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Original Article

# Effects of adding dexmedetomidine to lidocaine on the onset and duration of axillary block for upper extremity surgeries

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## Article Info

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## Abstract

**Introduction:** Dexmedetomidine, which is an alpha 2 agonist, reduces the transmission of pain signals and has an independent inhibitory effect on nerve action potential. The purpose of this study was to examine the effects of adding dexmedetomidine to lidocaine in an axillary block.

**Methods:** In this randomized, double-blind study 40 patients included patients were divided randomly into two groups of 20: In the first group, 39 cc of 1% lidocaine plus 1cc of normal saline was administered and the 2nd group received dexmedetomidine 1cc (100µg) in addition to 39 cc of 1% lidocaine. The onset and persistence of the sensorimotor block and hemodynamic changes including heart rate and systolic and diastolic blood pressure before, during, and after surgery were compared.

**Results:** Age, sex, type of surgery, duration of surgery, and other demographic characteristics were not significantly different in two groups (P>0.05). Onset of the sensory and motor block was similar in both groups, but the persistence of the sensory and motor block and analgesia in the treatment group was significantly higher (P<0.05). The VAS score was lower in cases than controls. Hemodynamic change differences between the two groups were statistically significant (P<0.05).

**Conclusion:** The results of this study showed that adding dexmedetomidine to lidocaine in an axillary block did not alter the onset of the sensory and motor block, but the sensory and motor block duration and analgesia was increased. Despite significant differences in hemodynamic responses between the two groups, these changes were not clinically significant in ASA1 patients.

## Introduction

A regional nerve block is one of the important and effective ways of providing analgesia and anesthesia during surgery and providing post-operative pain control (1), which, while providing optimum conditions for surgery and faster mobility after surgery, has lower risks related to general anesthesia, fewer adverse effects, and also reduces hospital costs (2, 3).

An axillary plexus block is performed for hand and forearm anesthesia in most outpatient surgeries (2). The basis of this regional nerve block is injection of anesthetics near the nerve roots or the trunk (4). To improve the strength, quality, time, and duration of anesthesia in these blocks, other drugs such as opioids, bicarbonates, adrenalines, ketamine, and dexamethasone are used in combination with local anesthetics (5, 6).

Dexmedetomidine, like clonidine, is a α2 receptor agonist, which is more than 8 times specific for α2 receptors in comparison with clonidine (7). The mechanism of analgesia and sedation is not fully understood, but it seems that it works by reducing the secretion of norepinephrine in presynaptic α2 receptors, reducing the transmission of pain signals and by an independent inhibitory effect on the onset of action potentials of nerves (8, 9). Analgesia and sedation occurs in the central post synaptic α2 receptors by preventing the release of substance P in the dorsal root nociceptive direction and by enabling α2 receptors in the locus coeruleus, which results in decreased blood pressure and heart rate, cause sedation, analgesia, and has anxiolytic properties (8, 9). Today, the effects of systemic administration of dexmedetomidine on hemodynamic and sedation are known but there is little information about the effects of dexmedetomidine in combination with local anesthetics in regional blocks and limited research. Only a few studies have been done in this area over the last few years regarding the mechanism of action and properties of the drug, but it seems that dexmedetomidine can improve the length and quality of regional anesthesia such as an axillary block.

In this study, we designed a double-blind randomized clinical trial to evaluate the effects of adding dexmedetomidine to lidocaine for an axillary block.

## Materials and Methods

This randomized, double blind, clinical trial was performed on 40 patients scheduled for hand and forearm surgery. Inclusion criteria for the study included physical status 1 and 2 patients who were between 18 and 70 years of age. Patients with a history or symptoms suggestive of adrenal insufficiency, patients who received alpha-adrenergic receptor agonists or antagonists, were pregnant or lactating, had an infection at the block site, had any neuropathy or limited range of motion in the arm and shoulder, or had a history of drug abuse were excluded. After obtaining written informed consent, the patients were randomly divided into two groups of 20 each.

In the control group, 39 mL of 1% lidocaine plus 1 ml of normal saline and in the case group 39 mL of 1% lidocaine plus 1 ml (100 µg) of dexmedetomidine (Hospira, Inc., Lake Forest, IL USA) was injected.

After administration of 5 ml/kg normal saline and recording HR, SPO2, NIBP, and RR baselines, Patients were placed in the supine position. Fentanyl 2 µg/kg and midazolam 0.015 mg/kg of body weight was used for sedation. After cleaning and sterilization, the axillary block was performed with a nerve stimulator-guided 22-gauge needle 5 mm in length. The radial, ulnar, median, and musculocutaneus nerves were blocked separately.

The sensory block was assessed with the pinprick test and motor block of the radial, ulnar, median, and musculocutaneus nerve were evaluated with thumb abduction, thumb adduction, thumb opposition, and elbow flexion separately.

Sensory and motor block were assessed after local anesthetic injection every 3 minutes for half an hour and then every 30 minutes until the end of using the blocks. Onset of both sensory and motor block was measured. The duration of the sensory and motor blocks was determined separately from time of onset of complete block to the return of sensation and movement of limbs. Duration of analgesia was defined as the time of onset of sensory block to the time of onset of pain. Pain scores were measured with the visual analogue scale (VAS) in the recovery room. Patient's level of consciousness during surgery was measured with the Modified Observer Assessment of Alertness/Sedation (MOAA/S) scale (10) (Table 1).

**Table 1**. Modified Observer's Assessment of Alertness/Sedation Scale (MOAA/S)

| Responsiveness | Score |
|---|---|
| Agitated | 6 |
| Responds readily to name spoken in normal tone ("Alert") | 5 |
| Lethargic response to name spoken in normal tone | 4 |
| Responds only after name is called loudly and/or repeatedly | 3 |
| Responds only after mild prodding or shaking | 2 |
| Does not respond to mild prodding or shaking | 1 |
| Does not respond to deep stimulus | 0 |

Heart rate, systolic and diastolic arterial pressure, arterial oxygen saturation, and level of consciousness of the patient were monitored and recorded during the surgery (at baseline, 5 min after injection of sedatives, and 5, 10, 15, 30, 60, 90, and 120 minutes after the block).

Patients with a heart rate less than 50/min or more than 25% loss of baseline were treated with atropine 0.01 mg/kg and hypotension (a drop of more than 25% of baseline) was treated with normal saline and 5 mg of ephedrine.

All obtained data were analyzed using SPSS statistical software version 16. Quantitative data are shown as mean and standard deviation and qualitative data as frequency. The chi-square test and t-test were used for comparison between qualitative and quantitative data that were normally distributed and nonparametric tests were used in other cases. In this study, the level of significance was set at 0.05.

## Results

Table 2 shows the baseline characteristics of the patients in the two groups separately. The mean age in the control group was 27.3 ± 6.1 and in the case group, 30±10.1 years. The difference was not significant statistically (P=0.316).

The results of the study are shown in Table 3. The mean pain score in the control group was 2.65 and in the case group it was 0.55, which indicates that the case group had a less painful recovery with a significant difference (P=0.0001). The mean time of onset of the sensory block in the control group was 8.075 min and in the case group it was 8.675 min, a difference that was not statistically significant (P=0.408). The mean time of onset of complete motor block in the control group was 14.8 minutes and 19.4 minutes in the case group, a difference that was statistically significant (P=0.005), and the control group was quicker to achieve a complete motor block. The mean duration of the sensory block in the control group was 105.75 minutes and in the case group it was 369.5 minutes, a difference that was statistically significant (P<0.0001) and shows that the duration of sensory block in the case group had increased substantially. The mean duration of the complete motor block in the control group was 95.5 minutes and in the case group it was 322 minutes (P=0.0001), a difference that was statistically significant and shows that the duration of the motor block in the case group had increased considerably. The mean duration of analgesia in the case group was 523.25 minutes and in the control group it was 160. 25 minutes, a statistically significant difference (P<0.0001), which shows that the analgesia duration in the case group had increased substantially.

None of the patients in the case group required analgesia but in control group patients requested analgesia, although the difference was not significant (P=0.147). In both groups, significant complications were not observed and differences in

**Table 2**. Basic characteristics of the two groups

| | case group Lidocaine+Dexmedetomidine | control group Lidocaine + normal saline | P Value |
|---|---|---|---|
| Age | 30±(10.1) | 27.3 ± (6.1) | 0.316 |
| Male | 14(70%) | 18(90%) | 0.114 |
| Female | 6(30%) | 2(10%) | 0.114 |
| Height (cm) | 167±(8.29) | 172±(5.67) | 0.033 |
| Weight (kg) | 74.7±(10.8) | 75.65±(8.54) | 0.750 |
| HR (B/min) | 79±(16) | 80±(11) | 0.783 |
| Systolic BP (mmHg) | 130.15±(13.23) | 130.80±(11.39) | 0.869 |
| Diastolic BP (mmHg) | 82.30±(12.88) | 84.65±(8.34) | 0.498 |
| Surgery (soft tissue/bone) | 1/19 | 1/19 | 1 |
| Duration of surgery (min) | 104±(51.74) | 90.25±(21.67) | 0.280 |
| Tourniquet time (min) | 65.15±(20.01) | 64.75±(19.89) | |

minor complications such as mild hypotension between the two groups were not statistically significant (P=0.217). According to these findings, only 30 and 60 minutes after the block, a significant difference was observed between the two groups in terms of heart rate, which was lower in the case group (respectively P=0.01 and P=0.009). After repeated measurements and statistical analysis, it was observed that overall differences in heart rate between the two groups was significant and the mean heart rate was significantly lower in the case group (P=0.021). The mean systolic blood pressure at minutes 10, 15, 30, and 60 in the case group were also significantly different (P=0.003, P=0.003, P=0.0001, and P=0.002, respectively). Comparisons of diastolic blood pressure at 10, 15, 30, 60, and 90 minutes showed lower diastolic blood pressure in the case group (P=0.025, P=0.030, P=0.0001, P=0.002, and P=0.039, respectively).

According to the results obtained at minutes 10, 15, 30, 60, and 90, the group that received dexmedetomidine had a lower sedation level (P=0.004, P=0.0001, P=0.0001, P=0.0001, and P=0.0001, respectively). After repeated measures and statistical analysis, there was a significant difference between the two groups and significantly lower levels of sedation in the case group (P=0.0001) (Diagram 1).

In the case group, 2 cases had HR<50 that resolved spontaneously and did not require treatment with atropine. One patient in the case group became hypotensive, which was recovered with 5 mg ephedrine.

**Table 3.** Results of the study

| | Case group Lidocaine+Dexmedetomidine | Control group Lidocaine + normal saline | P Value |
|---|---|---|---|
| Pain score in recovery (VAS) | 0.55 | 2.65 | 0.0001 |
| Onset of sensory block (min) | 8.675±2.18 | 8.075±2.35 | 0.408 |
| Onset of complete motor block (min) | 19.14±3.51 | 14.8±5 | 0.005 |
| Duration of sensory block (min) | 369.5±78.3 | 105.75±27.44 | 0.0001 |
| Duration of complete motor block (min) | 322±40.85 | 95.5±16.05 | 0.0001 |
| Duration of analgesia (min) | 523.25±104.53 | 160.25±37.43 | 0.0001 |

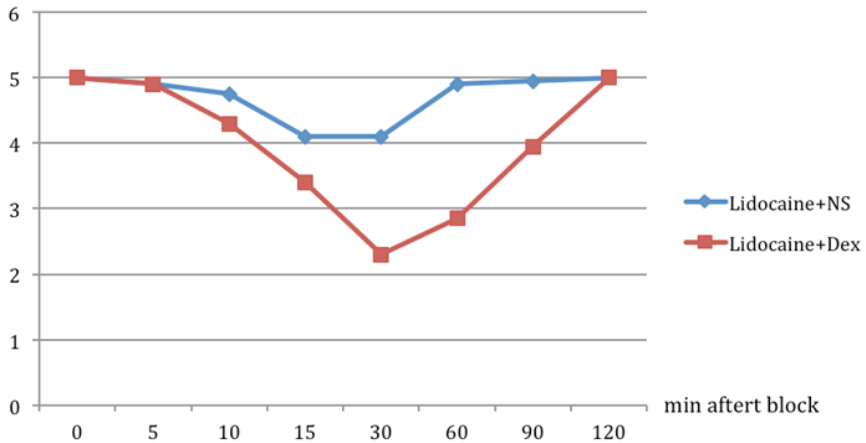


**Diagram 1**. Comparison of level of sedation in the two groups was measured with the Modified Observer Assessment of Alertness/Sedation (MOAA/S) scale

## Discussion

This study was conducted to evaluate the effects of adding dexmedetomidine to lidocaine for an axillary block. The results of this study show that the addition of lidocaine to dexmedetomidine can improve the duration of a sensory and motor block, analgesia, and the quality of motor block but is ineffective for the speed of onset of sensory and motor blockade. It can also reduce the VAS during recovery but does not reduce the need for narcotics. Increased complications in the study group were not statistically significant. Hemodynamic changes recorded in the case group showed a total reduction of

systolic, diastolic, heart rate, and sedation levels.

Duma (11) and colleagues in a study that was conducted in 2005 added clonidine to levobupivacaine and bupivacaine for an axillary block. It did not cause any difference in the onset of the sensory and motor block but increased the duration of the sensory block, which is consistent with the results of this study.

In the study of Iohom (12) and colleagues, clonidine was added to mepivacaine for an axillary block and the same improvement was observed in anesthesia and analgesia.

Memis (13) and colleagues added 0.05µg/kg dexmedetomidine to lidocaine for an intravenous block. In that study it was shown that the VAS scores in cases such as those included in the present study were lower than in the control group. Analgesic requirements, onset of sensory block, and motor block onset in the case group were significantly lower than in the control group. This may be due to differences in the type of surgery, in which all patients underwent surgical treatment of carpal tunnel syndrome.

In our study the duration of the sensory and motor block and analgesia significantly increased in the experimental group compared to the lidocaine group and also showed that the quality of the motor block and anesthesia in the dexmedetomidine group significantly improved.

Abosedira and colleagues who added clonidine and dexmedetomidine to an intravenous block in their study (14) and Brummett who added dexmedetomidine to bupivacaine (15) for a sciatic block came to the same conclusion.

Unlike the present study, in the study by Esmaoglu and colleagues (16) the onset of anesthesia and motor block was accelerated, but this difference could be due to the nature of the local anesthetic that was used. The duration of complete and partial sensory and motor block and analgesia in their study as in the present study was increased. In another study by the same researchers (17) in 2005 and in the study was conducted by Abosedira in which dexmedetomidine was added to lidocaine for an intravenous block (14), the duration of the complete and incomplete sensory and motor block was increased.

In the present study, the mean heart rate and the systolic and diastolic blood pressure in the dexmedetomidine group were significantly lower than in the lidocaine group, and these results are consistent with the findings of Esmaoglu.

According to these results, dexmedetomidine can be used to increase the duration of a sensory and motor block and improve the quality of anesthesia. Due to bradycardia and hypotension, dexmedetomidine is recommended in patients with a heart rhythm disorder and other heart problems when used cautiously or after adjusting the dose. Due to appropriate sedation and lack of respiratory failure with the use of dexmedetomidine, repeated doses of benzodiazepines and opioids can be avoided during surgery.

Designing new studies to compare different doses of dexmedetomidine in order to achieve the ideal dose is recommended.

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