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Research Article

Dexmedetomidine Added to Ropivacaine Prolongs Axillary Brachial Plexus Block

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Abstract

Background and Objectives: We evaluated the effect of adding dexmedetomidine to ropivacaine for axillary brachial plexus blockade. The primary endpoints were the onset and duration of sensory and motor block and duration of analgesia.

Methods: Eighty patients scheduled for elective forearm and hand surgery were divided into 2 equal groups in a randomized, double-blind fashion. The 4 main nerves in the axilla (musculocutaneus, radial, median, ulnar) were identified using neural stimulation. Patients were assigned randomly into 2 groups. In group R (n=40), 40ml (200 mg) of 0.5% ropivacaine +1ml saline and in group RD (n=40), 40ml (200 mg) of 0.5% ropivacaine +1ml dexmedetomidine (50µg) were given. Motor and sensory block onset times, block durations, and duration of analgesia were recorded.

Results: Demographic data and surgical characteristics were similar in both groups. Sensory and motor block onset times were shorter in group RD than in group R ($P<0.05$). Sensory and motor blockade durations were longer in group RD than in group R ($P<0.001$). Duration of analgesia was longer in group RD than in group R ($P<0.001$). Systolic arterial blood pressure levels in group RD at 10, 15, 30, 45, 60, 90, and 120 minutes were significantly lower than those in group R ($P<0.05$). Diastolic arterial blood pressure levels in group RD at 60, 90, and 120 minutes were significantly lower than those in group R ($P<0.05$). Heart rate levels in group RD, except basal measurements, were significantly lower than those in group R ($P<0.05$). In group RD, bradycardia was observed in 7 patients, although there was no bradycardia in group R ($P<0.05$).

Conclusions: Dexmedetomidine added to ropivacaine for axillary brachial plexus block shortens the onset time and prolongs the duration of the block and the duration of postoperative analgesia. However, dexmedetomidine also may lead to bradycardia.

Keywords: Dexmedetomidine, Ropivacaine, Brachial Plexus Block

1. Introduction

Ropivacaine is a local anesthetic with long duration of action, having similar pharmacology to bupivacaine; however, it has a wider safety margin and was shown to possess less cardiotoxicity in comparison with bupivacaine. The use of clonidine, a partial α_2 adrenoreceptor agonist, in peripheral nerve blocks, has been reported to be safe and beneficial and prolongs the duration of anesthesia and analgesia[1,2]. Dexemedetomidine is a α_2 receptor agonist, and its α_2/α_1 selectivity is 8 times more than clonidine. It has been reported to improve the quality of intrathecal and epidural anesthesia[3,4]. However, its use in axillary blocks has not been described. In this study, we investigated the effect of adding dexemedetomidine to ropivacaine for axillary brachial plexus blocks. Our primary endpoints were the onset time and duration of motor and

sensory blocks.

2. Methods

After ethical commitee approval and informed consent, 80 ASA physical status I–II patients scheduled for forearm and hand surgery under axillary block were enrolled in a prospective, double-blind controlled trial. Patients receiving adrenoreceptor agonist or antagonist therapy; those with a history of cardiac, respiratory, hepatic,or renal failure; and pregnant women were excluded. Patients were not premedicated before the block. After insertion of a 20-gauge IV cannula in the nonoperated arm, a 5 ml/kg/h infusion of 0.9% NaCl solution was started. After standard anesthesia monitoring, baseline measurements of heart rate (HR), noninvasive arterial blood pressure, peripheral oxygen saturation (Spo2), and respiratory rate were recorded before the block was performed. Axillary blockade was performed with the patient in the supine position with the upper arm abducted 90°. After preparation of the area, the axillary artery was palpated and a skin wheal was injected using 2ml of lidocaine 2%. Neural localization was achieved using a nerve stimulator (Stimuplex, Braun, Germany) connected to a 22-gauge, 50-mm-long stimulating needle (Biometer, Melsungen, Germany). Patients were randomly allocated using a sealed envelope technique to receive either 40ml of ropivacaine 0.5% with 1ml of isotonic sodium chloride solution (group R, n=40), or 40ml of ropivacaine 0.5% with 1ml (50 g) of dexmedetomidine (group RD, n=40) in a double-blind fashion. The drug solutions were prepared by an anesthesiologist not involved in the study. Ten milliliters of solution were injected after location of the median, ulnar, radial, and musculocutaneous nerves.

Sensory block (4 nerves) was assessed by pinprick test using a 3-point scale: 0 = normal sensation, 1 = loss of sensation of pinprick (analgesia), 2 = loss of sensation of touch (anesthesia). Motor block was evaluated by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion at the elbow (musculocutaneous nerve) on a 3-point scale for motor function (0 = normal motor function, 1 = reduced motor strength but able to move fingers, 2 = complete motor block).

Sensory and motor blocks were evaluated every 3 minutes until 30 minutes after injection, and then every 30 minutes after surgery, until they had resolved. Onset time was defined as the time interval between the end of total local anesthetic administration and complete sensory block. Complete sensory block was defined by anesthetic block (score 2) on all nerve territories. Duration of sensory block was defined as the time interval between the end of local anesthetic administration and the complete resolution of anesthesia on all nerves. Complete motor block was defined as absence of voluntary movement on hand and forearm (score 0). Duration of motor block was defined as the time interval between the end of local anesthetic administration and the recovery of complete motor function of the hand and forearm. HR, systolic arterial blood pressure (SAP), and diastolic arterial blood pressure (DAP) were recorded at 0 minutes, 5 minutes, 10 minutes, 15 minutes, 30 minutes, 45 minutes, 60 minutes, 90 minutes, and 120 minutes. Adverse events comprised hypotension (a 20% decrease in relation to the baseline value), bradycardia (HR <50 beats per minute [bpm]), hypoxemia (SpO_2<90%), or nausea and vomiting. Pain was assessed using the Visual Analog Scale (0–10). Nursing staff administered IM diclofenac 75mg when the Visual Analog Scale >4. The time between the end of local anesthetic administration and the first analgesic request was recorded as the duration of the analgesia.

Data were entered and analyzed with the Graph Pad.com (version 5, 2010). Statistical tests used for comparison is upaired t-test. Results are presented as mean (SD) and number (%) of cases as appropriate. The level of significance was set at $P < 0.05$, and 95% confidence intervals were calculated for the main outcome measures.

3. Results

The demographic data and surgical characteristics were similar in each group (Table 1). Sensory and motor block onset time were significantly shorter in group RD than in group R, and the difference was statistically significant (Table 2;$P < 0.05$).

Sensory and motor blockade durations were longer in group RD than in group L ($P< 0.001$). Duration of analgesia was significantly longer in group RD than in group R ($P< 0.001$) (Table 2). SAP levels in group RD at 10, 15, 30, 45, 60, 90, and 120 minutes were significantly lower than those in group R ($P < 0.05$). DAP levels in group RD at 60, 90, and 120 minutes were significantly lower than those in group R. HR levels in group RD, except basal measurements, were significantly lower than those in group R (Figure:1). In group RD, bradycardia was observed in 7 patients, and all of these patients were treated with atropine. There were no episodes of bradycardia in group R. No side effects including nausea, vomiting, hypotension, and hypoxemia were reported in either group.

Figure 1: Alterations of Mean Pulse Rate at different intervals between the groups

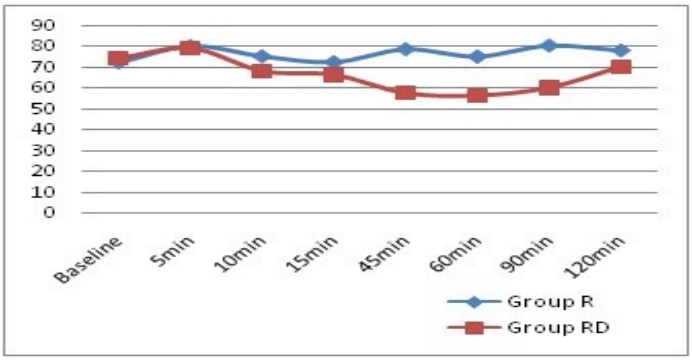

Table 1:Demographic data and surgical characteristics

	Group R	Group RD
Age(years)	36.96±12.59	36.06±11.02
Weight (kg)	71.43±12.35	72.20 ±9.01
Height (cm)	168.16 ± 6.41	170.66 ± 7.21
Gender (F/M)	10/30	8/32
Duration of tourniquet (minutes)	60.88 ±25.21	71.73 ±31.30
Duration of surgery (minutes)	61.70 ±30.02	74.13 ±39.74
Type of surgery (bone/soft tissue)	21/19	23/17
The results are expressed as Mean ±SD		

Table2: Sensory and Motor block onset time, block and analgesia durations in groups

Parameters	Group R	Group RD	P value
Onset time of sensory block (minutes)	13.12±2.30	11.31± 2.61	0.0003
Onset time of motor block (minutes)	15.61± 4.27	13.40±3.73	0.0157
Duration of sensory block (minutes)	412.61± 73.77	590.61±80.30	<0.0001
Duration of motor block (minutes)	350.71± 40.23	470.41 ±60.62	<0.0001
Duration of analgesia (minutes	600.14±90.82	760.69±120.12	<0.0001
The results are expressed as Mean ±SD (p-value<0.05, was considered statistically significant)			

4. Discussion

In this study, we demonstrated that in patients undergoing axillary brachial plexus block, dexmedetomidine, added to ropivacaine, shortens sensory and motor block onset time and extends block durations.

In brachial plexus block, though it has been demonstrated in some studies that clonidine added to local anesthetics extended the duration of anesthesia and increased the quality of analgesia[2]. Erlacher *et al* did not find an advantage in the quality and the duration of the block in their axillary block that was formed with the addition of 150 µg clonidine to 0.75% 40 mL ropivacaine[5]. The authors concluded that because ropivacaine itself had an intrinsic vasoconstrictor effect, adding α_2 adrenoreceptor did not increase this effect; that is, adding clonidine had no benefit. Clonidine may lead to side effects such as bradycardia, hypotension, and respiratory depression. In their axillary block studies with 0.150 mg clonidine added to 40 mL bupivacaine and 40 mL levobupivacaine, Duma *et al*[6] reported that there was no significant difference regarding sensory and motor block onset time. However, in this study it was reported that the block period was longer in the 2 groups with clonidine than in the 2 groups without. The mechanism by which α_2 adrenergic receptor agonists produce analgesia and sedation is not fully understood but is likely to be multifactorial. Peripherally, α_2 agonists produce analgesia by reducing release of norepinephrine and causing α_2 receptor-independent inhibitory effects on nerve fiber action potentials. Centrally,

α_2 agonists produce analgesia and sedation by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neuron and by activation of α_2 adrenoceptors in the locus coeruleus[7,8].

Several studies have found dexmedethomidine to be safe and effective in various neuraxial and regional anesthetics in humans, including intrathecal and IV regional anesthesia[3,9,10]. A dexmedetomidine–lidocaine mixture has been used to provide Bier's block and was shown to improve the quality of anesthesia and tourniquet pain and reduce postoperative analgesic requirement[9.10]. Another study compared the effect of adding either clonidine or dexmedetomidine to lidocaine during Bier's block and reported that adding dexmedetomidine to lidocaine during Bier's block is superior in quality of anesthesia, tourniquet tolerance, and intraoperative and early postoperative analgesia than is the addition of clonidine[11]. Brummet *et al*[12] reported that large-dose dexmedethomidine enhances the duration of bupivacaine anesthesia and analgesia of the sciatic nerve block in rats. In addition they histopathologically showed that the nerve axon and myelin were normal in both groups at 24 hours and at 14 days. Same authors in another experimental study reported that clinically relevant doses of dexmedetomidine enhanced blockade when added to ropivacaine[13]. Dexmedetomidine may lead to side effects such as hypotension and bradycardia with increased dosage, along with its effects such as sedation and anxiolysis[14]. In this study the incidence of bradycardia was high, despite the absence of hypotension in the RD group. Further studies are needed to determine the side effects and the safe optimal dose of dexmedetomidine.

In conclusion, 50µg of dexmedetomidine added to ropivacaine for brachial plexus block shortens sensory and motor block onset time, extends motor and sensory block durations, and also extends the analgesia period. However, dexmedetomidine may also lead to bradycardia.

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