Introduction

Brachial plexus blocks have been successfully used for upper limb surgeries. Lignocaine and bupivacaine being the most commonly used local anaesthetics. Various adjuvants like opioids, midazolam, neostigmine have been used to improve the onset, quality and duration of block.

Of late, α2 agonists like clonidine and dexmedetomidine have been used as adjuvants with local anaesthetics. They had been used as antihypertensive agents initially but subsequently found to have sedative and anesthetic properties. They are also known to have antinociceptive action and enhance the effect of local anaesthetics when given intrathecally, epidurally and in peripheral nerve blocks. So, we decided to compare the onset time, duration and analgesic efficacy of clonidine with dexmedetomidine when added as adjuvant to bupivacaine (0.25%) for brachial plexus block by supraclavicular approach.

Materials and methods:

After obtaining institutional ethical committee approval, 50 patients belonging to age group 20 - 55years of ASA PS I & II to undergo forearm surgeries under ultrasound guided supraclavicular brachial plexus block in a tertiary care hospital were included in the study. Patient refusal, coagulation abnormalities, known hypersensitivity to clonidine or dexmedetomidine and infection at local site were excluded from the study.

The patients were randomized by computer generated randomization table into two groups.

Group BD – received bupivacaine 0.25% (19 ml) + Dexmedetomidine 1 μg/kg to make 20 ml Group BC – received bupivacaine 0.25% (19 ml) + Clonidine 1 μg/kg to make 20 ml Pre-anaesthetic evaluation was done on the day before the procedure included history, general physical examination and routine investigations. The study protocol was explained to the patients and a written informed consent obtained. Patient were kept NPO for 8 hours. On arrival in the operation theatre Intravenous access was obtained with 20G IV cannula, standard monitors connected and baseline HR, BP and SpO2 recorded Study drugs were prepared by an Assistant professor who was not involved in this study. Administering block and monitoring was done by the principal investigator who did not know about the preparation.

The patient was positioned supine with the head turned slightly to the opposite side. After aseptic preparation of the supraclavicular fossa, under ultrasound guidance (Sonoray ultrasound machine) with high frequency (10MHz) linear probe the brachial plexus and the adjacent anatomical structures (subclavian artery, cervical pleura, and first rib) were identified. The bunch of grape appearance on Ultrasound was noted and then the study drug combination was given after negative aspiration using a 22 G, 6cm needle. A total of 20 ml of solution containing study drug was injected to get a classical doughnut appearance on USG.

Sensory blockade was tested using pin prick method along the distribution of median nerve, radial nerve, ulnar nerve and musculocutaneous nerve. Sensory block was graded as Grade 0 = no sensation felt, Grade 1 = dull sensation felt, Grade 2 = sharp pain felt.

Duration of sensory block was defined as the time from the onset of sensory block to regaining of sensation completely in all the dermatomes.

Motor block was assessed using a modified Bromage scale 3 (3 = extension of elbow against gravity, 2 = flexion of wrist against gravity, 1 = finger movement, and 0 = no movement). Onset of motor blockade was considered when there was Grade2 motor blockade.

Duration of motor blockade was defined as the time interval between the administration of local anaesthetic and the return of complete motor function (grade 3). The duration of analgesia is noted from the time of onset of complete analgesia to the time at which the first rescue analgesic was required. After drug injection measurements of onset of sensory and motor blockade was carried out every 5 min for 30 minutes.

Vital parameters like HR, BP and SpO2 vital parameters were monitored. Patients in whom the block was unsuccessful or those who needed intravenous supplementation or general anaesthesia were excluded from the study. Any complications like sedation, nausea, vomiting, intravascular injection, pneumothorax and post-operative neuropathy was also noted. Postoperatively motor and sensory blockade and vitals of the patient was noted half hourly till the block completely wore off.

Statistical analysis was performed using SPSS 16.0. Quantitative data was analyzed using student’s unpaired ‘t’ test. Qualitative data was analyzed by Fisher’s chi square test. P value of <0.05 was considered statistically significant.

Results:
The demographic variables such as age, weight, ASA status and duration of surgery was comparable in both the groups. (P>0.05) (Table 1).

| Table 1: Demographic variables and duration of surgery |
|-----------------|-----------------|-----------------|-----------------|
|                | Group BC        | Group BD        | p value         |
| Age             | 36.8±12.26      | 34.88±9.03      | 0.12            |
| Weight          | 65.7±10.15      | 64.5±8.27       | 0.65            |
| ASA I/II        | 18/7            | 20/5            |                 |
| Duration of surgery (min) | 95.5±8.2      | 98.2±3.5        | 0.14            |

The mean time of onset of sensory and motor block was significantly faster in Group BD (8.8±0.91min. 11.36±0.952) than Group BC (11.14±1.13min. 13.65±1.11), (p <0.001). The mean duration of sensory block in group BC was 7.06±0.66hrs and in group BD was 9.3±0.667hrs. (p < 0.001). The mean duration of motor block in group BC was 6.66±0.657hrs and in group BD was 8.5±0.692hrs (p <0.001) (Fig 1). The mean duration of analgesia in group BC was 6.25 ± 0.96 compared to 7.86 ± 1.23 hours in groupBD (p <0.0001) (Table 2).

There were no significant side effects or complications in any of the patients.

| Table 2: Sensory and motor block characteristics |
|-----------------|-----------------|-----------------|-----------------|
|                | BC              | BD              | p value         |
| Onset of sensory block (min) | 11.14±1.13      | 8.8±0.91        | <0.001          |
| Onset of motor block (min)   | 13.62±1.11      | 11.36±0.952     | <0.001          |
| Sensory block duration (hr)  | 7.06±0.666      | 9.3±0.667       | <0.001          |
| Motor block duration (hr)    | 6.66±0.657      | 8.5±0.692       | <0.001          |
| Duration of analgesia        | 6.25±0.96       | 7.86±1.23       | <0.0001         |

DISCUSSION:
The α2 agonists have peripheral analgesic and anaesthetic actions that are independent of α2receptors. Both dexmedetomidine and clonidine have been successfully used in central neuraxial and peripheral nerve blocks with good results Kanazi et al;Brummett et al,Congedo et al and Esmaoglu A et al. We decided to compare the effects of 1 µg / kg of dexmedetomidine and clonidine as adjuvants to 0.25% bupivacaine in supraclavicular block.

We used a dose of 1 µg / kg of both dexmedetomidine and clonidine like others S Swami et al, Preeti More et al, Joby mathew et al. Since the equipotent doses of these drugs as adjuvants in brachial plexus blocks have not been documented.

In our study, it was found that the onset of sensory block and motor block were significantly faster in patients who received dexmedetomidine than clonidine. This is in conjunction with others. S Swami et al, Preeti More et al, Bajwa SJ et al. The α2 agonists causes faster sensory and motor onset by reducing norepinephrine release that causes inhibition of nerve fiber action potential. This effect is supposed to be not mediated through α2receptors.

Previous studies S Swamiṣet al Archana Tripathi et al, Preeti More et al Munshi et al have found the prolongation of sensory and motor block with dexmedetomidine when compared with clonidine. Our study also confirmed these findings. The prolongation of sensory and motor block is due to the reduction of the peak amplitude of compound action potential, the effect of which is maximum with as demonstrated by Kosugi et al.

Other studies have found that the dexmedetomidine group had longer duration of analgesia than clonidine group for brachial plexus block S Swamiṣ et al, Archana Tripathi et al, Preeti More et al Munshi et al. Similar results have been found in Epidural anaesthesia. Bajwa SJ et al. Our study also concurs with the above findings. The reasons have already been elucidated above.

All the patients in both the groups were adequately sedated though the dexmedetomidine group had slightly higher sedation than clonidine group. This may be due to the systemic absorption of the drug that causes sedation by their action on locus coeruleus. Other studies concur with the above findings S Swamiṣ et al, Preeti More et al.

Though there was a fall in Heart rate and Systolic blood pressure, none of the patients required treatment. These findings are in conjunction with other studies. S Swamiṣ et al, Preeti More et al.

Conclusion:
To conclude, we would like to state that dexmedetomidine shortens the time of onset and prolongs the duration of sensory and motor block as compared with clonidine when used as an adjuvant to Bupivacaine in supraclavicular block.

References: