

Effect of Adding Potassium Chloride to Bupivacaine in Brachial Plexus Block: A Cross Sectional Case Control Study

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Abstract

Background: Brachial plexus blockade for upper limb surgeries is the most common major peripheral nerve block technique and a significant difference exists between the onset times of various agents when these blocks are used. Adjuvants are added to improve the quality of anaesthesia and the duration of post-operative analgesia. The study was aimed to compare the addition of potassium chloride to bupivacaine in supraclavicular brachial plexus block and to assess the onset of blockade – sensory and motor blockade, duration and quality of sensory and motor blockade. **Methods:** Sixty patients of age group between 18 and 60 years with ASA grade I and II, were selected for the study. Each patient was randomly assigned to one of the two groups of 30 patients each, group I or group II. Group – I was bupivacaine who received 30 ml of 0.375% bupivacaine. (control group) and group – II received 30 ml of 0.375% bupivacaine with 0.2 mmol of potassium chloride (prepared by adding 0.1ml of potassium chloride and 10 ml of normal saline to 20ml of 0.5% bupivacaine). (Study group). Both group were compared with respect to onset, duration and quality of sensory and motor blockade.

Results: The onset of sensory and motor blockade was early in study/potassium group when compared to plain bupivacaine group. The duration of blockade was prolonged in study/potassium group when compared to other group. However, the quality of sensory blockade was better in potassium group when compared to other group. But the quality of motor blockade was similar that of plain bupivacaine group. **Conclusion:** Addition of potassium to bupivacaine when compared to plain bupivacaine is beneficial in supraclavicular brachial plexus blockade.

Keywords: Bupivacaine; Adjuvants; Brachial Plexus Block; Potassium.

Introduction

In response to tissue injury there is pain which is considered as unpleasant sensory and emotional effect. Various methods have been used for pain relief like oral drugs, nerve blocks but adequate pain relief is adequately provided by interrupting the transmission of pain. Despite increased knowledge and scientific advances, the diagnosis and effective treatment of pain remains one of the most formidable challenges with many difficulties and pitfalls. Regional

nerve blocks are based on the concept that the pain conveyed by nerve fibres; which are amenable to interruption anywhere along their pathway. The idea that pain is conducted in the nervous system originated with the specific theory of Johannes P Muller, described in 1826. This was followed by the alternate intensity theory of Erb in 1874; an idea that later culminated in the gate theory of pain by Melzack and Wall in 1965 [1]. In 1855, Rynd described the idea of introducing a solution of morphine hypodermically around a peripheral nerve [2]. Wood in 1855, was the first person to perform a subcutaneous injection with a graduated glass syringe and a hollow needle, a device developed initially by Pravaz for injection of ferric chloride into an aneurysm to produce coagulation [3]. Trephination was practiced by Incas, and their tradition holds that the 'Shaman' performing the procedure chewed Cocoa Leaves

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and spat into the wound producing local anesthetic effect [4]. In 1881, Carl Koller demonstrated ocular surface anesthesia with cocaine [5]. Ester local anesthetics which were developed later lost their value due to short duration of action, allergic reaction and systemic toxicity. Later amide anesthetics were synthesized. In the recent years peripheral nerve blocks are gaining importance for their longer duration of action and post operative analgesic effect. It avoids the side effects of general anaesthesia. Use of continuous plexus and nerve blocks addresses the wind up mechanism of pain.

The longest latencies and duration are observed after brachial plexus blocks, when bupivacaine is administered for brachial plexus blocks. The time of onset is approximately 20 to 30 mins, and the duration of anesthesia (or atleast analgesia averages 10 hrs). The onset of brachial plexus block is slow because the anesthetic is usually deposited at some distance from the nerve and must diffuse through various tissues barriers before reaching the nerve membrane. The prolonged block with brachial plexus may be related to several factors including comparatively slower rates of vascular absorption from the brachial plexus sheath; larger doses of the drug required for this technique; comparatively longer segments of nerves exposed to local anesthetics [6].

Several means have been tried in the past to shorten onset of action; prolong the duration of action. Hence adjuncts like neostigmine, alkalinization, clonidine, enzymes, buffer and carbonated solution, opioids, vasoconstricting agents, warming up of local anesthetic solutions, potentiation of blockade by pain and muscular exercise are done to enhance of the action and improve the quality of anesthesia [7-18].

Hence here an attempt has been done to compare the addition of potassium chloride to bupivacaine in supraclavicular brachial plexus block. The present study is aimed to compare the addition of potassium chloride to bupivacaine in supraclavicular brachial plexus block and to assess the onset of blockade – sensory and motor blockade, duration of the blockade – sensory and motor blockade and quality of sensory and motor blockade

Methodology

A cross sectional case control study was carried out at Basaveshwar Teaching and General Hospital, Gulbarga in a double blind manner. Sixty patients of age group between 18 and 60 years with ASA grade I and II, were selected for the study. The patients were

undergoing elective operative procedures for upper limb surgeries (i.e. elbow, forearm and hand surgeries) were taken. Exclusion criteria included patient's refusal, history of bleeding disorders or patients on anticoagulant therapy, patient's with neuromuscular disorders, burns, local infection, hyperkalemia, respiratory disease, known allergy to local anesthetic drugs and ASA grade III and IV patients. Each patient was visited pre operatively and the procedures were explained and informed written consent was obtained. Investigation like CBC, ESR, blood grouping, urine examination for albumin, sugar and microscopy, RBS, Bleeding time, clotting time, serum creatinine, blood urea, serum electrolytes (sodium, potassium), chest X-ray, ECG were done. All the patients were premedicated orally with tablet diazepam 0.2mg/kg one and a half hours before the procedure.

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

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 anesthesiologist stands on the side of the patient to be blocked, facing the head of the patient, since this position allows better control of needle.

An intradermal wheal was raised approximately 1cm 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 10 ml syringe with a 23 gauge, 32 mm 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) roiled 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, 30 ml of the respective drug was injected depending on whether the patient as 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 1 minute 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 23G needle as 0-no pain, I-mild pain-grimace, II-moderate pain-withdrawal, and III-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 hi 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 under general anaesthesia (grade III) due to complete failure of block, whether opioids were used during intra operative period (grade II) or if adjuvants of any kind were not used throughout the surgery (grade I).

The heart rate, saturation, respiratory rate and blood pressure were recorded at intervals of 5 minutes. The patients were watched for bradycardia, convulsions, restlessness, disorientation, drowsiness and any other complications. All the values were expressed as mean ± standard deviation Statistical comparison was performed by student's 't' test and Chi-Square test. A 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

The demographic profile of patient in shown in Table 1. Table 2 shows the comparison of group 1 and 2 on the basis of onset of blockade sensory and motor. Table 3 shows the duration of sensory and motor blockage respectively. The quality of sensory and motor blockade is shown in Table 4(a) and (b) respectively. The adjuvant used is shown in table 5 and the variations in heart rate, blood pressure respiratory rate arterial oxygen saturation at 5 mins, 15 min, 30 min and 45 min are shown in Table 6(a), 6(b), 6(c) and 6(d) respectively.

Table 1: Demographic profile of patients

Age	Study Group/Group II			Control Group/Group I			Grand total
	Male	Female	Total	Male	Female	Total	
18-30	14	4	18	12	3	15	33
31-40	3	3	6	4	1	5	11
41-50	2	0	2	0	3	3	5
51-60	2	2	4	4	3	7	11
Total	21	9	30	20	10	30	60

Gender $\chi^2 = 0.004$ P > 0.05 not significant

Table 2: Comparison of study and control group on the basis of onset of blockade sensory and motor

Onset Blockade (min)	No	Study group/ Group II			No	Control group/ Group I			t-test t-test	P-value & significance
		Min	Max	Mean ± S.D		Min	Max	Mean ± S.D		
Sensory	30	11	23	14.53 ± 2.09	30	17	29	25.46 ± 3.04	t = 16.12	P < 0.001 very highly significant
Motor	30	12	26	17.7 ± 2.81	30	20	30	27.86 ± 2.53	t = 16.42	P < 0.001 very highly significant

Table 3: Duration of blockade (min)

Duration of blockade	No	Study group/ Group II			No	Control group/ Group I			t-test	P-value & significance
		Min	Max	Mean ± SD		Min	Max	Mean ± SD		
Sensory	30	225	567	440.77 ± 85.09	30	156	483	336.46 ± 81.89	t = 4.84	P < 0.01 highly significant
Motor	30	151	555	357.6 ± 88.23	30	122	395	256 ± 76.4	t = 4.77	P < 0.01 highly significant

Table 4a: Quality of sensory blockade

Quality	Study group/ Group II	Control group/ Group I	Total
1	4	16	20
2	26	14	40
Total	30	30	60

$\chi^2 = 10.8$, P < 0.001, highly significant

Table 4 b: Quality of motor blockade

Quality	Study group/ Group II	Control group/ Group I	Total
0	21	14	35
1	0	1	1
2	6	10	16
3	3	5	8
Total	30	30	60

$\chi^2 = 2.5$, P > 0.05, not significant

Table 5: Adjuvant used

	Study group/ Group II	Control group/ Group I	Total
1	21	13	37
2	08	16	24
3	01	01	02
Total	30	30	63

$\chi^2 = 3.99$, P < 0.05 significant

Table 6a: Variations in heart rate, blood pressure respiratory rate arterial oxygen saturation at 5 mins

5 mins	Study group		Control group		t - test	P value significant
	No	Mean ± SD	No	Mean ± SD		
Heart rate	30	72.6±6.59	30	74.53±6.23	t = 1.16	P>0.05
Blood pressure systolic	30	116.46±9.24	30	117.6±8.61	t = 0.49	P>0.05
Blood pressure diastolic	30	78.8±6.89	30	80.67±4.62	t = 1.22	P >0.05
SPO ₂	30	100 ± 0.0	30	99.93 ± 0.3	t = 1.34	P >0.05
Respiratory rate	30	15.07±0.97	30	15.46 ± 1.14	t = 1.48	P > 0.05

Table 6b: Variations in heart rate, blood pressure respiratory rate arterial oxygen saturation at 15 mins

15 mins	Study group		Control group		t - test	P value significant
	No	Mean ± SD	No	Mean ± SD		
Heart rate	30	90.51±3.12	30	92.65±4.32	t = 2.25	P<0.05 (S)
Blood pressure systolic	30	139.4±5.3	30	141.86±6.12	t = 1.91	P<0.05 (S)
Blood pressure diastolic	30	85.26±5.7	30	88.34±5.48	t = 2.16	P<0.05 (S)
SPO ₂	30	99.83 ± 0.52	30	99.56 ± 0.98	t = 1.29	P>0.05 (NS)
Respiratory rate	30	18.0 ± 0.42	30	18.23 ± 0.61	t = 2.04	P<0.05 (S)

Table 6c: Variations in heart rate, blood pressure respiratory rate arterial oxygen saturation at 30 mins

30 mins	Study group		Control group		t - test	P value significant
	No	Mean ± SD	No	Mean ± SD		
Heart rate	30	87.8±7.14	30	86.13±11.3	t = 0.84	P>0.05 (NS)
Blood pressure systolic	30	118.66±100.4	30	122.26±11.5	t = 1.28	P>0.05 (NS)
Blood pressure diastolic	30	78.73±5.6	30	80.13±6.42	t = 1.18	P>0.05 (NS)
SPO ₂	30	99.5 ± 0.72	30	99.57 ± 0.91	t = 0.33	P>0.05 (NS)
Respiratory rate	30	17.4 ± 1.2	30	18.48 ± 3.09	t = 1.48	P>0.05 (NS)

Table 6d: Variations in heart rate, blood pressure respiratory rate arterial oxygen saturation at 45 mins

45 mins	Study group		Control group		t - test	P value significant
	No	Mean ± SD	No	Mean ± SD		
Heart rate	30	76.89±8.18	30	78.4±8.5	t = 0.71	P>0.05 (NS)
Blood pressure systolic	30	117.48±8.7	30	118.2±8.9	t = 0.34	P>0.05 (NS)
Blood pressure diastolic	30	78.73±3.67	30	77.43±4.32	t = 1.42	P>0.05 (NS)
SPO ₂	30	99.57 ± 0.72	30	99.53 ± 0.68	t = 0.24	P>0.05 (NS)
Respiratory rate	30	16.80 ± 2.5	30	17.33 ± 2.2	t = 0.88	P>0.05 (NS)

Discussion

Brachial plexus blockade for upper limb surgeries is the most common major peripheral nerve block technique. A significant difference exists between the onset times of various agents when these blocks are used. In general, agents of intermediate potency exhibit a more rapid onset than the more patient compounds do. Onset times of approximately 14 minutes for lidocaine and mepivacaine have been reported versus approximately 23 minutes for bupivacaine. The variation in duration of anesthesia after the plexus blockade is also considerably greater than that observed with other types of conduction block. For example: duration of anesthesia varying from 4 to 30 hrs have been reported for bupivacaine [2].

Potassium salts were first used as adjuvants to local anesthetics in 1912. They have been proved to enhance the onset of action and duration of the block.

The onset of blockade in group II (Potassium/study group) was earlier when compared to group I (control/plain bupivacaine group). In our study, the mean onset of sensory and motor blockade in potassium group II was 14.53 minutes and 17.7 minutes respectively. The results of our study support the findings of Khosa et al who showed that addition of potassium chloride to bupivacaine significantly enhanced the onset of the sensory and motor blockade [19].

In our study duration of sensory and motor blockade was significantly increased (P <0.001) in potassium group when compared with group I (plain bupivacaine group). The quality of sensory blockade was significantly better with potassium chloride when compared to other group. Bromage and Burfoot also found that quality of blockade was intense when potassium was added to lignocaine in epidural blockade [20].

But the quality of motor blockade with potassium chloride was the same that of plain bupivacaine group. This is in contrast to Bromage and Burfoot who found that quality of motor blockade was also

intense with potassium containing lignocaine when used for epidural blockade [20].

The requirement of adjuvants was decreased in potassium group when compared to other group this implies that better quality anesthesia was found with group II. This was accordance to finding by Parriss and Chambers [21].

In our study heart rate blood pressure, SPO₂ and respiratory rate were recorded at every 5 mins throughout the procedure. Variations in heart rate, blood pressure, SPO₂, and respiratory at 5 mins were statistically not significant between the two groups were as group II (potassium chloride group) showed lesser heart rate, blood pressure and respiratory rate at 15 mins implying that block was successful so lesser sympathetic stimulation and SPO₂ in both the groups were comparable. Variations in heart rate, blood pressure, SPO₂, and respiratory at 30 and 45 mins were statistically not significant between the two groups. No patient in our study developed in significant side effects.

Thus potassium chloride definitely has a role as an adjuvants to bupivacaine hydrochloride in enhancing they onset of blockade prolonging the duration of action and improving the quality of blockade in supraclavicular brachial plexus block.

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