INTRODUCTION
Brachial plexus block provides both intraoperative anaesthesia and postoperative analgesia without any systemic side-effects [1]. Ropivacaine has lower lipid solubility and have produced less central nervous system toxicity and cardiotoxicity than bupivacaine for which it is gaining popularity over peripheral nerve blocks [2]. There has always been a search for ideal adjuvant to local anaesthetics for regional nerve block that prolong the duration of sensory motor block. Several clinical studies have shown that clonidine prolongs sensory motor blockade when used with bupivacaine but effect of clonidine on ropivacaine is not well defined. The aim of the study was to evaluate the effect of clonidine on ropivacaine, for supraclavicular brachial plexus blockade. In a prospective randomised double blind placebo controlled study supraclavicular brachial plexus blockade was performed in 80 patients using 35 ml ropivacaine (0.5 %). Group A (n=40) had 150 μg clonidine and in Group B (n=40) 1ml normal saline added to ropivacaine. Sensory and motor blockade was assessed every 5 min till 30 min and at 15 min interval thereafter. Mean sensory onset time in group A was 10.44 ± 5.7 min and in group B was 15.85 ± 6.55 min, which was statistically significant. Patients of group A had a mean motor onset time 14.35 ± 7.8 min and patients of group B had a mean motor onset time 18.55 ± 7.64 min, the difference being statistically significant. Mean duration of sensory block in group A was 484.15 ± 63.4 min and in group B was 390.85 ± 72.65 min, which was statistically significant. Patients in group A had a mean duration of motor block 550 ±60.3 min and patients in group B had a mean duration of motor block 430.45±68.7 min, which was statistically significant. In conclusion, the addition of clonidine to ropivacaine, for brachial plexus blockade, increases the onset and duration of motor and sensory block.

METHODOLOGY
Following approval from the local hospital ethics committee, we conducted this study in the Department of anaesthesiology and critical care, SCB Medical College, Hospital from October 2012 to October 2013. Eighty patients using 35 ml ropivacaine (0.5 %). Group A(n=40)- ropivacaine 0.5% (35 ML) with 150 μg clonidine(1ml)

Group B (n=40)- ropivacaine 0.5%  with 1 ml normal saline

Identical syringes were loaded with 1 ml of either clonidine or normal saline and labelled by the anaesthesiologist, not involved in nerve block.
On arrival in the operation room, baseline heart rate, blood pressure and oxygen saturation were recorded and monitored throughout the procedure. An intravenous line was secured in the unaffected limb and Ringer’s lactate was started. All the patients received brachial plexus block through the supraclavicular approach by an experienced anaesthesiologist using nerve locator. Following negative aspiration, 35mL of a solution containing local anaesthetic combined with clonidine or normal saline was injected. A 3-min massage was performed to facilitate an even drug distribution.

Sensory block was assessed by the pin prick method. Assessment of sensory block was done at every 5 minute after completion of drug injection in the dermatomal areas corresponding to median nerve, radial nerve, ulnar nerve and musculocutaneous nerve till complete sensory blockade. Sensory onset was considered when there was a dull sensation to pin prick with a 23G needle along the distribution of any of the above-mentioned nerves. Complete sensory block was considered when there was complete loss of sensation to pin prick.

Sensory block was graded as-[6]
- Grade 0: Sharp pin felt
- Grade 1: Analgesia, dull sensation felt
- Grade 2: Anaesthesia, no sensation felt.

Assessment of motor block was carried out by the same observer at each 5 minute till complete motor blockade after drug injection. Onset of motor blockade was considered when there was Grade 1 motor blockade. Peak motor block was considered when there was Grade 2 motor blockade. Motor block was determined according to a modified Bromage scale for upper extremities on a 3-point scale [7]:
- 0 − normal motor function with full extension and flexion of elbow, wrist, and fingers,
- 1 − decreased motor strength, with ability to move only fingers,
- 2 − complete motor block with inability to move elbow, wrist, and fingers

The block was considered incomplete when any of the segments supplied by median, radial, ulnar and musculocutaneous nerve did not have analgesia even after 30 min of drug injection. These patients were supplemented with intravenous fentanyl (1 μg/ kg) and midazolam (0.02 mg/kg). When more than one nerve remained unaffected, it was considered a failed block. In this case, general anaesthesia was given. Patients were monitored for haemodynamic variables such as heart rate, blood pressure and oxygen saturation every 15 min after the block intraoperatively and every 60 min post-operatively. Sedation of patient was assessed by the Ramsay Sedation Score [8].

Assessment of blood loss was done and fluid was administered as per the loss. The intra- and post-operative assessment was done by an anaesthesiologist who was unaware of the drug used. Patients were assessed for duration of analgesia as per visual analogue scale of 0 to 10. The visual analogue scale was recorded post-operatively every 30 min till the score of 5. The rescue analgesia was given in the form of inj. IV paracetamol at the visual analogue scale of 5 and the time of administration was noted. All patients were observed for any side-effects like sedation, nausea, vomiting, dryness of mouth and complications like pneumothorax, haematoma, local anaesthetic toxicity.

The duration of sensory block was defined as the time interval between the end of local anaesthetic administration and the complete resolution of anaesthesia on all nerves. The duration of motor block was defined as the time interval between the end of local anaesthetic administration and the recovery of complete motor function of the hand and forearm.

**Statistical analysis**

Results are expressed as mean ± standard deviation. Chi-square test was applied for demographic data, haemodynamic parameters. Onset and duration of sensory and motor blockade and duration of analgesia was analysed by unpaired t-test. Statistical analysis was performed by SPSS (VERSION 16). P-value was considered significant if <0.05 and highly significant if <0.001.

**RESULTS**

There was no statistically significant difference between the demographics (age, sex, height, weight, ASA grade, duration of surgery). Also there was no statistical significance in baseline parameters like heart rate, systolic blood pressure, diastolic blood pressure, mean atrial pressure and oxygen saturation (Table I). Eighty patients were studied. Of these two patients were excluded from each group for incomplete / failed block to whom general anesthesia was administered.

Parametric data expressed as mean±standard deviation, categorical data (sex) expressed as frequency (%) using Chi-square analysis. P<0.05: Statistically significant. ASA= American society of anaesthesiologists, BPM=Beats per minute, SBP=Systolic blood pressure, DBP=diastolic blood pressure, MAP=mean arterial pressure, SPO₂= oxygen saturation.

No statistical difference was noted in the hemodynamic parameters (mean HR, MAP, SPO₂) before and after giving the block, throughout the surgery and postoperatively (fig. 1,2).
Table 1: Distribution of subject according to demographic profile and vital signs

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>30.6±10.2</td>
<td>32.5±9.6</td>
<td>0.3936</td>
</tr>
<tr>
<td>Sex ratio (M/F)</td>
<td>32/8</td>
<td>30/10</td>
<td>0.7895</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>60.4±11.42</td>
<td>61.2±10.64</td>
<td>0.7467</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>162±12.64</td>
<td>164.3±11.68</td>
<td>0.4006</td>
</tr>
<tr>
<td>ASA Grade (I/II)</td>
<td>30/10</td>
<td>32/8</td>
<td>0.7895</td>
</tr>
<tr>
<td>Duration of Surgery</td>
<td>92.6±28.4</td>
<td>91.8±29.6</td>
<td>0.9022</td>
</tr>
<tr>
<td>Base line heart rate (BPM)</td>
<td>79.2±14.4</td>
<td>80.4±13.8</td>
<td>0.7046</td>
</tr>
<tr>
<td>Base line SBP (mm of Hg)</td>
<td>126.0±8.9</td>
<td>124.4±9.1</td>
<td>0.4290</td>
</tr>
<tr>
<td>Base line DBP (mm of Hg)</td>
<td>78.3±8.4</td>
<td>77.6±7.6</td>
<td>0.6970</td>
</tr>
<tr>
<td>Base line MAP (mm of Hg)</td>
<td>91.6±7.8</td>
<td>92.1±8.2</td>
<td>0.7807</td>
</tr>
<tr>
<td>Base line SPO2 (%)</td>
<td>97.2±1.4</td>
<td>97.6±1.2</td>
<td>0.1740</td>
</tr>
</tbody>
</table>

Fig. 1: Comparison of intraoperative vitals (heart rate, mean arterial pressure, and oxygen saturation) in between Group A and Group B

Fig. 2: Comparison of postoperative vitals (heart rate, mean arterial pressure, and oxygen saturation) in between Group A and Group B

Table 2: Characteristics of sensory and motor block in group A and group B

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of sensory block (min)</td>
<td>10.44±5.7</td>
<td>15.85±6.55</td>
<td>0.0002</td>
</tr>
<tr>
<td>Onset of motor block (min)</td>
<td>14.35±7.8</td>
<td>18.55±7.64</td>
<td>0.0173</td>
</tr>
<tr>
<td>Duration of sensory block (min)</td>
<td>484.15±63.4</td>
<td>390.85±72.65</td>
<td>0.0001</td>
</tr>
<tr>
<td>Duration of motor block (min)</td>
<td>550±60.3</td>
<td>430.45±68.7</td>
<td>0.0001</td>
</tr>
<tr>
<td>Duration of analgesia (min)</td>
<td>586±56.8</td>
<td>465.8±62.5</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Data expressed as mean ± standard deviation. P<0.05= statistically significant
The onset of block was earlier in group A patients, that was 10.44 ± 5.7 min for sensory block and 14.35 ± 7.8 min for motor block than those in group B 15.85 ± 6.55 min for sensory block and 18.55 ± 7.64 min for motor block, which was statistically significant ($P < 0.05$)(table 2)(fig. 3).

Mean duration of sensory block in group A was $484.15 \pm 63.4$ min and in group B was $390.85 \pm 72.65$ min. Patients belonging to group A had a mean duration of motor block $550 \pm 60.3$ min and group B had a mean duration of motor block $430.45 \pm 68.7$ min, which was statistically significant (table 2) (fig. 4). Postoperative analgesia lasted $586 \pm 56.8$ min in group A and $465.8 \pm 62.5$ min in group B which was also statistically significant ($P < 0.05$).

Sedation score of patients in group A (42.5%) were higher than those in group B (15%) intraoperatively and postoperatively and this was statistically significant ($P < 0.05$).

DISCUSSION
The analgesic properties of clonidine when administered intrathecally or epidurally as adjuvant have been well demonstrated [9]. They seem to be attributable to its $\alpha_2$-agonist properties. Its action on large no $\alpha_2$ receptors present in the central nervous system, at locus coeruleus and dorsal horn of the spinal cord, is the main mechanism of centrally mediated sedation and analgesia [10]. Specific peripheral effects of clonidine appear less obvious because $\alpha_2$-adrenoreceptors are not present on the axon of the normal peripheral nerve [10]. It has been postulated that clonidine improved the duration of postoperative analgesia only when used as an adjuvant to intermediate-acting local anaesthetics and that it was not worthwhile to combine it with long-acting local anaesthetics.

Two reviews have addressed this issue [11, 12]. Murphy et al. [11] analyzed randomized trials that investigated the usefulness of a variety of adjuvants, including clonidine added to local anaesthetics for brachial plexus block. On the basis of data from six trials (349 patients), they concluded that clonidine in doses up to 150 $\mu$g increased the duration of postoperative analgesia with minimal adverse effects. McCartney et al. [12] reviewed 27 studies (1,385 patients) using clonidine as an adjuvant to local anaesthetics for a variety of peripheral nerve blocks. They concluded that clonidine was beneficial only when added to intermediate-acting local anaesthetics.

In this randomized, double-blinded trial, we compared clonidine as an adjuvant to ropivacaine in suprACLavicular brachial plexus block, and found that there was significant decrease in onset and increase in duration of sensory and motor blockade in the clonidine...
In the present study, group A (clonidine) mean onset time of sensory block was 10.44 ± 5.70 minutes and in group B (saline) it is 15.85 ± 6.55 min. Mean onset of motor block was 14.35 ± 7.8 minutes in group A (clonidine) and 18.55 ± 7.64 min in group B (saline). (Table 2). Mean duration of sensory block and motor block in patients of group A (clonidine) was 484.15 ± 63.40 minutes and 550 ± 60.3 minutes respectively (Table 2). Mean duration of sensory block and motor block in patients of group B (saline) was 390.85 ± 72.65 minutes and 430.45 ± 68.7 minutes respectively. The difference in both the groups is statistically significant. Postoperative analgesia lasted 586 ± 56.8 min in group A and 465.8 ± 62.5 min group B which was also statistically significant (P < 0.05). The results of present study in regards to onset of sensory block and motor block are in agreement with study by Antonucci S and its duration of sensory motor blockade in group A are in agreement with study of El Saied et al. [14].

Antonucci S [13] evaluated effects of tramadol used as adjuvant in brachial plexus block and compared with clonidine and sufentanil. He used ropivacaine for block and concluded that tramadol as adjuvant provides a significant reduction of onset time of sensory motor block and also provides a prolongation of anesthesia and analgesia with a quality of block similar that obtained with clonidine and sufentanil.

El Saied et al. [14] conducted a study in which axillary brachial plexus blockade was performed in 50 patients using 40 ml ropivacaine 0.75%. Group (A) had 150 μg clonidine and Group (B) 1 ml normal saline added to the local anesthetic. There was no difference in onset of sensory motor blockade. They concluded that the addition of 150 μg of clonidine to ropivacaine, for brachial plexus blockade, prolongs motor and sensory block and analgesia, without an increased incidence of side effects.

Giovanni Cucchiaro et al. [15], evaluated the effects of clonidine on the duration of sensory and motor block and analgesia time in 215 children who underwent a variety of peripheral nerve block including brachial plexus block and concluded that the addition of clonidine to bupivacaine and ropivacaine can extend sensory block by a few hours, and increase the duration of motor blocks.

Casati et al. [16] added low dose clonidine to ropivacaine (0.75%) in sciatic-femoral nerve block for foot surgery. They found that addition of 1 μg /kg clonidine to 0.75% ropivacaine prolongs the duration of postoperative analgesia by 3 h, with only a slight and short-lived increase in the degree of sedation and no hemodynamic adverse effects.

Casati et al. [17] has done a prospective, randomized, double-blind study in which he evaluated the effects of adding 1 μg/kg clonidine to 20 ml of ropivacaine 0.75% for axillary brachial plexus anesthesia. They concluded that adding 1 μg/kg clonidine to 20 ml of ropivacaine 0.75% for axillary brachial plexus anesthesia provided a 3 hour delay in first analgesic request postoperatively, without clinically relevant effects on the degree of sedation and cardiovascular homeostasis.

Danelli G et al. [18] evaluated the effects of adding 50 μg clonidine to 150 mg ropivacaine for superficial cervical plexus block in patients undergoing elective carotid endarterectomy. They opined that adding 50 μg clonidine to 150 mg ropivacaine for superficial cervical plexus block shortened the onset time and improved the quality of surgical anesthesia.

Mehdi Trifa et al. [19] added clonidine to ropivacaine in axillary plexus block in 60 patients of 1-6 yr age group. They concluded that ropivacaine (0.2%) 0.4 ml/kg for axillary plexus block provides sufficient postoperative analgesia in children scheduled for forearm or hand surgery. The addition of clonidine increased the time to first analgesia request.

Marhofer D et al. [20] evaluated dexmedetomidine as an adjuvant to ropivacaine in peripheral nerve block. Profound prolongation of ulnar nerve block was detected with perineural dexmedetomidine when added to 0.75% ropivacaine.

Brummett CM et al. [21] found that dexmedetomidine when added to ropivacaine in peripheral nerve block caused approximately a 75% increase in the duration of analgesia.

The above studies show that selective α2-adrenoceptor agonist like clonidine or dexmedetomidine when added as adjuvant to ropivacaine in different peripheral nerve blocks potentiates the sensory motor blockade. The mechanism is not clear. Probably peripherally, α2-agonists produce analgesia by reducing release of norepinephrine and causing α2-receptor-independent inhibitory effects on nerve fibre action potentials. Centrally, α2-agonists cause analgesia and sedation by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neurone and by activation of α2-adrenoceptors in locus coeruleus [5]. So the action of clonidine would then more likely be via a synergistic mechanism of action in combination with the local anesthetic resulting in the prolonged effect.

There are few studies which did not found any impact of clonidine when added to ropivacaine in peripheral nerve blocks.

Erlacher W et al. [22] in 2000 studied the efficacy of clonidine on ropivacaine in axillary plexus block. They found that there was no significant impact.
on onset/ duration of sensory and motor blockade in clonidine group

Erlacher W et al. [23] in 2001 evaluated clonidine as adjuvant for mepivacaine, ropivacaine and bupivacaine in axillary plexus block and opined that clonidine prolonged both sensory and motor blockade of mepivacaine and bupivacaine but not ropivacaine.

Jaiswal et al. [24] evaluated the effect of adding clonidine (150 μg) to ropivacaine (0.5%) for axillary plexus blockade among 60 patients and found that addition of clonidine (150 microgram) is of no benefit in the onset and duration of both sensory and motor block.

The limitation of present study was small sample size and ropivacaine or clonidine was not used as per body weight in kg.

CONCLUSION
In conclusion, clonidine added to ropivacaine is an attractive option for improving the quality and duration of supraclavicular brachial plexus block in upper limb surgeries. Clonidine in supraclavicular brachial plexus block not only hastens the onset, but also prolongs the duration of sensorimotor blockade, and provides a longer pain-free period without significant hemodynamic alterations. As it has more sedative potential, it can reduce patient anxiety and provide optimal intraoperative and postoperative patient comfort.

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