IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 16, Issue 11 Ver. XI (Nov. 2017), PP 28-35
www.iosrjournals.org

A Comparative Evaluation of Clonidine, Dexmedetomidine And A Combination of Both As Adjuvants For Ropivacaine In Supraclavicular Brachial Plexus Block

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Abstract

Background: *Brachial plexus block is a versatile and reliable regional anaesthesia technique with multiple applications. It is among the most commonly studied and performed peripheral nerve blocks, owing to its high success rate and ability to provide prolonged intraoperative and postoperative analgesia. One of the newer long acting amide local anaesthetic, Ropivacaine is the stereoisomer of Bupivacaine,has been shown in earlier a studies to have less central nervous system toxicity and less cardiotoxicity than bupivacaine. In human studies, Ropivacaine has been shown to be less toxic than bupivacaine when injected intravenous. Dexmedetomidine, a highly selective alpha 2 adrenoreceptor agonist with sedative, sympatholytic and analgesic properties, is currently considered a super selective alpha-2 adrenergic agonist prototype.It has sedative, analgesic, sympatholytic and anxiolytic effects that blunt many of the cardiovascular responses in the perioperative period. Clonidine, an imidazole, with selective partial agonist activity at alpha 2 adrenergic receptors, has been used for many years as a centrally acting antihypertensive agent. Now clonidine is being used to prolong the duration of analgesia when used in combination with local anaesthetic agents or when injected into peripheral nerve sheaths.*

With the introduction of multimodality approach to pain management, wherein two or more drugs with different mechanisms of actions are used, newer adjuvants like clonidine have been used as adjunct to local anaesthetics in various regional techniques, to extend the duration of block.

Materials and Methods*: Eighty patients ASA Grade I and II aged 18-60 yrs undergoing upper limb surgery were randomly divided into four groups of 20 patients each. Following were the drug mixtures received by each group ;*

Group 1*:0.75% Ropivacaine (39ml) + Normal saline 1ml,* ***Group 2****:0.75% Ropivacaine (39ml) + Clonidine 1ml (150µgm),* ***Group 3****:0.75% Ropivacaine (39ml) + Dexmedetomidine 1ml (100µgm),****Group 4****:0.75% Ropivacaine (39ml) + Clonidine 0.5ml (75µgm) and Dexmedetomidine 0.5ml (50µgm).*

Supraclavicular block was given by KULENKAMPFF CLASSIC approach. Sensory block was assessed by pinprick method and compared with the same stimulation on contralateral arm.

0: Sharp pain., 1: Touch sensation only, 2: Not even touch sensation.

Sensory score of 2 was taken as the time of onset of sensory block.

Motor block was assessed by Bromage scale on the three point scale:

0: Normal motor function with full flexion and extension of elbow, wrist and fingers.

1: Decreased motor strength with ability to move fingers only.

2: Complete motor block with inability to move fingers.

Motor score of 2 was taken as the onset time of complete motor block.

Results: *It was observed that in Group III there was faster onset of sensory block compared to Group I, Group II and Group IV and was found to be statistically significant(p<0.001). Similarly we observed that in Group III there was faster onset of motor block compared to Group I, Group II and Group IV, which was found to be statistically significant(p<0.001). Duration of sensory block in Group III was again significantly prolonged as compared to Group I, Group II and Group IV (p<0.001). Duration of motor block in Group III was significantly prolonged as compared to Group I, Group II and Group IV (p<0.001).*

Conclusion: *The addition of clonidine and dexmedetomidine to ropivacaine is effective in supraclavicular brachial plexus block but dexmedetomidine was found to be the better in speeding up the onset time of sensory and motor block and also prolonging duration of sensory and motor block and postoperative analgesia compared to clonidine and plain ropivacaine group. Although the addition of reduced doses of both the*

adjuvants resulted in prolongation of sensory and motor blockade and postoperative analgesia but there was no additional synergitic benefit of adding both of them together.
Key words: *Brachial Plexus,Dexmeditomidine,Clonidine.*

Date of Submission: 09-11-2017 Date of acceptance: 30-11-2017

Introduction:

Brachial plexus block is a versatile and reliable regional anaesthesia technique with multiple applications. The supraclavicular approach has many advantages over other approaches to block brachial plexus. It is performed over trunk level where brachial plexus is present most compactly. The anatomic compactness is responsible for complete and reliable anaesthesia produced by supraclavicular block. Even small volume of anaesthetic solution results in a rapid onset of block because of the anatomic compactness.

Bupivacaine is the most commonly used amide local anaesthetic agent for intrathecal/epidural analgesia. Introduced in the market in 1963, it was followed by reports of central nervous system and cardiovascular system toxicity leading to restriction of its use and identification of a special treatment resistant cardiovascular toxicity. This lead to the development of pure agonists with less toxic potential such as ropivacaine in 1996 and levobupivacaine in 1999. One of the newer long acting amide local anaesthetic, ropivacaine is the stereoisomer of bupivacaine and has been shown in earlier animal studies to have less central nervous system toxicity and less cardiotoxicity than bupivacaine (Feldman HS et al 1989). In human volunteers, Ropivacaine has been shown to be less toxic than bupivacaine when injected intravenous by Scott D et al (1989). Furthermore its decreased propensity for motor block is useful for rapid patient mobilization in the postoperative period.

With the introduction of multimodality approach to pain management, wherein two or more drugs with different mechanisms of actions are used, newer adjuvants like clonidine have been used as adjunct to local anaesthetics in various regional techniques, to extend the duration of block.

Clonidine, an imidazole, with selective partial agonist activity at alpha 2 adrenergic receptors, has been used for many years as a centrally acting antihypertensive agent. Now clonidine is being used to prolong the duration of analgesia when used in combination with local anaesthetic agents, when injected into peripheral nerve sheaths (EL Saied et al, 2000)[1]. Dexmedetomidine, a highly selective alpha 2 adrenoreceptor agonist with sedative, sympatholytic and analgesic properties, is metabolized in liver to inactive metabolites. It is made up of medetomidines dextrogyrous enantiomer and is currently considered a super selective alpha-2 adrenergic agonist prototype.It has sedative, analgesic, sympatholytic and anxiolytic effects that blunt many of the cardiovascular responses in the perioperative period. It reduces the volatile anaesthetic, sedative and analgesic requirements of the patients without causing significant respiratory depression. Various studies have found dexmedetomidine to be safe and effective in various neuraxial blocks and regional anaesthesia and has been shown to improve the quality of anaesthesia and reduce postoperative analgesia requirement(Kanazi GE,Acta Anaesthesia Scandinavia,2006). Not much research has been done comparing the effectivness of ropivacaine 0.75% with alpha 2 agonists as adjuvants in supraclavicular block. It is in this context that the present study was undertaken to compare the relative effectiveness and safety of these commonly used adjuvants for ropivacaine in terms of potentiation and prolongation of analgesia in the intraoperative and postoperative period. This study was undertaken to assess the effect of clonidine, dexmedetomidine and a combination of both in reduced doses, as adjuvants for ropivacaine in supraclavicular brachial plexus block.

II. Aim & Objectives

The aim of the present study was to compare the efficacy and safety of clonidine, dexmedetomidine and a combination of both, in reduced doses, as adjuvants for ropivacaine in supraclavicular brachial plexus block.

III. Material & Methods

The study was conducted on 80 healthy individuals of ASA Grade 1 and Grade 2 in the age group of 18-60 years, of either sex, undergoing upper limb surgery.Patients with history of convulsions and neurological deficit, history of bleeding disorders and those receiving anticoagulants,patients with history of significant cardiac, respiratory, hepatic or renal diseases,pregnant females & patients having history of reaction to local anaesthetic agent were excluded from the study.

The patients taken up for study were randomly divided into 4 groups of 20 patients each.

Drug were prepared as follows:

Group 1:0.75% Ropivacaine (39ml) + Normal saline 1ml.

Group 2:0.75% Ropivacaine (39ml) + Clonidine 1ml (150µgm)

Group 3:0.75% Ropivacaine (39ml) + Dexmedetomidine 1ml (100µgm).

Group 4:0.75% Ropivacaine (39ml) + Clonidine 0.5ml (75µgm) and Dexmedetomidine 0.5ml (50µgm).

Anaesthetic Technique

Supraclavicular block be given by KULENKAMPFF CLASSIC approach.The patient was made to lie supine on OT table without a pillow, with the head turned towards the opposite side.The following landmarks were marked with the marker : (a). Midpoint of clavicle. (b).Point 1cm superior to the clavicular mid point.
Local anaesthesia was given with 1% lidocaine using 24G needle at the point marked above clavicle.Neural localization was achieved using a nerve stimulator connected to a 22-gauge, 50mm long stimulating needle.Sensory block was assessed by pinprick method and compared with the same stimulation on contra lateral arm.Sensory score of 2 was taken as the time of onset of sensory block. Motor block was assessed by Bromage scale on the three point scale:
0: Normal motor function, 1: Decreased motor strength with ability to move fingers only, 2: Complete motor block with inability to move fingers. Motor score of 2 was taken as the onset time of complete motor block.

Parameters

1) Onset of sensory block was considered as the time interval between drug administration and offset of paraesthesia and was noted by pinprick method every 2 minutes till a score 2 is achieved.
2) Onset of motor block was considered as the time interval between drug administration and motor paralysis distal to injection site and was noted by bromage scale every 2 minutes till a score of 2 is achieved.
3) Duration of sensory block defined as the time from the onset of the complete motor block to the full regresson of the block, (Bromage scale of 0) was noted postoperatively every 30minutes till the effect lasts.
4) Duration of motor block defined as the time from the onset of complete sensory block to restoration of sensation (by pinprick method score 0) was noted postoperatively every 30minutes till the effect lasts.
5) Haemodynamic variables : Baseline HR,SBP,DBP and MAP was recorded and every 10mins thereafter till the surgery lasts.
6) Mean duration of post operative analgesia.

IV. Observation And Results

There was no statistical difference between the two groups with respect to age, weight, gender and duration of surgery. Effect on onset of sensory block was compared between four groups and was subjected to statistical analysis. This was done by Bonferroni's 't' test(Posterior test after ANOVA). It was observed that, in Group III there was faster onset of sensory block compared to Group I, Group II and Group IV and was found to be statistically significant($p<0.001$) Comparisons between Group I, Group II and Group IV were found statistically insignificant that is, they were comparable to each other in onset of sensory block($p>0.05$)

Mean onset of Sensory Block(minutes)

Intergroup comparison and statistical I significance (Bonferroni's 't' test)

Group	Bonferroni's 't' Statistic	Inference
I-II	1.000	NS
I-III	0.001	S
I-IV	1.000	NS
II-III	0.001	S
II-IV	1.000	NS
III-IV	0.001	S

Effect on onset of motor block was compared between four groups and was subjected to statistical analysis. This was done by Bonferroni's 't' test (Posterior test after ANOVA). It was observed that, in Group III there was faster onset of motor block compared to Group I, Group II and Group IV. It was found to be statistically significant($p<0.001$). Comparisons between Group I, Group II and Group IV were found statistically insignificant that is, they were comparable to each other in onset of motor block($p>0.05$).

Mean onset of Motor Block(minutes)

Intergroup comparison and statistical I significance (Bonferroni's 't' test)

Group	Bonferroni's 't' Statistic	Inference
I-II	1.000	NS
I-III	0.001	S
I-IV	0.820	NS
II-III	0.001	S
II-IV	1.000	NS
III-IV	0.001	S

Mean Duration of Sensory Block

Intergroup comparison and statistical significance (Bonferroni's 't' test)

Group	Bonferroni's 't' Statistic	Inference
I-II	0.001	S
I-III	0.001	S
I-IV	0.001	S
II-III	0.001	S
II-IV	0.210	NS
III-IV	0.001	S

Mean duration of sensory block was recorded in each group in minutes and then subjected to statistical analysis for intergroup comparison using Bonferroni's 't'test(Posterior test after ANOVA) . Duration of sensory block in Group III was significantly prolonged as compared to Group I, Group II and Group IV ($p<0.001$). In Group II increased duration of sensory block as compared to Group I was found to be statistically significant($p<0.001$).In Group IV, it was observed that mean duration of sensory block was almost same as in Grouy II and there was no statistical significance($p>0.05$).

Mean Duration of Motor Block (minutes)

Group	Duration of Motor Block (minutes) Mean ± SD
I	308.9±69.46
II	579.5±50.06
III	896.6±48.79
IV	550.7±63.85
Significance	'f' value = 337.5 ; p<0.001 ; HS

In Group I, mean duration of motor block was 308.9±69.46 minutes.
In Group II, mean duration of motor block was 579.5±50.06 minutes.
In Group III, mean duration of motor block was 896.6±48.79 minutes.
In Group IV, mean duration of motor block was 550.7±63.85 minutes.
The above observations were found to be statistically significant by ANOVA test($p<0.001$).

Mean heart rate per minute at an interval of 10 minutes till 120 minutes.

IN MINUTES	GROUP I (MEAN ±SD)	GROUP II (MEAN ±SD)	GROUP III (MEAN ±SD)	GROUP IV (MEAN ±SD)	P value
0	78.5±4.25	79.0±5.22	79.6±4.42	77.6±4.44	0.544
10	78.6±4.11	79.9±5.13	79.9±5.12	78.3±3.95	0.571
20	79.6±3.84	79.7±6.03	79.2±4.27	77.7±4.67	0.527
30	80.2±5.16	78.2±5.55	77.1±4.42	78.3±3.01	0.228
40	78.5±3.79	79.1±4.79	79.1±4.79	78.6±2.98	0.949
50	78.7±3.75	79.2±5.11	77.4±4.90	78.6±4.98	0.670
60	78.6±3.90	79.1±4.69	77.3±4.68	78.3±3.76	0.592
70	78.7±3.33	78.8±5.00	79.1±3.86	79.4±4.22	0.948
80	79.2±4.16	79.0±4.77	78.6±5.62	79.0±4.77	0.984
90	79.3±4.62	79.9±4.78	78.7±4.56	78.7±3.92	0.826
100	79.3±4.20	79.8±4.44	78.6±5.35	78.4±4.39	0.751
110	78.7±4.54	80.0±4.76	80.1±4.76	79.2±3.38	0.711
120	79.4±4.69	78.7±3.78	78.4±4.15	78.2±2.88	0.784

The heart rate was recorded intraoperatively every 10 minutes upto 120 minutes, in each group.The findings so recorded were put to the statistical analysis and there was no statistically significant changes in mean heart rate of four groups($p>0.05$).

Mean map at an interval of 10 minutes till 120 minutes.

In Minutes	Group I (Mean ±Sd)	Group Ii (Mean ±Sd)	Group Iii (Mean ±Sd)	Group Iv (Mean ±Sd)	P Value
0	91.4±4.33	90.3±5.36	89.5±4.18	89.4±5.15	0.513
10	91.4±4.08	90.7±4.42	89.9±4.16	90.2±4.22	0.701
20	91.2±4.22	90.5±5.01	89.1±4.36	90.8±4.45	0.505
30	90.9±4.88	91.2±4.56	91.2±3.44	90.6±4.96	0.962
40	90.4±4.77	90.9±4.17	90.7±4.60	90.6±4.93	0.991
50	90.2±5.35	90.2±3.26	89.8±4.22	90.6±3.18	0.933
60	90.6±4.59	90.2±3.09	91.0±3.05	90.5±3.88	0.926
70	89.6±4.56	90.8±4.67	90.2±4.33	91.8±3.88	0.434

80	**90.1±5.07**	**90.5±3.51**	**90.3±4.01**	**90.5±3.97**	**0.988**
90	**90.7±5.06**	**89.9±3.17**	**90.9±2.96**	**89.8±3.28**	**0.725**
100	**90.5±3.91**	**89.9±3.17**	**91.6±3.45**	**90.3±4.49**	**0.713**
110	**89.9±4.76**	**90.7±3.89**	**91.9±3.25**	**90.6±3.96**	**0.503**
120	**89.6±4.70**	**90.6±5.37**	**91.9±3.95**	**90.6±5.19**	**0.531**

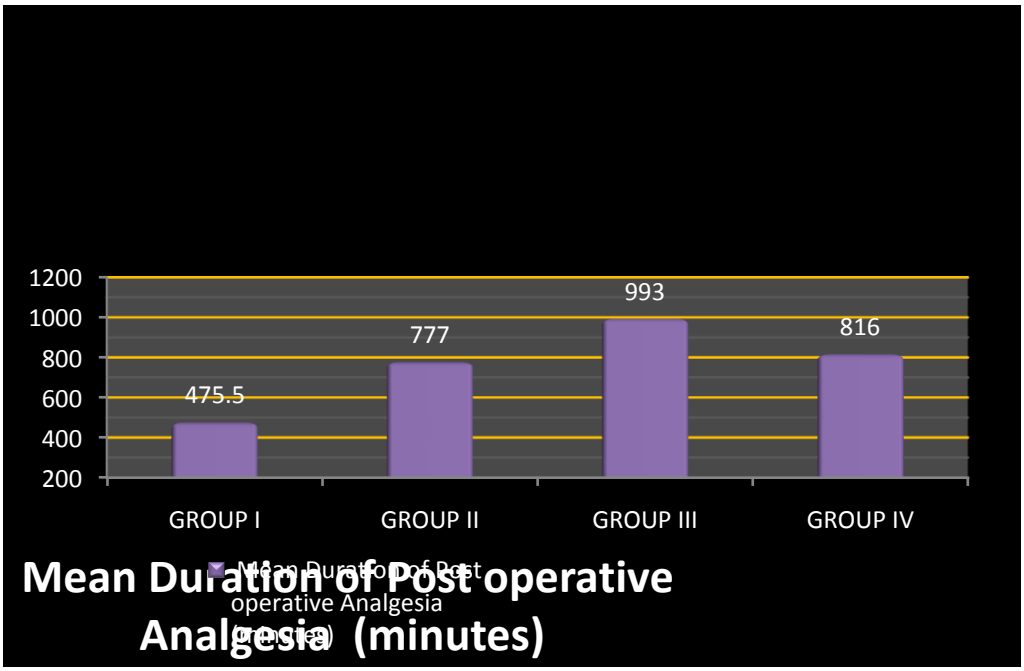
Mean arterial pressure was recorded intraoperatively every 10 minutes upto 120 minutes in each group.The findings so recorded were put to the statistical analysis and there was no statistically significant changes in mean arterial pressure of four groups(p>0.05).

Mean Duration Of Post Operative Analgesia (Minutes)

Mean Duration of Postoperative Analgesia Intergroup comparison and statistical I significance (Bonferroni's 't' test)

Group	Bonferroni's 't' Statistic	Inference
I-II	**0.001**	**S**
I-III	**0.001**	**S**
I-IV	**0.001**	**S**
II-III	**0.001**	**S**
II-IV	**0.185**	**NS**
III-IV	**0.001**	**S**

Mean duration of postoperative analgesia was recorded in each group in minutes and then subjected to statistical analysis for intergroup comparison using Bonferroni's 't'test(Posterior test after ANOVA) . Duration of postoperative analgesia in Group III was significantly prolonged as compared to Group I, Group II and Group IV (p<0.001). In Group II increased duration of postoperative analgesia as compared to Group I was found to be statistically significant(p<0.001). In Group IV, it was observed that mean duration of postoperative analgesia was almost same as in Group II and there was no statistical significance(p>0.05).



V. Discussion

Brachial plexus block results in anaesthesia that is limited to a restricted portion of body, not disturbing the metabolism of rest of the body, early ambulation and early discharge of the patient. The usual indications for supraclavicular plexus block are surgeries of hand and arm. It can also be used for shoulder surgery but may require supplementation of the supraclavicular nerve (C3-C4) to ensure anaesthesia of the cape of the shoulder.

Supraclavicular approach has many advantages over other approaches to block brachial plexus therefore, in present study brachial plexus block was given through supraclavicular approach.To improve the quality of block and prolong the duration of postoperative pain relief numerous adjuncts have been added to anaesthetic agent when used in brachial plexus block. **Murphy DB et al** in **2000**[2] added epinephrine, clonidine,

opioids, bicarbonate, tramadol, neostigmine , verapamil, butorphenol and studied their effects on quality and duration of brachial plexus block.

In our study we carried out a comparative evaluation of clonidine, dexmedetomidine and a combination of both as adjuvants for ropivacaine 0.75% in supraclavicular brachial plexus block using nerve stimulator for upper limb orthopaedic surgery.Our observations and results showed the following:

The mean onset of sensory block was **12.4 ± 1.90** in group I, **12.4 ± 1.90** in group II, **8.4 ± 2.79** in group III and **12.4 ± 3.15** in group IV. The difference was found to be statistically significant ($p<0.001$). It was observed that in group III that is , those patients in whom dexmedetomidine is used with 0.75% ropivacaine, there was faster onset of sensory block compared to group I, group II and group IV. This is in accordance with a study done by **Esmaoglu et al** in **2010**[3] in which he added dexmedetomidine to levobupivacaine in axillary brachial plexus block and showed that it shortens the onset time of sensory block. In group I, group II and group IV, no statistically significant difference was found in the onset time of sensory block which is in accordance with the study done by **El Saied AH et al** in **2000**[1] who also stated that no difference was noted by adding 150µg clonidine to 0.75% ropivacaine.

The mean onset of motor block was **16.6 ± 2.26** in group I, **17.5± 2.33** in group II, **12.4± 2.72** in group III and **17.8± 2.74** in group IV. The difference was found to be statistically significant ($p<0.001$). It was observed that in group III that is , those patients in whom dexmedetomidine is used with 0.75% ropivacaine, there was faster onset of motor block compared to group I, group II and group IV. This is in accordance with a study done by **Esmaoglu et al** in **2010**[3] in which there is faster motor onset after adding dexmedetomidine with levobupivacaine. While in other groups there was no statistically significant difference in the motor onset which is in accordance with a study done by **El Saied AH et al** in **2000**[1]**.**

The mean duration of sensory block in group I was **371.6±45.70,** in group II was **649.1±43.16,** in group III was **948.6±48.03** while in group IV was **614.6±63.81.** Thus the difference in these four groups was found to be statistically highly significant ($p<0.001$) with group II having a much longer duration of sensory block compared to group I and group III having longer duration than group II. The duration of sensory block in group II and group IV was found to be comparable. Our study is in comparison with the study done by **El Saied AH et al** in **2000**[1] who used clonidine with 0.75% ropivacaine and found the increased duration of sensory block.

Esmaoglu et al in **2010**[3] in their study also found increased duration of sensory block after adding dexmedetomidine to levobupivacaine, so the results of our study are in accordance with this study.

A study done by **Kaygusuz MD** in **2012** also found that addition of dexmedetomidine with 0.5% ropivacaine increases duration of sensory block which is in accordance with our study.

The mean duration of motor block in group I was **308.9±69.46**, in group II was **579.5±50.06**, in group III was **896.6±48.79** while in group IV was **550.7±63.85.** Thus the difference in these four groups was found to be statistically highly significant ($p<0.001$) with group II having a much longer duration of motor block compared to group I and group III having longer duration than group II. The duration of motor block in group II and group IV was found to be comparable. Our study is in comparison with the study done by **El Saied AH et al** in **2000**[1] who used clonidine with 0.75% ropivacaine and found the increased duration of motor block. **Esmaoglu et al** in **2010**[3] in their study also found increased duration of motor block after adding dexmedetomidine to levobupivacaine, so the results of our study are in accordance with this study. Another study done by **Singh S et al** in **2010**[4] also supported our findings suggesting that addition of 150µg clonidine to bupivacaine prolonged the duration of motor block. A study by **Kaygusuz MD** in **2012** also showed an increase in duration of motor block after adding dexmedetomidine with 0.5% ropivacaine which is in accordance with our study.

The heart rate was recorded every 10 minutes intraoperatively for 120 minutes in all the four groups. The difference in all the four groups was statistically insignificant ($p>0.05$). No statistically or clinically significant fall in heart rate was observed in all the measured intervals during the study. This was in accordance with the study done by **El Saied AH et al** in **2000**[1], where they added 150µg clonidine to 40ml of 0.75% ropivacaine in axillary brachial plexus block and another study done by **Duma et al** in **2005**[5] where they compared and evaluated the effect of adding 150µg clonidine to 40ml of 0.5% levobupivacaine and 40ml of 0.5% bupivacaine in axillary brachial plexus block while a study done by **Esmaoglu et al** in **2010**[3] showed that addition of dexmedetomidine with levobupivacaine in axillary brachial plexus block may lead to bradycardia which donot match the observations of the present study.

The mean systolic blood pressure in all the four groups recorded every 10 minutes for 120 minutes intraoperatively was found to be statistically insignificant ($p>0.05$). There was no statistically or clinically significant fall in systolic blood pressure at all the measured intervals during the study. This is in accordance with the studies done by **El Saied AH et al** in **2000**[1] and **Duma et al** in **2005**[5] who also claimed, to have no episode of significant hypotension being reported, during the entire study period. Another study conducted by **Yoon CS et** in **1996**[6] also showed no episode of hypotension during the study period, hence in accordance with our findings.

The mean diastolic blood pressure was recorded in all the four groups every 10 minutes for 120 minutes intraoperatively and the difference was found to be statistically insignificant ($p>0.05$). There was no statistically or clinically significant fall in diastolic blood pressure at all measured intervals during the study. This is in accordance with the studies done by **El Saied AH et al** in **2000**[1] and **Duma et al** in **2005**[6], both of which showed haemodynamic stability during the study period.

The duration of postoperative analgesia was measured till a visual analogue score of >3 was noted. The mean duration of analgesia in group I was **475.5±39.27**, in group II was **777.0±51.41**, in group III was **993.0±68.06** while in group IV was **816.0±61.25.** The difference in the groups was statistically highly significant ($p<0.001$) with group II having longer duration of analgesia than group I and group III having much longer duration of analgesia than group II. The duration of analgesia in group II and group IV was found to be statistically insignificant. This was in accordance with the study done by **El Saied et al** in **2000**[1] which showed an increase in analgesic duration in the clonidine group. Another study done by **Chakraborty S et al** in **2010**[7] also showed the prolongation of duration of analgesia when clonidine was added to bupivacaine. A study by **Esmaoglu et al** in **2010**[3] after adding dexmedetomidine with levobupivacaine also showed an increase in the duration of analgesia. **Kaygusuz MD** in **2012** also showed an increase in duration of analgesia after adding dexmedetomidine with 0.5% ropivacaine which is in accordance with our study.

Clonidine was initially used for its antihypertensive properties. The central actions are mediated through α_2 adrenoceptors, which are situated at locus coeruleus and dorsal horn of spinal cord. But, specific peripheral effects of clonidine appear to be less obvious because α_2 adrenoceptors are not present on the axon of the normal peripheral nerve. There have been four proposed mechanisms for the action of clonidine in peripheral nerve blocks. These mechanisms are centrally mediated analgesia, α_2 β adrenoceptor-mediated vasoconstrictive effects, attenuation of inflammatory response and direct action on peripheral nerve. The direct action of clonidine on the nerve can be explained on the basis of a study conducted by **Dalle et al** in **2001**[8]. They proposed that clonidine, by enhancing activity-dependent hyperpolarisation generated by the Na/K pump during repetitive stimulation, increases the threshold for initiating the action potential causing slowing or blockage of conduction. **Kosugi et al** in **2010**[9] examined the effects of various adrenoceptor agonists including dexmedetomidine, tetracaine, oxymetazoline and clonidine, and also an α_2 adrenoceptor antagonist (atipamezole) on compound action potential (CAP) recorded from frog sciatic nerve, and found that CAPs were inhibited by α_2 adrenoceptor agents so that they are able to block nerve conduction.

Popping et al in **2009**[10] in their metaanalysis of randomized trials showed that the beneficial effect of clonidine on the duration of analgesia was observed with all tested local anaesthetics. They observed that the prolongation of motor block was higher when clonidine was added to bupivacaine as compared with ropivacaine. The least effect was noted with prilocaine.

Erlacher et al in their study in **2000**[11] did not find an advantage in the quality and the duration of block in their axillary block that was formed with the addition of 150µg clonidine to 0.75% 40ml ropivacaine. The authors concluded that because ropivacaine itself had an intrinsic vasoconstrictor effect, adding α2 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.

VI. Conclusion

In present study, it was found that addition of clonidine and dexmedetomidine to ropivacaine is effective in supraclavicular brachial plexus block but dexmedetomidine was found to be the better in speeding up the onset time of sensory and motor block and also prolonging duration of sensory and motor block and postoperative analgesia compared to clonidine and plain ropivacaine group. Although the addition of reduced doses of both the adjuvants resulted in prolongation of sensory and motor blockade and postoperative analgesia but there was no additional synergitic benefit of adding both of them together.

Conflicts of intrest:
There are no conflicts of interest.

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*Naveed Iqbal. "A Comparative Evaluation of Clonidine, Dexmedetomidine And A Combination of Both As Adjuvants For Ropivacaine In Supraclavicular Brachial Plexus Block." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.11 (2017): 28-35