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A Study to Evaluate the Efficacy of Intravenous Dexmedetomidine in Brachial Plexus Block With Ropivacaine

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Abstract:
Background *We designed this prospective, double-blinded, controlled study to investigate the effects of intravenous dexmedetomidine, an alpha 2 agonist, on supraclavicular brachial plexus block (SBPB) with ropivacaine in ASA grade I and II patients undergoing upper extremity surgery.*
Methods *Nerve stimulator guided SBPB was performed in 60 patients undergoing upper extremity surgeries with ropivacaine 0.5% 30 mls. Patients in group R (numbers 30) received ropivacaine for SBPB followed by 10 mls of normal saline intravenously. Patients in group RD (numbers 30) received ropivacaine for SBPB followed by dexmedetomidine 0.5 mcg/ kg body weight. SBPB related sensory and motor scores were evaluated.*
Results *Onset of sensory and motor block was significantly faster in group RD when compared to group R [mean (SD)] [13.77 (0.94) vs 17.57 (1.10) minutes, P < 0.0001 and 15.05 (1.07) vs 20.03 (1.16) minutes, P < 0.0001 respectively]. Prolongation of sensory and motor block between group R and group RD was not statistically significant. Moreover, intravenous dexmedetomidine resulted in satisfactory intraoperative sedation without clinically significant hemodynamic complications.*
Conclusion *Administration of intravenous dexmedetomidine 0.5 mcg/ kg body weight bolus following SBPB with 0.5% ropivacaine 30 mls in ASA I and II patients having upper extremity surgeries resulted in faster onset of sensory and motor block as well as satisfactory intraoperative sedation without clinically significant hemodynamic complications when compared to the patients who had only SBPB without intravenous dexmedetomidine.*
Keywords: *brachial plexus block, dexmedetomidine, intravenous, ropivacaine*

I. Introduction

Supraclavicular Brachial plexus block (SBPB) is a common regional anesthetic technique and is used to provide anesthesia to the hand, forearm, and arm sparing the shoulder[1] for a wide range of orthopedic and reconstructive surgeries. Besides anesthesia, SBPB provides postoperative analgesia, and improved regional blood flow owing to sympathetic blockade without systemic side effects.[2] Ropivacaine, a S(-) enantiomer, is a long acting local anesthetic (LA). It is less lipophilic than bupivacaine and thus shows reduced potential for central nervous and cardiovascular toxicity[3] and a lesser degree of motor blockade than bupivacaine.[4]
Studies have confirmed that alpha2 adrenoceptor agonist clonidine and dexmedetomidine used as adjuvants to LAs enhanced the effects as well as duration of central and peripheral nerve blockade.[5,6] Dexmedetomidine has eight times higher alpha2/alpha1 selectivity than clonidine and thus a much more effective sedative and analgesic agent.[7] Dexmedetomidine has been used as an adjuvant to LA in ulnar nerve block,[8] axillary brachial plexus block[9] and epidural[10] as well as subarachnoid block.[11] Supplementation of regional anesthesia with intravenous dexmedetomidine has been shown to be effective in subarachnoid[12] and epidural[13] blocks. However, there is lack of data regarding the effects of systemically administered dexmedetomidine on SBPB except one study in patients with end-stage renal disease.[14]
We therefore designed this prospective, double-blinded, randomized controlled study to investigate the efficacy of intravenously administered dexmedetomidine on SBPB with ropivacaine.

II. Methods

The study was approved by the Ethics Committee of KPC Medical College Hospital, Kolkata, India. 2.1 Screening visit Sixty patients of age group between twenty to sixty years of either sex of ASA physical status I and II undergoing planned elective surgery for lower one third of humerus, olecranon, forearm and wrist of less than two hours thirty minutes duration were enrolled in the study. Before inclusion in the study, we

informed the patients about the nature, and the procedures of the study as well as the particular study-related risks.

Exclusion criteria were:

- Patient refusal;
- Anatomical deformity in the site of block;
- Hyper-sensitivity to ropivacaine or other amide local anesthetics as well as to dexmedetomidine;
- Infection at block site;
- Coagulopathy;
- BMI $\geq$ 30 kg m^{-2} ;
- History of neurological, psychiatric, or neuromuscular disease;
- Prolonged surgery more than three hours.

After signing the informed consent, each patient underwent a history taking and physical examination followed by routine blood test, fasting blood sugar, urea, creatinine, and coagulation profile. Other investigationse.g.12leadECG,chestXRayetc.weredoneas appropriate.

The screening visit took place within one week before the study day. Fasting instruction was 6 hours for solids and 2 hours for clear liquids.

2.2. Blinding

The principal study physician who was supposed to administer the block and inject either normal saline or dexmedetomidine intravenously waited outside the operation theatre (OT) while the second physician prepared the drug inside the OT. Three similar syringes of 10 mls each were used for drawing the LA. Another 10 mls syringe was loaded with either normal saline or dexmedetomidine and was kept aside for intravenous use. A third physician unaware of the intravenously administered injection (containing either normal saline or dexmedetomidine) performed the sensory and motor tests to confirm the block success and duration. The patients were unaware of the injected intravenous drug (normal saline or dexmedetomidine).

2.3. Monitoring

The patients received standard monitoring, e.g. noninvasive blood pressure (NIBP), oxygen saturation (SpO_2), and electrocardiography (ECG) (leads II and V4) before performance of the block until complete resolution of the SBPB. Bradycardia was defined when heart rate decreased > 20% from pre-injection values. Hypotension was defined when mean arterial pressure decreased > 20% from pre-injection values. Glycopyrrolate 0.01 mg kg^{-1} and phenylephrine 0.2 mg intravenous bolus were kept prepared as rescue medications for bradycardia and hypotension respectively. Nausea vomiting, if any, was treated with ondansetron 4 mg intravenous bolus. Level of sedation was monitored according to the modified Ramsay Sedation Scale (RSS).[15]

2.4. Nerve stimulator guided SBPB

The patients were allocated on the basis of computer generated random numbers into two study groups: Ropivacaine group (R) and Ropivacaine plus intravenous dexmedetomidine group (RD). Intravenous access was done with 18 G cannula in the non operated arm. Ringer's lactate as intravenous fluid was started at 5mls/kg body weight/hour.

SBPB was done to the patients in supine position with ipsilateral arm adducted and head turned away to the opposite side in the following sequence:-

- The interscalene groove, subclavian artery pulsation, and midpoint of the clavicle were identified and marked with marker pen.
- After disinfection and sterile preparation of the injection site, a skin wheel was raised with lignocaine 2% plain 1.5-2 cms lateral to the site of pulsation of the subclavian artery.
- An insulated 1.5 inch 25G stimuplex needle (B Braun, Germany) was introduced through the infiltrated skin backward downward and laterally. The needle was connected to the nerve locator and 0.5mA current at 1Hz was applied with the nerve stimulator (B Braun, Germany).
- Localization of the plexus was considered optimal when an output current of 0.5mA caused contraction of the muscles of the hand and forearm.

Once contractions were obtained, local anesthetic was injected after repeated negative aspirations to avoid inadvertent intravascular injection. Both Ropivacaine group (R) as well as Ropivacaine plus Dexmedetomidine group (RD) received 20 mls 0.75% Ropivacaine (Ropin 0.75%, Neon Laboratories, Mumbai) plus 10 mls normal saline (made to a total volume of 30 mls of 0.5% ropivacaine) for SBPB.

2.5. Systemic administration of dexmedetomidine or saline

In the RD group, patients received dexmedetomidine 0.5mcg/kg body weight (Dextomid 100 mcg in 1 ml ampoule, Neon Laboratories, Mumbai) plus normal saline (appropriate amount made to a total volume of 10mls) via intravenous route over 10 minutes after the block was completed. In group R, patients received 10mls normal saline intravenously over 10 minutes.

2.6. Evaluation of sensory scores

A pinprick test in comparison to the contra-lateral area supplied by the ulnar, radial, median, and musculocutaneous nerves was performed on a 3 point scale [16]:
Scale 0 = normal sensation
Scale 1= loss of sensations to pinprick (analgesia)
Scale 2 = loss of sensation to touch (anesthesia)

2.7. Evaluation of motor scores

Motor block was assessed by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), flexion of the elbow (musculocutaneous nerve) according to the Modified Bromage Scale (MBS)[17] as follows:-
Grade 0: Normal motor function with full flexion and extension of elbow, wrist, and fingers.
Grade 1: Decreased motor strength with ability to move fingers only.
Grade 2: Complete motor block with inability to move fingers.
Sensory and motor blocks were evaluated as follows: prior to the block, then at 2,4,6,8,10,15,20,30, 60 minutes after the block, and finally every 30 minutes until complete recovery.

2.8. Definition of block success

SBPB was considered successful when there was both effective sensory and motor blockade in at least three out of four nerve territories (ulnar, radial, median, and musculocutaneous). Achievement of scale 2 of pinprick testing for sensory and grade 2 of MBS for motor blockade were considered effective.

2.9. Definitions of time points

Sensory onset time: time from performance of the block to achieving scale 2 of pinprick testing in the sensory areas. Duration of sensory block: time from achieving scale 2 of pinprick testing in sensory areas till regression of the block to scale 1.Complete recovery from sensory block: time from performance of the block to achieving scale 0 of pinprick testing in the sensory areas.Motor onset time: time from performance of the block to achievement of Grade 2 of MBS.Duration of motor block: time from achievement of grade 2 of MBS till regression to grade 0.

2.10. Evaluation of Intraoperative Sedation

Sedation was assessed according to the modified Ramsay Sedation Scale (RSS).[15]
1 = anxious, agitated, restless
2 = cooperative, oriented, tranquil
3 = drowsy but responds to commands
4 = brisk response to light glabellar tap or loud noise
5 = sluggish response to light glabellar tap or loud noise
6 = asleep and no response

2.11. Post study investigations

Within 72 hours after the study, patients underwent a final examination to investigate for any clinical signs of nerve damage (full recovery from the nerve block) and for any signs of inflammation or infection in the puncture area.

2.12. Statistical Analysis

We used the data published by Marhofer and colleagues[8] who showed prolongation of sensory block from 350 (54) to 395 (40) minutes in patients receiving ulnar nerve block without and with intravenously administered dexmedetomidine. Keeping the power of 80% with $P < 0.05$, the minimum sample size required was 12 patients in each group to detect at least 13% difference in duration of analgesia. However, we included 30 patients in each group for better validation of the results. All parametric data are presented as mean (SD). Non-parametric data are tabulated. Parametric data were analyzed using Student's t-test and non-parametric data were analyzed using Chi-square test and Fischer exact test as appropriate. $P < 0.05$ was considered statistically significant. Statistical analysis was performed with the help of Epi Info ™ 3.5.3; which is a trademark of the Centers for Disease Control and Prevention (CDC).

III. Results

The demographic data are presented in Table 1. Age, gender, ASA status, height, weight, and BMI of the patients were comparable.Distribution of the operative procedures is shown in Table 2. Corrected Chi-square showed that type of surgery is more or less equally distributed in the two groups ($\chi^2 = 2.36$; $P = 0.66$). Duration of surgery which has been tabulated in Table 3 shows no significant difference between the two groups. Table 4 shows that mean onset of sensory block (in minutes) in group RD is significantly less than that in group R ($P<0.0001$). However, there is no significant difference in mean duration of analgesia (in minutes) between the two groups. Table 5 shows that mean onset of motor block (in minutes) in group RD is significantly less than that in group R($P<0.0001$). There is no significant difference in mean duration of motor block (in minutes) between the two groups. Mean intra-operative heart rate (HR) of the patients in group RD was significantly lower than that in group R at 10 minutes after the intravenous dexmedetomidine injection which lasted up to 90 minutes ($P < 0.05$) (Table 6, Fig.1). Mean intra-operative mean arterial pressure (MAP) of the patients in group RD was lower than that in group R at 10 minutes after the intravenous dexmedetomidine injection which lasted up to 90 minutes; but was not statistically significant ($P > 0.05$) (Table 6, Fig. 2).

Mean RSS of the patients in group RD was significantly higher than that in group R at 30 minutes after the intravenous dexmedetomidine injection remaining until 120 minutes post injection ($P < 0.0001$) (Table 7, Fig. 3).

IV. Discussion

Brachial plexus block for upper extremity surgeries is widely considered as the anesthesia of choice because this causes profound sensory and motor block of the upper extremity, good muscle relaxation, less intra operative blood loss due to sympathetic blockade, little or no systemic side effects like nausea and vomiting, as well as excellent postoperative analgesia with consequent opioid sparing effect.[18] The plexus is most compactly present and easily accessible in the posterior triangle of the neck.[19] Consequently, in our study supraclavicular approach to block the brachial plexus was chosen as it could result in predictable analgesia of the hand and forearm extending to the lower third of the humerus. We used ropivacaine as LA of choice because it is a pure S (-) enantiomer with considerable cardiovascular and neurological safety profile. It also results in less motor block than bupivacaine due to its reduced lipophilicity.[3] In an effort to enhance the effects of LAs in regional nerve blockade, alpha2 agonists clonidine and dexmedetomidine among other group of drugs are being used as adjuvants either perineurally[20] or intravenously[21]. Dexmedetomidine is a highly selective alpha2 agonist with a relatively high ratio of alpha2/alpha1 activity (1620: 1 as compared to 220: 1 for clonidine).[22]

Clinical evidence for the use of dexmedetomidine as an adjuvant to LA for peripheral nerve blocks is limited mainly to animal studies,[23,24,25,26] as well as one study each in patients undergoing hand surgery,[9] and repair of cleft palate.[27] The clinically more relevant study done by Esmaoglu et al showed that dexmedetomidine added to levobupivacaine for axillary brachial plexus block shortened the onset time, prolonged the duration of the block and provided good postoperative analgesia.[9]

Clinical study is lacking specifically regarding SBPB along with intravenously administered dexmedetomidine. Rutkowska et al investigated the effect of intravenous dexmedetomidine in a dose of 1mcg/kg body weight bolus followed by 0.2-0.7 mcg/kg body weight infusion on SBPB done with bupivacaine and adrenaline in patients with end-stage renal disease sedated with either dexmedetomidine or midazolam for the formation of arteriovenous fistula. The motor and sensory blocks were significantly longer in the dexmedetomidine group.[14] In the study by Marhofer et al, even 20 mcg intravenous bolus of dexmedetomidine resulted in prolongation of sensory and motor blocks by 13% during ulnar nerve block with 0.75% ropivacaine.[8]

In our study we opted for dexmedetomidine intravenous 0.5mcg/kg body weight. We found that the onset of sensory block was significantly earlier in RD group (13.77 minutes) than in R group (17.57 minutes) ($P <0.0001$). The onset of motor block was also significantly earlier in RD group (15.50 minutes) in comparison to R group (20.03 minutes) ($P<0.0001$). Results of our study is comparable to the study done by Marhofer et al who found early onset of sensory and motor blocks in the systemic dexmedetomidine group. Mean duration of sensory and motor blocks in RD group were also increased although statistically insignificant which was in contrast to the study done by Rutkowska et al who used dexmedetomidine intravenous bolus in a much higher dose followed by continuous infusion whereas in the present study we used dexmedetomidine intravenous bolus only without continuous infusion. The study by Marhofer et al showed prolongation of ulnar nerve block with intravenous 20 mcg dexmedetomidine bolus; the dose of dexmedetomidine being less than that used in our study. The reason why our study showed statistically insignificant prolongation of SBPB could be that brachial plexus is a much thicker bundle of nerves in comparison to the ulnar nerve. Hence, intravenous dexmedetomidine bolus followed by infusion was probably needed to produce statistically significant prolongation of SBPB which had been found in the study of Rutkowska et al who used bolus followed by continuous infusion along with SBPB.

The mechanism by which alpha2 adrenoceptor agonists produce analgesia and sedation is not fully understood but can be multifactorial.[22] Peripherally, alpha2 agonists produce analgesia by reducing release of norepinephrine. Centrally, they cause inhibition of substance P release in the nociceptive pathway at dorsal root neuron. Dexmedetomidine have shown to block hyperpolarization-activated cation current (I_h current) resulting in prolonged hyperpolarization of the nerve, more for unmyelinated C fibres (pain) than A alpha fibres (motor).[28]

Dexmedetomidine can cause dose dependent side effects such as bradycardia and hypotension probably due to its central sympatholytic action and enhanced vagal activity.[29] In our study, although mean heart rate reduced between the time period starting 10 minutes after dexmedetomidine intravenous injection until 90 minutes post injection, heart rate in no patient was less than 60 per minute at any point of time (Fig. 1) and we did not have to use rescue medication in a single patient. Mean arterial pressure, in our study, did not reduce below 75 mmHg (Fig. 2) and no patient needed rescue medication. This is in accordance with the study of Rutkowska et al where none of the patients, although in end stage renal disease, was excluded from the study because of serious hemodynamic complications. In our study there was significant difference between the two groups of patients when sedation was assessed by RSS (Fig. 3) although not a single patient in the RD group showed excessive sedation requiring airway support. The level of sedation that we achieved kept the patients calm, and cooperative. The findings are similar to what Rutkowska et al observed the RSS between 3 and 4 in their study.

We feel that further studies are required to determine the dose-response and the effects of dexmedetomidine on complex nerve structures such as brachial plexus. These studies should test systemic administration of dexmedetomidine in a control arm. In the present study, we could not use perineural dexmedetomidine due to the paucity of recommendation regarding the perineural use of the drug in the Indian Pharmacopeia. However, the data available from the few animal and human studies in the past and our present study are promising.

The present study shows that administration of intravenous dexmedetomidine 0.5 mcg/ kg body weight along with SBPB using 0.5% ropivacaine 30 mls resulted in quicker onset of both sensory and motor block than when ropivacaine was used alone. The incidence of hemodynamic variations e.g. bradycardia was statistically more significant in the group of patients who had intravenous dexmedetomidine with ropivacaine than the group receiving ropivacaine alone. Patients who had intravenous dexmedetomidine showed statistically insignificant reduction in MAP. However, the complications did not appear to be clinically significant as none of the patients required rescue interventions. Satisfactory intra operative sedation without any respiratory depression was an added advantage obtained in the dexmedetomidine with ropivacaine group, which was absent in the group receiving ropivacaine alone.

Table-1: Demographic Data

Demographic Parameters	Group R (n=30)	Group RD (n=30)	t_{58}-value	p-value
Age (years)	44.27±13.91	42.97±14.13	0.35	0.72
Gender (Male:Female)	15:15	18:12	0.60	0.43
ASA (I:II)	16:14	17:13	0.06	0.79
Height (cm)	167.33±9.63	169.77±9.68	0.97	0.33
Weight (kg)	65.40±6.17	65.73±6.53	0.20	0.84
BMI (kg/m^2)	23.40±1.78	22.86±2.05	1.08	0.28

Table-2: Comparison of type of surgery

Type of Surgery	Group-R (n=30)	Group-RD (n=30)	TOTAL
BB PLATING	8	7	15
Row %	53.3	46.7	100.0
Col %	26.7	23.3	25.0
ORIF-HUMERUS	4	7	11
Row %	36.4	63.6	100.0
Col %	13.3	23.3	18.3
ORIF-OLECRANON	4	4	8
Row %	50.0	50.0	100.0
Col %	13.3	13.3	13.3
ORIF-RADIUS	9	5	14
Row %	64.3	35.7	100.0
Col %	30.0	16.7	23.3
ORIF-ULNA	5	7	12
Row %	41.7	58.3	100.0
Col %	16.7	23.3	20.0
TOTAL	30	30	60
Row %	50.0	50.0	100.0
Col %	100.0	100.0	100.0

Table-3: Duration of surgery (minutes)

	Group-R (n=30) (Mean ± s.d.)	Group-RD (n=30) (Mean ± s.d.)	Test Statistic (t_{58})	p-value
Duration of surgery (min)	120.83±11.07	121.17±12.43	0.11	0.91

Table-4: Comparison of Sensory Block

	Group R (n=30) (Mean ± s.d.)	Group RD (n=30) (Mean ± s.d.)	Test Statistic (t_{58})	p-value
Onset of Sensory Block (min)	17.57 ±1.10	13.77±0.94	14.38	<0.0001*
Duration of Sensory Block (min)	428.83±5.84	431.10±4.71	1.65	0.10

* Statistically Significant

Table-5: Comparison of Motor Block

	Group-R (n=30) (Mean ± s.d.)	Group-RD (n=30) (Mean ± s.d.)	Test Statistic (t_{58})	p-value
Onset of motor block (min)	20.03±1.16	15.50±1.07	15.72	<0.0001*
Duration of motor block (min)	411.00±11.99	412.03±5.92	0.42	0.67

* Statistically Significant

Table-6: Comparison of peri-operative complications

Side Effect	Group-R (n=30) (Mean ± s.d.)	Group-RD (n=30) (Mean ± s.d.)	Z-value	p-value
Bradycardia	0 (0.0%)	4(13.3%)	2.07	<0.05*
Hypotension	0 (0.0%)	3(10.0%)	1.77	>0.05
Nausea/Vomiting	0 (0.0%)	3(10.0%)	1.77	>0.05

* Statistically Significant

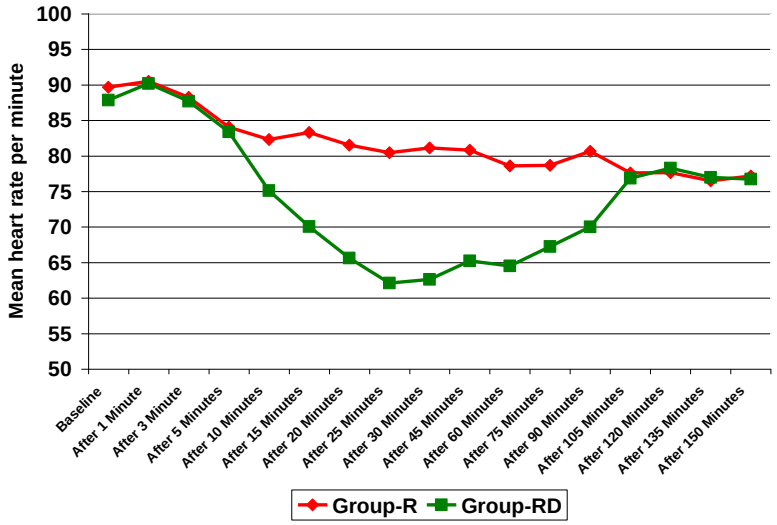

Figure 1: mean heart rate at different times

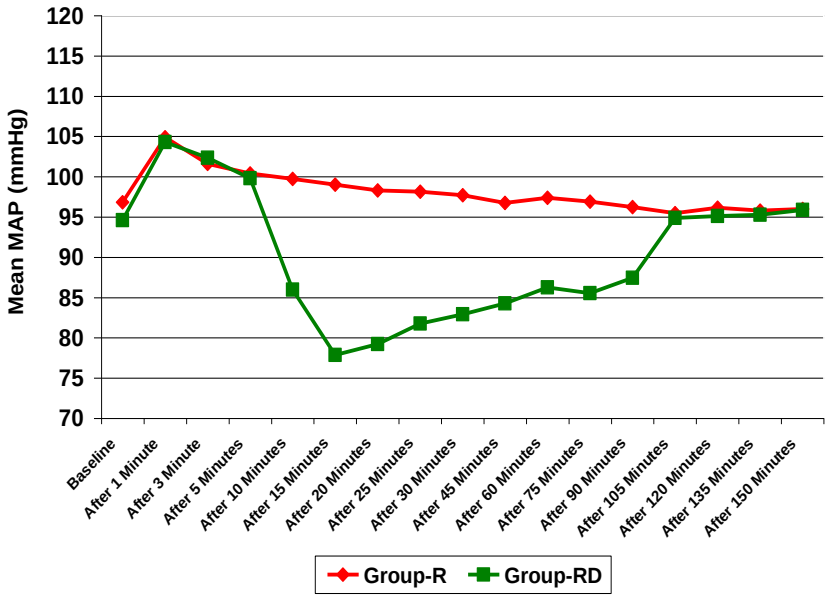

Figure 2: mean MAP at different times

Table 7: Comparison of RSS

Time interval (in minutes)	**Group-R (n=30) (Mean ± s.d.)**	**Group-RD (n=30) (Mean ± s.d.)**	**Test Statistic (t_{58})**	**p-value**
RSS_0	1.50±0.51	1.57±0.50	1.52	0.61
Intra-operative				
RSS_10	1.67±0.48	1.60±0.50	0.52	0.59
RSS_30	1.67±0.48	3.50±0.51	14.85	0.0001*
RSS_60	1.67±0.48	3.77±0.43	17.85	0.0001*
RSS_90	1.67±0.48	3.87±0.35	20.38	0.0001*
RSS_120	1.67±0.48	2.97±0.56	9.69	0.0001*
RSS_150	1.57±0.50	1.83±0.65	1.78	0.08

* Statistically Significant

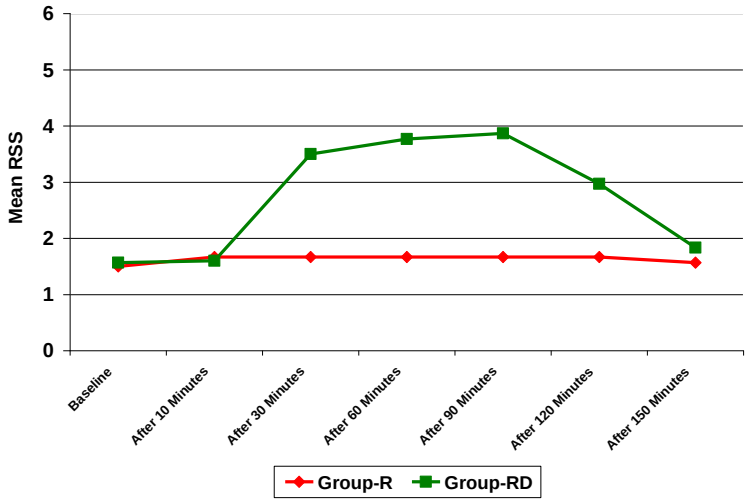

Figure 3: RSS at different times

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