ORIGINAL ARTICLE

COMPARATIVE STUDY ON THE EFFECT OF ADDITION OF POTASSIUM CHLORIDE TO BUPIVACAINE ON THE ONSET TIME AND DURATION OF BRACHIAL PLEXUS BLOCK

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ABSTRACT: Background and objectives: Regional anaesthetic technique such as brachial plexus block by supraclavicular approach is widely used for upper limb surgeries. This study compared the addition of potassium chloride to bupivacaine on the onset time and duration of brachial plexus block. **METHODS:** Sixty patients between 20 - 70 years of either sex of ASA I and ASA II were randomly allotted to one of the two groups of thirty each. Group I received 30ml of 0.375% bupivacaine with 0.2mmol of potassium chloride. Group II received 30ml of 0.375% plain bupivacaine. Sensory blockade were determined by pinprick test in the C_4- T_2 skin dermatomes, while motor blockade was graded according to the movement of upper limb by the patient. **RESULTS:** Potassium chloride group showed rapid onset of sensory and motor blockade when compared to other group ($p< 0.001$). The decreased requirements of adjuvants in potassium group suggest greater quality of blockade with respect to other group. The duration of anaesthesia was prolonged in potassium group in relation to other group ($p<0.001$). **CONCLUSION:** These data suggest that addition of potassium chloride to bupivacaine for brachial plexus block significantly decreases onset time, increases depth of blockade and prolongs the duration.

KEYWORDS: Local anaesthesia, Bupivacaine, Brachial plexus, Potassium chloride.

INTRODUCTION: Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage[1] It is always a subjective experience. Pain has been a major concern of human kind and it has been the object of ubiquitous efforts to understand and to control it. Peripheral nerve blocks provide longer and more localized pain relief than neuraxial techniques while also avoiding the side effects of systemic medication. Regional anaesthesia of the extremities and of the trunk is a useful alternative to general anaesthesia in many situations.

Regional anaesthesia denotes interruption of pain impulse by physiological blockade at a certain point along their pathway of transmission in peripheral nerves. Trephination was practiced by Incas, and their tradition holds that the 'Shaman' performing the procedure chewed cocoa leaves and spat into the wound producing local anaesthetic effect.[2]

Koller's demonstration in 1884 of ocular surface anaesthesia with cocaine has marked a new era in medicine namely the use of regional anaesthetics for prevention of pain associated with surgery. Brachial plexus nerve block was reportedly first accomplished by Halsted, when he 'freed the cords and nerves of the brachial plexus - after blocking the roots in the neck with

cocaine solution.[3]

Brachial plexus block has evolved into a valuable and easy procedure for upper limb surgeries. Hers oh el introduced axillary and supraclavicular techniques. Most of the local anaesthetic agents developed in between 1900 - 1940 were basically ammo ester compounds. They lost their importance due to short duration of action, associated allergic reactions and systemic toxicity.

Lofgren and associate continued the work with great energy and after investigating with more than hundred compounds found Xylocaine, a local anaesthetic preparation, which marked a considerable advance. In 1957 Ekenstam synthesized bupivacaine, an amide local anaesthetic and was first clinically used in 1963 by Telivuo.

The main drawback of long acting drugs was delayed onset of action. To overcome this drawback following were tried like, addition of enzymes,[4] buffered and carbonated solutions,[5] opioids,[1] vasoconstricting agents,[1] alkalization and warming up of local anaesthetic solutions,[6] and potentiation of blockade by pain and muscular exercise[7] of these, only additions of carbonates and potassium to local anaesthetics have stood the test of time. Hence an attempt was made to compare the effects of adding potassium chloride to bupivacaine for the onset time and duration of sensory and motor blockade following supraclavicular brachial plexus block.

MATERIALS AND METHODS: The present study entitled "Comparative study of the effects of addition of potassium chloride to bupivacaine on the onset time and duration of brachial plexus block" was carried out at Rajah Muthiah Medical College Hospital in a double blind-manner.

Sixty patients of age group between 20-70 years of either sex of ASA grade I and II, were selected for the study. The patients were undergoing elective and emergency surgery of the upper limb.

Exclusion criteria included:

1. Patient's refusal,
2. Progressive neurological discords,
3. Severe kidney or liver dysfunction and
4. History of bleeding disorders.

Each patient was visited pre operatively and the procedures were explained and informed written consent was obtained.

Investigations like Hb%, TC, DC, ESR, random blood sugar, electrolytes, urine albumin and sugar, chest X-ray, ECG were done.

All the patients were pre-medicated with Inj Diazepam 5mg IM 30 minutes surgery.

Each patient was randomly assigned to one of the two groups (30 patients each), group I or Group II. Group I (Potassium group) received 30 ml of 0.375% bupivacaine with 0.2 mmol of potassium chloride (prepared by adding 0.1ml of potassium chloride and 10ml distilled water to 20 ml of 0.5% bupivacaine). Group, II (Plain bupivacaine group) received 30ml of 0.375% bupivacaine only.

Each patient was made to lie supine without a pillow, arms at the side, head turned slightly to the opposite side with the shoulders depressed posteriorly and downward by moulding the shoulders over a roll placed between the scapulae.

The supraclavicular area was aseptically prepared and draped. The anaesthesiologist stands at the head end of the patient, facing the leg, since this position allows better control of needle.

An intradermal wheal was raised approximately l cm above the midclavicular point. The subclavian artery palpable in supraclavicular fossa was used as landmark. The tip of index finger was rested in supraclavicular fossa directly over the arterial pulsation. A filled 10ml syringe with a 23 gauge, 32mm needle attached was held in right hand and patient was instructed to say "now" and not to move as soon as he felt a "tingle" or "electric shock like sensation" going down his arm. The needle was inserted through skin and advanced slowly downward (caudal) rolled slightly inward (medially) and slightly backward (posteriorly).

As soon as paraesthesia was elicited, the needle was fixed in position and after confirming negative aspiration of blood, 30ml of the respective drug was injected depending on whether the patient was allotted to either of group I or II.

Time of onset of sensory block was recorded using pinprick in skin dermatomes C4-T2 once in every 3 minutes for the first 30 minutes after injection and there after every 30 minutes till patient regained normal sensations. The same observer assessed the motor block at same time intervals.

The person doing the procedure did not know whether the dilution contained plain bupivacaine or with potassium chloride. Onset of sensory block was from the time of injection of drug to time of loss of pain on pinprick. Onset of motor block was from the time of injection to time of complete loss of movement.

Sensory block was assessed by pinprick with a short beveled 25G needle as:

0 - No pain
1 - Mild pain-grimace,
2 - Moderate pain-withdrawal, and
3 - Severe pain-screams.

Motor block was graded according to the movement of upper limb by the patient as:
Grade:

5 - Normal movement of upper limb,
4 - Movement against resistance,
3 - Movement against gravity,
2 - Movement along gravity but not against resistance,
1 - Flickering movement and
0 - No movement.

Grade 3, 2, 1 were partial block.

Grade 0 - complete motor paralysis that is when the patient could not move his limb at all.

Duration of sensory blockade was the time in minutes from the onset of analgesia to the recurrence of pain to pin prick. Duration of motor blockade was the time in minutes from the onset of paresis to the recurrence of motor movements.

The quality of sensory and motor block was studied and graded as per whether the blocks were complete, incomplete or totally absent.

The usage of adjuvants after the block was graded according to whether the surgery was done.

Grade III: Surgery done under general anaesthesia due to complete failure of block.
Grade II: Opioids were used during intra operative period. (or)
Grade I: Adjuvants of any kind were not used throughout the surgery.

The heart rate and blood pressure were recorded at intervals of 5 minutes. The patients were watched for bradycardia, convulsions, restlessness, is orientation, drowsiness and any other complications.

All the values were expressed as mean ± standard deviation. Statistical comparison was performed by student-'t' test and CHI-SQUARE test.

p value of >0.05 was considered to be statistically not significant, a p value <0.05 as 'statistically significant, a p value of <0.01 as statistically highly significant and a p value of <0.001 as statistically very highly significant.

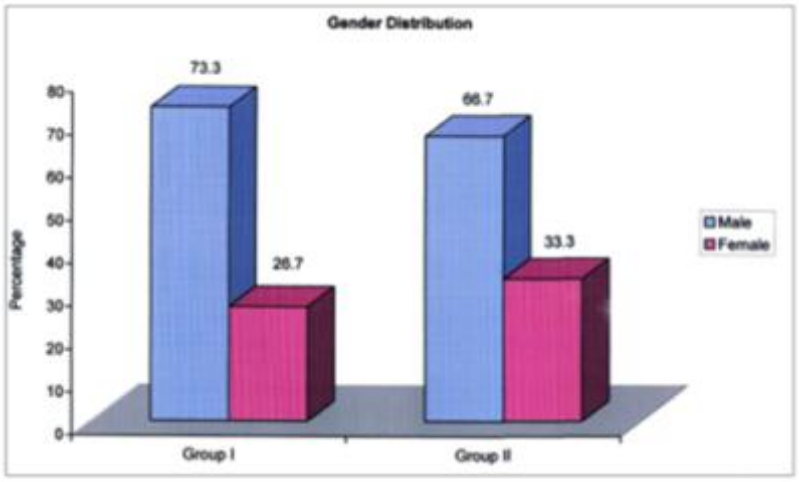
RESULTS AND ANALYSIS: The present study was conducted on 60 consenting patients aged between 20-7 years. Group I received 30ml of 0.375% bupivacaine with 0.2mmol of potassium chloride. Group II received 30ml of 0.375% bupivacaine for branchial plexus block by supraclavicular approach.

DEMOGRAPHIC DATA:

	Group I (%)	**Group II (%)**
Male	73.3%	66.7%
Female	26.7%	33.3%
Total	**100%**	**100%**
Gender distribution		

X^2 (1)=0.317, p=0.573, ns

The two groups were similar in sex wise distribution as shown in the above figure.



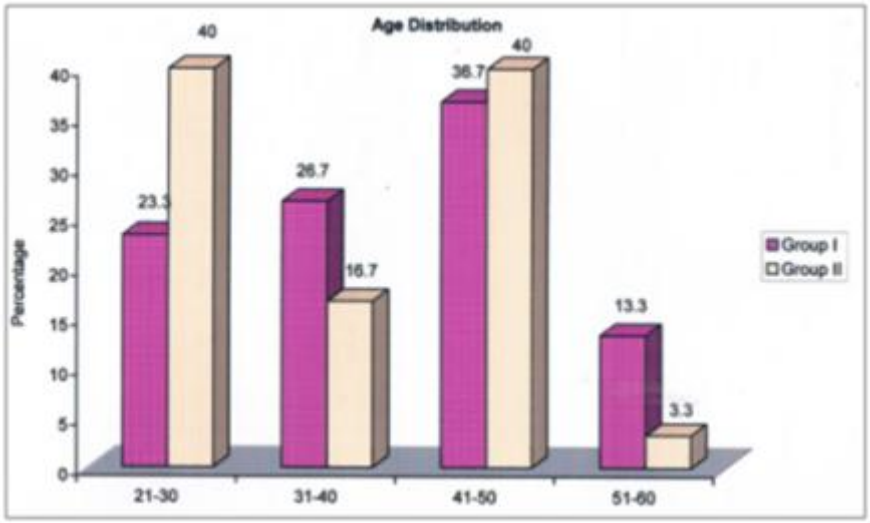
AGE DISTRIBUTION:

		Group		Total
		Group I	**Group II**	
Age (years)	21-30	7(23.3%)	12(40.0%)	19(31.7%)
	31-40	8(26.7%)	5(16.7%)	13(21.7%)
	41-50	11(36.7%)	12(40.0%)	23(38.3%)
	51-60	4(13.3%)	1(3.3%)	5(8.3%)
Total		**30(100%)**	**30 (100%)**	**30(100%)**

Table 1: Age Distribution

X^2 (3)=3.852, p=0.278, ns

The two groups were similar in age as shown in the above table.



WEIGHT DISTRIBUTION:

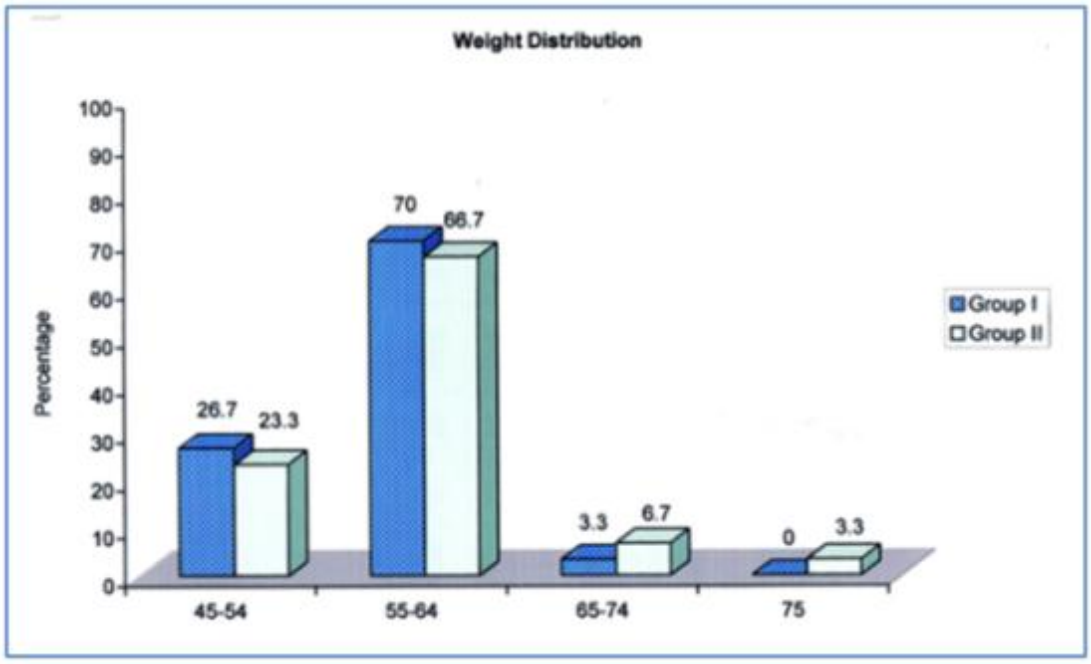
	Group I	Group II
45-54	26.7	23.3
55-64	70	66.7
65-74	3.3	6.7
75	0	3.3

X^2 (1)=0.317, p=0.573, ns

The two groups were similar in weight as shown in the above figure.

Group		Mean	N	Std. Deviation	Minimum	Maxi mum	
Age	Group I	41.03	30	10.301	22	60	t(58)=1.135 p=0.261, ns
	Group II	38.00	30	10.396	25	60	
	Total	39.52	60	10.374	22	60	
WtKgs	Group I	56.37	30	4.476	48	65	t(58)=-1.303 p=0.198, ns
	Group II	58.27	30	6.617	50	80	
	Total	**57.32**	**60**	**5.682**	**48**	**80**	

Figure 5: Weight Distribution



Group		Mean	N	Std. Deviation	Minimum	Maximum	
Onset of blockade (Mins) Sensory	Group I	10.43	30	1.633	8	13	t(58)=20.889 p=0.0001HS
	Group II	26.33	30	3.836	20	33	
Onset of blockade (Mins) Motor	Group I	9.46	30	1.547	8	12	t(58)=20.740 p=0.0001 HS
	Group II	23.93	30	3.503	18	30	
Duration of blockade (Mins) Sensory	Group I	467.67	30	26.579	420	520	t(58)=44.60 p=0.0001 HS
	Group II	205.67	30	18.134	170	240	
Duration of blockade (Mins) Motor	Group I	477.67	30	26.579	430	540	t(58)=44.60 p=0.0001 HS
	Group II	215.67	30	18.134	180	250	

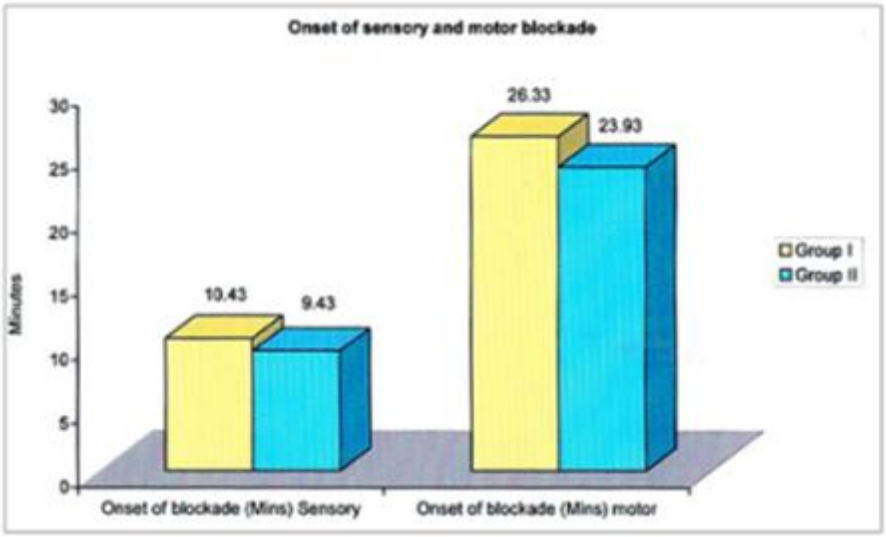
Table 3: comparison of onset and duration of sensory and motor blockade

	Group I	Group II
Motor Blockade	9.43	23.93
Sensory Blockade	10.43	26.33

Onset of sensory and motor blockade

Figure 6: Comparison of onset of sensory and motor blockade.

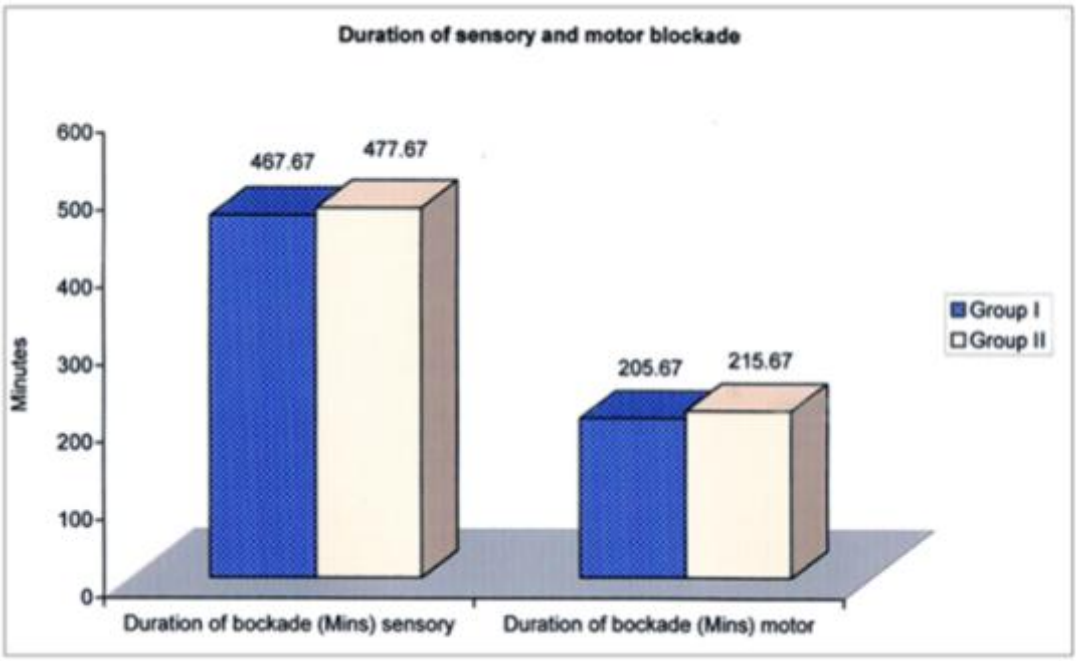
In group I the mean onset time of sensory blockade was 10.43 min and motor blockade was 9.43 min when compared to group II having sensory onset of 26.33 min and motor onset of 23.93 min.



	Group I	Group II
Motor Blockade	477.67	467.67
Sensory Blockade	215.67	205.67

Table 7: Comparison of duration of sensory and motor blockade

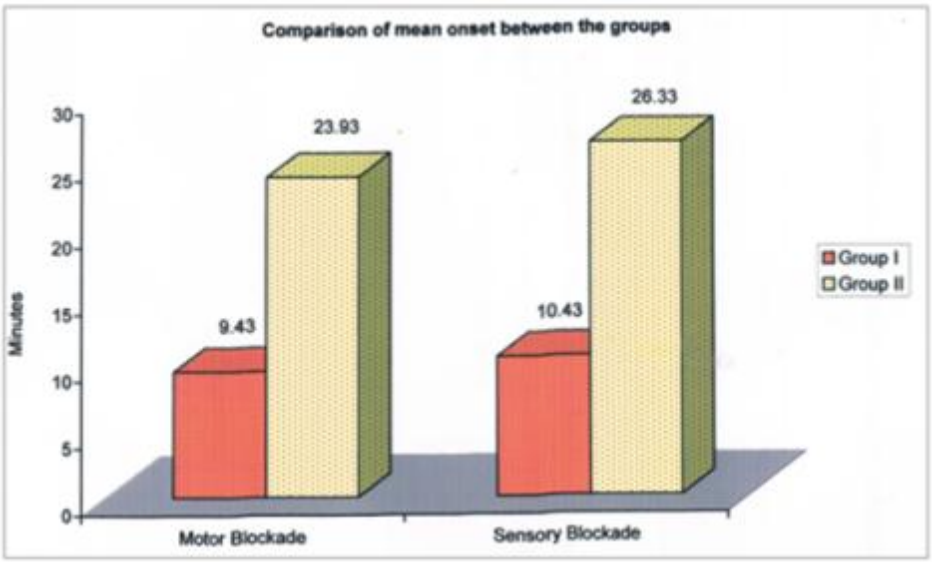
In group I the mean onset time of sensory blockade was 467.67 min and motor blockade was 477.67 min when compared to group II having sensory onset of 205.67 min and motor onset of 215.67 min.



	Group I	**Group II**
Motor Blockade	9.43	23.93
Sensory Blockade	10.43	26.33

Figure 8: Comparison Mean onset of sensory and motor blockade

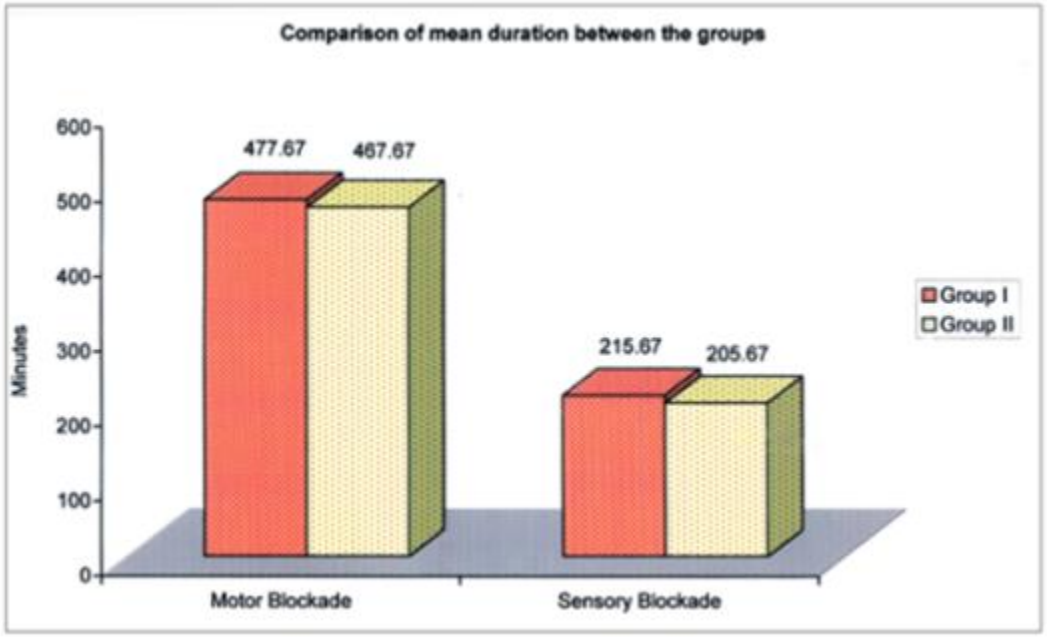
Onset of sensory and motor blockade was earlier in case of Group I when compared with group II. The p value was < 0.001 which is statistically highly significant.



	Group I	Group II
Motor Blockade	477.67	467.67
Sensory Blockade	215.67	205.67

Figure 9: Comparison Mean duration of sensory and motor blockade

The duration of both sensory and motor blockade was prolonged in Group I when compared to group II. The p value was < 0.001, which is very highly significant.

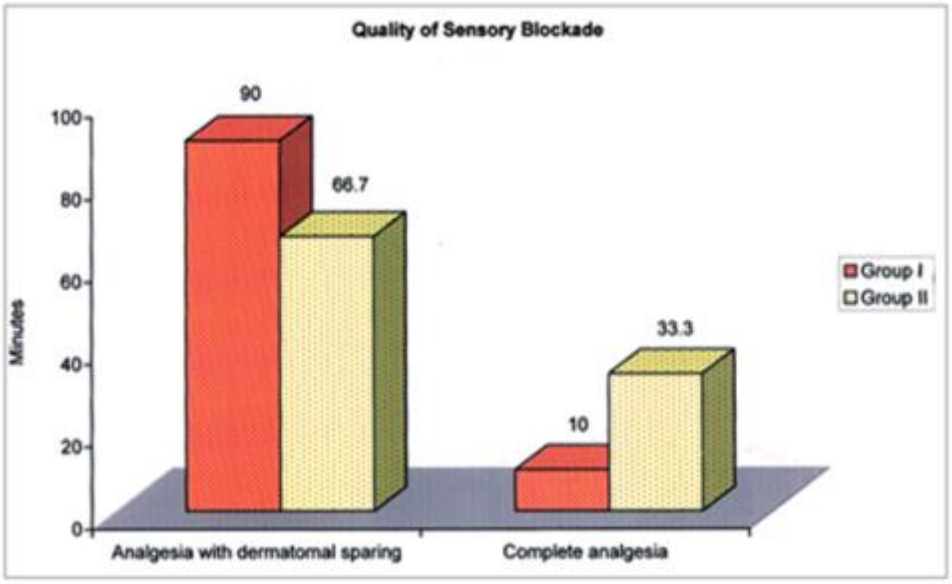


	Group I	Group II
Analgesia with dermatomal sparing	90	66.7
Complete analgesia	10	33.3

Table 10: Quality of sensory blockade

X^2 (1) =4.812, p=0.028 sig

The quality of sensory blockade was better in group I and the value was statistically significant when compared with group II.

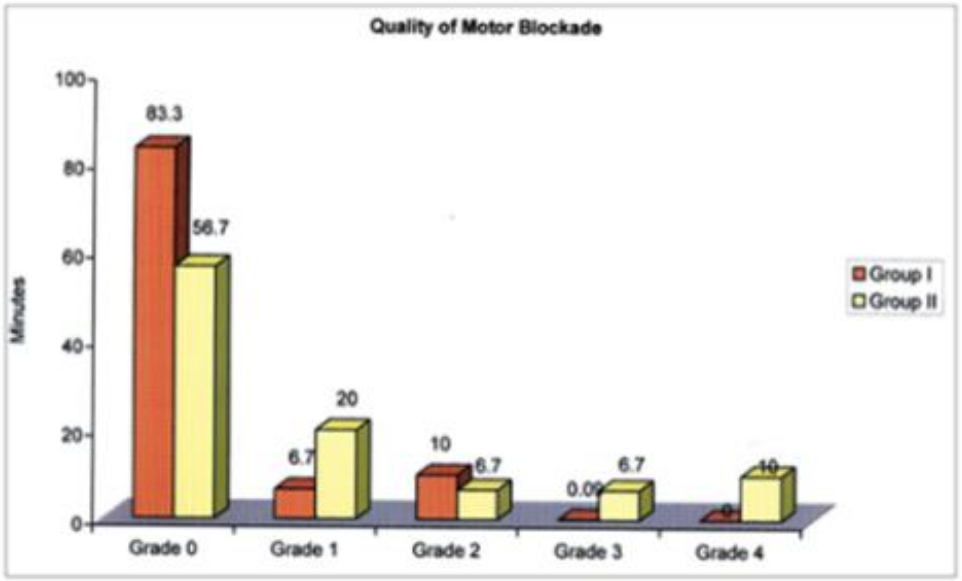


	Group I	Group II
Grade 0	83.3	56.7
Grade 1	6.7	20
Grade 2	10	6.7
Grade 3	0.09	6.7
Grade 4	0	10

Table 11: Quality of motor blockade

LRX^2 (4) =10.759, p=0.029 sig

The quality of sensory blockade was better in group I and the value was statistically significant when compared with group II.

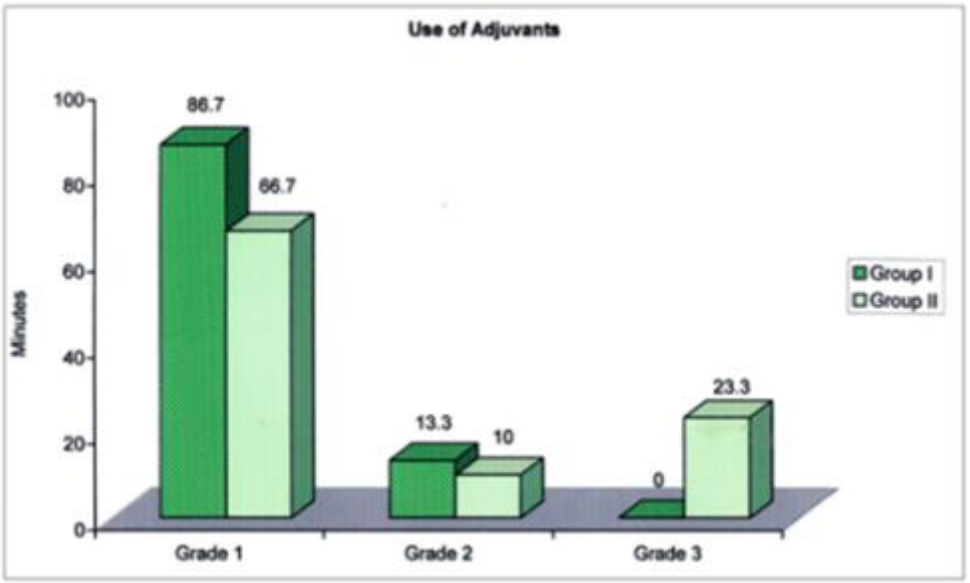


	Group I	Group II
Grade 1	86.7	66.7
Grade 2	13.3	10
Grade 3	0	23.3

Table 12: Use of adjuvants

LRX^2 (2)=10.632, p=0.005 sig

The number of adjuvants used in group I were significantly less when compared to group II. The p value was <0.05 which is significant.



DISCUSSION: Brachial plexus' block is widely used in our practice for elective forearm and hand surgeries. It provides good intraoperative and postoperative analgesia. Many substances have been added to local anaesthetic agents in- an attempt to prolong their duration 'of action. Among them addition of carbonated solution and potassium to local anaesthetic have stood the test of time.

Addition of potassium chloride to local anaesthetic solutions increases the extracellular potassium concentrations and depolarizes the membrane.

We conducted studies on sixty patients with demographic data in terms of age, weight and sex being similar in both the groups. The data collected was analyzed for statistical significance by student 't' test and CHI-SQUARE test.

The onset of blockade in potassium group- was earlier when compared to plain bupivacaine group. In our study the mean onset of sensory and motor blockade in potassium group was 10.43 and 9.43 minutes respectively. The results of our study support the findings of Khosa et al.[8] who showed that addition of potassium chloride to bupivacaine significantly enhanced the onset of both sensory and motor blockade. In contrast to our study, the delayed onset of blockade proposed by Parris and Chamber[9] may be due to the lower concentration of bupivacaine (0.25%) when compared to our study (0.375%).

The duration of sensory and motor blockade was significantly increased ($p<0.00l$) in potassium group when compared to other group.

This is in agreement with Khosa et al's[8]. Findings who found prolonged duration of analgesia. We have found that depth of sensory and motor blockade was significantly better in potassium group when compared to other group. Bromage and Burfoot[10] also found intense quality of blockade when potassium was added to lignocaine in epidural blockade.

The decreased requirement of adjuvants in potassium group when compared to other group suggests greater quality of anaesthesia. The results of our study support the findings of. Parris and Chamber.[11]

Thus potassium chloride definitely has a role as an adjuvant to bupivacaine hydrochloride in shortening the onset time, prolonging the duration of, action and improving the quality of blockade in brachial plexus block.

Apart from anatomic variations, individual patient's responses and discrepancies in the number of patients studied should also be taken into account to explain the differences among the studies. Further study of other agents and sites of blockade is required.

BIBLIOGRAPHY:

1. Aldrete JA, Barnes DR, Sidon MA, McMullen RB. Studies on effects of addition of potassium chloride to lidocaine. Anesthesia and Analgesia 1969; 48:269-276.
2. Bonica JJ, Backup PH and Pratt WH. Use of vasoconstrictors to prolong spinal anaesthesia. Anaesthesiology 1951; 12: 431-441.
3. Bouaziz H, Brian PK, Macalou D et. al. Sufentanil does not prolong the duration of analgesia in a mepivacaine brachial plexus block: A dose response study. Anaesthesia and Analgesia 2000; 90:383-387.

4. Charles BB and Gary' RS. Local anaesthetics. Miller RD. Anesthesia, 5th ed. New York: Churchill Livingstone. 2000:491-517.
5. Collins. VJ. Principles of Anesthesiology. General and Regional Anesthesia, 3rd ed. Lea and Febgier* Philadelphia. 1993: 1287.
6. Benjamin GC and John AW. Clinical pharmacology of local anaesthetic agents. In: Cousins MJ and Phillip OB. Neural blockade in clinical anesthesia and management of pain. 3r ed. Lippincott- Raven, Philadelphia. 1998:105.
7. Fink BR, Calkins DF. Role of glucose or potassium lack in nerve block. Anesthesiology 1981; 55: 172-175.
8. Khosa DS, Thind SS, Gupta HK, Jain S. Effects of adding potassium chloride to lignoeaine and bupivacaine solutions on the onset time and duration of brachial plexus block. Indian Journal of Anaesthesia 1990; 38: 119-122.
9. Lanz E, Theiss D, Jankovic D. Extent of blockade following, various technique of brachial plexus block. Anesthesia and analgesia 1983; 62:55-58.
10. Bromage PR, Burfoot ME, Crowell DE and Truant AP. Carbonated local anaesthetic solutions. British Journal of Anaesthesia 1967; 39: 197 -209.
11. F Z5. Parris MR, Chamber WA. Effects of the addition of potassium to prilocaine or bupivacaine. British Journal of Anaesthesia 1986;1, 58:297- 300

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