

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## Abstract

Butorphanol is a morphinan-type synthetic opioid analgesic exhibiting partial agonist at the  $\mu$  opioid receptors and agonist activity at  $\kappa$  opioid receptors. We designed a prospective, randomized, double blind study to evaluate the effect of butorphanol added to lidocaine on the onset and duration of axillary brachial plexus blockade. Fifty patients scheduled for hand and forearm surgery under axillary brachial plexus block were randomly allocated to receive either 40mL of 1% lidocaine with 2mL of isotonic saline ( control group, n=25) or 40mL of 1% lidocaine with 2mL(2mg) of butorphanol ( butorphanol group, n=25 ). Sensory and motor blockade were recorded at 5, 15, and 30 minutes. The onset time of sensory and motor block was similar in the two groups whereas the duration of sensory and motor blockade were significantly longer in butorphanol group than in the control group. This study demonstrates that admixture of butorphanol with lidocaine for brachial plexus blockade provides a significant prolongation of the blockade.

Study conducted in Bone and Joint Hospital associated to Government Medical College Srinagar, India

## INTRODUCTION

It has always been desirable to increase the quality and duration of local anesthetic action as it prolongs surgical anesthesia and analgesia. During the past years, different adjuncts have been added to local anesthetics to prolong the block and reduce the toxicity. For axillary brachial plexus blockade different additives like tramadol <sup>1</sup>, dexamethasone <sup>2</sup>, and clonidine <sup>3</sup> have been added to local anesthetics like mepivacaine and lidocaine. Butorphanol has been used alone and in combination with a local anesthetic, like mepivacaine <sup>4</sup>, for axillary brachial plexus blockade. Butorphanol is a synthetically derived opioid agonist-antagonist analgesic of the phenanthrene series. It exhibits partial agonist and antagonist activity at the  $\mu$  opioid receptor and agonist activity at the  $\kappa$  receptor. Stimulation of these receptors on central nervous system neurons causes an intracellular inhibition of adenylyl cyclase, closing of influx membrane calcium channels, and opening of membrane potassium channels. This leads to hyperpolarisation of the cell membrane potential and suppression of action potential transmission of ascending pain pathways <sup>5</sup>. The aim of this placebo controlled study was to evaluate the effect of butorphanol added to lidocaine for an axillary brachial

plexus block on the onset and duration of blockade.

## METHODS

After institutional ethics committee approval and informed patient consent, 50 patients of ASA-I and ASA-II physical status aged 20 to 60 years scheduled for elective surgery of hand and forearm of moderate duration (<90 minutes) under axillary brachial plexus block were included in the study. Patients with diabetes mellitus, hepatic or renal failure, those receiving any premedication like opioids, benzodiazepines, clonidine, and those with any other contraindication to axillary brachial plexus block were excluded from the study. Patients were allocated randomly into one of two groups of 25 patients each in a double blind design to receive either 40mL of 1% lidocaine with 2mL of isotonic saline (control group), or 40mL of 1% lidocaine with 2mL (2mg) of butorphanol tartarate ( butorphanol group). Axillary brachial plexus block was performed with the patient in supine position and the upper arm abducted 90° and elbow flexed at 110° after establishing an IV line by an 18-gauge catheter in a peripheral vein in the contra lateral arm and establishing standard monitoring (pulse oximetry, electrocardiography, and noninvasive arterial blood pressure monitoring). We used transarterial technique to administer axillary brachial plexus block with 22-gauge, 1.5 inch long needle in all the patients. Half of the drug was injected into the sheath anterior to the artery where as remaining half was deposited

posterior to the artery. Patients who didn't receive the satisfactory level of anesthesia were excluded from the study.

We recorded the sensory and motor blockade of radial, median, musculocutaneous and ulnar nerves 5, 10 and 30 minutes after the block, and every 10 minutes after the end of surgery. The assessment of the sensory block of each nerve was done by pinprick and was compared with the same stimulation in the contra lateral hand. Sensory blockade of each nerve was rated by the patient on a verbal analog scale from 100% (normal sensation) to 0% (no sensation). Motor block was assessed by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion of the elbow in supination and pronation of the forearm (musculocutaneous nerve). Measurements were performed using a modification of the Lovett rating scale (table 1) from 6 (normal muscular force) to 0 (complete paralysis) <sup>2</sup>. The onset time of sensory and motor blockade was defined as the time between the end of last injection and the total abolition of the pinprick response, and complete paralysis in all of the nerve distributions respectively. The duration of sensory block was considered as the period from the administration of the block and the first postoperative pain; whereas the duration of the motor block was taken as the time interval between the local anesthetic administration and complete recovery of motor functions.

The data obtained was subjected to statistical analysis using Kruskal-Wallis test, Mann-Whitney U-test, and independent student's t-test.

**RESULTS**

12 patients were excluded from the study because they did fail to achieve satisfactory blockade. The mean patient age, weight, height and duration of surgery were similar in two groups (table 2). The time of onset of the blockade did not differ significantly in the two groups (table 3). The duration of both sensory and motor blockade was significantly longer in butorphanol group than in the control group (table 3). The degree of sensory and motor blockade did not differ significantly in any of the nerve distribution in the two groups (table 4).

**Figure 1**

Table 1: Rating Scale for Quantification of Muscle force

6	Normal muscular force
5	Slightly reduced muscular force
4	Pronounced reduction of muscular force
3	Slightly impaired mobility
2	Pronounced mobility impairment
1	Almost complete paralysis
0	Complete paralysis

**Figure 2**

Table 2: Demographic data

Variable	Control group	Butorphanol group
Age (yr)	44 ± 11	48 ± 8
Weight	68 ± 11	71 ± 13
Height (cm)	165 ± 6	167 ± 5
Surgery duration (min)	67 ± 11	66 ± 13

**Figure 3**

Table 3: The onset time and duration of the motor and sensory blockade

	Control group	Butorphanol group	p-value
<b>Onset (min)</b>			
Sensory block	10 ± 5	12 ± 3	0.067
Motor block	25 ± 6	23 ± 7	0.112
<b>Duration (min)</b>			
Sensory block	101 ± 35	240 ± 80	0.001
Motor block	125 ± 30	313 ± 81	0.001

**Figure 4**

Table 4: Sensory and Motor Blockade of axillary block

	Control group			Butorphanol group		
	5min	15 min	30 min	5 min	15min	30 min
<b>Motor block*</b>						
MC	4.1 ± 1.5	2.4 ± 1.5	1.3 ± 0.12	4 ± 1.3	2.4 ± 0.75	1.2 ± 0.51
RAD	3.7 ± 1.5	2.2 ± 1.05	1.4 ± 0.65	3.5 ± 1.4	2.2 ± 0.76	1.1 ± 0.22
MED	3.6 ± 1.6	2.3 ± 1.0	1.2 ± 0.55	3.7 ± 1.6	2.5 ± 0.53	1.1 ± 0.3
ULN	3.4 ± 1.4	2.3 ± 1.06	1.4 ± 0.6	3.1 ± 1.5	2.4 ± 0.66	1.0 ± 0.2
<b>Sensory block<sup>†</sup></b>						
MC	15%	70%	100%	15%	75%	100%
RAD	15%	80%	100%	15%	80%	100%
MED	25%	85%	100%	20%	80%	100%
ULN	15%	75%	100%	20%	80%	100%

MC = musculocutaneous nerve, RAD = radial nerve; MED = median nerve; ULN = ulnar nerve  
 \* motor blockade was measured using a modification of Lovett rating scale from 6 (normal muscular force) to complete paralysis.  
<sup>†</sup> Sensory blockade was assessed by pinprick and compared with the same stimulation on the contra lateral hand. Values show the percentage of no sensation in each nerve distribution.

**DISCUSSION**

The result of this study shows that the addition of 2mg of butorphanol to 40mL of lidocaine 1% for axillary brachial plexus blockade results in a significant increase in the duration of the block without effecting the time of onset of the blockade. For past many years, various agents like epinephrine, clonidine, opioids, steroids have been added to local anesthetics to improve the quality and increase the duration of anesthesia, and to reduce the toxicity of the local anesthetic. Kapral et al<sub>2</sub> added tramadol to mepivacaine for axillary brachial plexus blockade and found that the admixture 100mg tramadol with 1% mepivacaine provides a pronounced prolongation of the axillary brachial plexus blockade without side effects. Tramadol and its metabolites have been demonstrated to have action on opiate receptors<sub>6,7,8</sub>. Wajima et al<sub>9</sub> showed that continuous infusion of butorphanol locally into the brachial plexus sheath provides superior analgesia to that of continuous IV systemic injection. In another study, Wajima et al<sub>4</sub> found that butorphanol 2mg with 0.5% mepivacaine provide sufficient postoperative analgesia after upper limb surgery. Opioids have been used with or without local anesthetics for epidural anesthesia and analgesia<sub>10, 11</sub>. Veil et al<sub>12</sub> showed that injection of buprinorphine into the brachial plexus sheath using supraclavicular technique is an efficient way to control postoperative pain after upper limb surgery.

We have found that the addition of butorphanol 2mg to 40mL lidocaine 1% prolongs the duration of axillary brachial plexus blockade. There is, of course, so much resemblance among butorphanol, buprenorphine and tramadol. Primary afferent tissues (dorsal roots) have been found to contain opioid binding sites<sub>13</sub>. Young et al<sub>14</sub> demonstrated that opioid receptors and various macromolecules in the nerve undergo axonal flow. Laiden<sub>15</sub> later showed that opioid binding proteins undergo

bidirectional axonal transport. Young et al speculated that these moving receptors circulate endorphins, their endogenous ligands, in addition to exogenous opioids. The existence of such receptors underlies the hypothesis that opioids act directly on peripheral nervous system.

We conclude that addition of butorphanol 2mg to 40mL lidocaine 1% prolongs the duration of axillary brachial plexus blockade. Furthermore, butorphanol can be used as an alternative to clonidine, tramadol or dexamethasone to increase the duration of such blockade.

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