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Comparative Study of Alkalined Lignocaine, Addition of Potassium Chloride to Lignocaine and Plain Lignocaine on Onset and Duration of Brachial Plexus Block By Supraclavicular Approach

Authors

Dhananjay Ambike[1], Narendra P L[2], D S Thoarat[3], Pushpa Agarwal[4], Maroof Ahmad Khan[5]

[1]MD, Associate Professor, Department of Anesthesiology, Dr D Y Patil Medical College Hospital and Research Centre, Pimpri Pune, 411018 Maharastra. India
Email: *varshadhananjay@gmail.com*

[2]MD, PDFC, FCARCSI (pri), MRCS-I, Assistant Professor, Dept of Anesthesiology, BLDE University s Shri B M Patil Medical College Hospital and Research Centre, BIJAPUR 586103, Karnataka. India
Email: *purohit72@gmail.com*

[3]MD, Professor (Retd), Department of Anesthesiology, Dr Vaishampayan Memorial Government Medical College Solapur 413003 Maharastra India
Email: *dsthorat@gmail.com*

[4]MD, Professor and Head, Department of Anesthesiology, Dr Vaishampayan Memorial Government Medical College Solapur 413003 Maharastra India
Email: *agrawalpushpa@yahoo.com*

[5]Assistant Professor, Dept of Biostatistics, All India Institute of Medical Sciences, Ansari Nagar New Delhi 110029
Email: *khanmaroofahmad@gmail.com*

Corresponding Author

Narendra P L

MD, PDCC, FCARCSI(pri), MRCS-I, Assistant Professor, Dept of Anesthesiology, BLDE University s Shri B M Patil Medical College Hospital and Research Centre , BIJAPUR 586103,Karnataka, India
Email: *purohit72@gmail.com*

Abstract

Background: *Several adjuvants have been used to hasten the onset and prolong the duration of analgesia during bracheal plexus block .In our study we evaluate clinically and co rrrelate the effect of addition of 2 ml of sodium bicarbonate and 0.2 mmol of potassium chloride to 1,5% lignocaine hydrochloride for supraclavicular bracheal plexus block*

Methods *75 ASA I and II patients posted for elective and emergency upper limb surgery were included and divided into three groups A receiving 30cc lignocaine 1.5% with adrenaline, B receiving 30 cc lignocaine 1.5% with adrenaline and 2 ml sodium bicarbonate and C receiving 30 cc lignocaine 1.5% with 0,2 mmol potassium chloride .The groups were assessed for the onset ,duration and intensity of analgesia.*

Statistical Analysis-*One way ANOVA and Tukey Post Hoc analysis*

Results: *The onset of motor blockade with lignocaine is earlier with lignocaine and sodium bicarbonate 4.10 ±0.9 mins v/s 9.1±1.5 and 9.2±1.9 mins There is no significant difference in the onset of motor blockade in the groups receiving Lignocaine and lignocaine and potassium chloride. The duration of both sensory 132 ±6.5 v/s 88 ±8.2 and 110±5.0 mins and motor blockade120±6.7 v/s 81±7.5 and 102±4.2 mins is longer in the group receiving lignocaine and potassium chloride .*

Keywords – *Adjuvant, duration ,brachial plexus block, potassium*

Introduction

Since the discovery of local anaesthetics in 1884, regional anaesthesia has been widely practised. A successful regional anaesthesia has advantages of simple techniques, preservation of protective reflexes, less stress response, prolonged post operative pain relief and valuable in patient with cardiac, pulmonary and renal disease. Brachial plexus is a common technique for surgery on upper limb. Disadvantages of regional techniques is their relative slow onset and limited duration of action. Various measures have been taken to hasten the onset and prolong the duration of local anaesthetics including compounding of local anaesthetics, addition of various adjuvants, addition of various adjuvants eg dextran, hyaluronidase, potassium, alkalinisation to adjust pH of solution [1,2,3,4,5].

We conducted a study to analyse the effect of addition of potassium chloride and sodium bicarbonate to lignocaine hydrochloride (1.5%) for brachial plexus block and to evaluate the effect on onset and duration of plexus block.

Objectives

To clinically correlate and analyse, effects of addition of Potassium chloride 6.67mmol/litre and 2 ml of Sodium Bicarbonate (7.4%) to 1.5% lignocaine HCl on onset of sensory block, onset of motor block, duration of surgical anaesthesia, intensity of sensory and motor block.

Materials and methods

After approval by Ethics Committee and informed consent, this study was undertaken in 75 patients of both sex scheduled for routine or emergency upper limb surgery. The criteria for sample selection was age 20-60 yrs, ASA I-II, co operative and both emergency and elective upper limb surgery.

Exclusion criteria – patients with serious illnesses like uncontrolled diabetes, cardiovascular, respiratory or renal disorder, severe liver disorder, history of hypersensitivity to local anaesthetics, neurological disturbances. epilepsy and patients receiving anticoagulants.

All patients were explained about the procedure and possible complications .Written informed consent was taken .It was proposed to study total of 75 patients divided into three groups of 25 patients depending on whether received lignocaine, lignocaine with sodabicarb or lignocaine with potassium chloride. Surgical consultation was obtained regarding surgical procedure and approximate duration of surgery. Equipment included 23G 1 ½ inch hypodermic needle 10 m0, 2 ml, 5 ml syringes, and equipment for sterility. Drug used lignocaine hydrochloride (1.5%) with adrenaline 1:200000,

Preparation of solutions

1. Lignocaine hydrochloride 1.5% with adrenaline 1:200000 -30 ml received by group A
2. Lignocaine hydrochloride 1.5% with adrenaline -30 ml with 2 ml of sodium bicarbonate 7.4% freshly added-Group B
3. Lignocaine hydrochloride 1.5% -30 ml with 0.2 mmol KCl (adding 0.1 ml of KCl) Group C

Each patient was thouroughly examined for cardiovascular, respiratory and central nervous system examination. Routine investigations like haemoglobin, bleeding time, clotting time urine

examination were performed in each case. Various parameters such as pulse, blood pressure, ECG, SPO2 were monitored intra operatively. All patients received 0.6 mg atropine IV prior to performing block.

Methods

A Positioning and Landmarks – Supine position with ipsilateral arm adducted, extended along ipsilateral knee as far as possible. A small pillow below the head with head turned to opposite side. In the classic technique, midpoint of clavicle was identified and marked .The palpating fingers roll over the belly of anterior scalene muscles into interscalene groove. A mark was made 1.5 -2 cm above the midpoint of clavicle and sublcvian artery is palpated.

Technique- After preparation, short bevelled 23G needle is introduced in a downward forward and slightly medial direction till paresthesia is elicited or first rib is encountered. 30 cc of drug is injected after elicitation of paraesthesia.

Time of onset of sensory block - Onset and spread of sensory and motor block was assesses every 2-3 min for 30 min after injecting local anaesthetic solution. Sensory loss was assessed by pinprick with a short bevelled 24G needle using a scale of 0-2, 0-sharp prick, 1- touch only, 2- cannot feel touch .The time to attain a score of 2 was taken as the time to onset of analgesia.

Time of onset of motor block- Motor block was assessed by scale of 0-3 (Paris and chambers - 1986) 0-able to move normally,1- inability to move wrist and elbow against resistance 2- inability to move wrist and elbow against gravity, 3- total inability to move arm. The time required to attain grade 3 was taken as time of onset of motor block.

Duration of sensory blockade –The time at complete loss of touch and pinprick was taken as 0 minutes. The time to return of pinprick sensation was taken as termination of analgesia. The difference these two gives the duration of sensory blockade.

Duration of motor blockade -The time at which there was total inability to move the arm was taken as O minutes. The time at which first sign of motor power in fingers was noted was taken as termination of motor blockade. The difference between these two gives duration of motor blockade.

Intensity of sensory and motor blockade –assessed by sensory and motor scores attained in each case.

Monitoring –SPO2, ECG and vital parameters. Patients were observed for signs and symptoms of local anaesthetic toxicity and pneumothorax Statistical analysis was done with one way ANOVA for three groups using online software tool statpages.org Washington DC USA

Observations and Results

Table 1 Sex Distribution

Group	A	B	C	Total
Male	13	15	11	39
Female	12	10	14	36
Total	25	25	25	75

Table 2 Age Distribution

Age /Group	A	B	C	Total
21-30 yrs	8	10	12	30
31-40 yrs	9	10	8	27
41-50	6	3	4	13
51-60	2	2	1	5
Total	25	25	25	25

Table 3

Group	Mean Age	SD
A	37	9.17
B	33.9	8.0
C	34.2	8.6

ANOVA F= 1.0213 p=0.3653
Tukey HSD Post-hoc Test...
Group A vs Group B: Diff=-3.1000, 95%CI=-8.8259 to 2.6259, p=0.4022
Group A vs Group C: Diff=-2.8000, 95%CI=-8.5259 to 2.9259, p=0.4747
Group B vs Group C: Diff=0.3000, 95%CI=-5.4259 to 6.0259, p=0.9914
It is seen that difference in mean age is not statistically significant

Table 4

Type of surgery	No of patients	%
Radius plating	04	5.32
Ulna plating	05	6.65
Radius nailing	07	9.31
Radius and ulna plating	12	16.00
Radius head excision	06	8.00
Ganglion excision	10	13.33
Tendon suturing	05	6.65
Colles Reduction	06	8.00
Olecrenon reduction	09	11.97
Amputation (below elbow)	07	9.31
External fixator application	04	5.33
TOTAL	75	100

Table 5 Mean time of onset of analgesia

Group	No of patients	Mean onset time	S.D.
A	25	9.10	±1.5
B	25	4.10	±0.9
C	25	9.25	±1.9

ANOVA F= 96.5986. p= 0.0000
Tukey HSD Post-hoc Test...
Group A vs Group B: Diff=-5.0000, 95%CI=-6.0093 to -3.9907, p=0.0000
Group A vs Group C: Diff=0.1500, 95%CI=-0.8593 to 1.1593, p=0.9327
Group B vs Group C : Diff=5.1500, 95%CI=4.1407 to 6.1593, p=0.0000 .
The mean time of onset of analgesia is significantly shorter than both group B and C .However the difference in mean onset of analgesia in Group C though slightly longer than Group A is not statistically significant

Table 6 Mean onset of motor blockade

Group	Mean onset time (mins)	S.D
A	10.10	±2.0
B	5.00	±1.5
C	11.00	±1.8

ANOVA F=82.7450 p=0.0000
Tukey HSD Post-hoc Test...
Group A vs Group B: Diff=-5.1000, 95%CI=-6.3039 to -3.8961, p=0.0000
Group A vs Group C: Diff=0.9000, 95%CI=-0.3039 to 2.1039, p=0.1805
Group B vs Group C: Diff=6.0000, 95%CI=4.7961 to 7.2039, p=0.0000

The mean time of onset of motor block is significantly shorter than Groups B and C .Mean onset time in group C is slightly longer than group A but not statistically significant.

Table 7 Duration of sensory blockade

Group	Time in minutes	S.D.
A	88	±8.2
B	110	±5.0
C	132	±6.5

ANOVA F 269.9085 p = 0.0000
Tukey HSD Post-hoc Test..
Group A vs Group B : Diff=22.0000, 95%CI=17.4679 to 26.5321, p=0.0000
GroupA vs Group C: Diff=44.0000, 95%CI=39.4679 to 48.5321, p=0.0088
Group B vs Group C: Diff=22.0000, 95%CI=17.4679 to 26.5321, p=0.0000
Duration of sensory blockade is longer than groups A and B .This difference is statitistically significant.

Table 8 Duration of motor blockade

Group	Mean Duration in mins	S D
A	81	±7.5
B	102	±4.2
C	120	±6.7

ANOVA F 240.5708 p =0.0000
Tukey HSD Post-hoc Test...
Group A vs Group B: Diff=21.0000, 95%CI=16.7409 to 25.2591, p=0.0000
Group A vs Group C: Diff=39.0000, 95%CI=34.7409 to 43.2591, p=0.0010
Group B vs GroupC3: Diff=18.0000, 95%CI=13.7409 to 22.2591, p=0.0000

The duration of motor blockade in Group C is longer than Groups A and B .This difference is statistically significant.

Table 9 Intensity of block

	Score			
Group	0	1	2	3
A	-	-	-	25
B	-	-	-	25
C	-	-	-	25

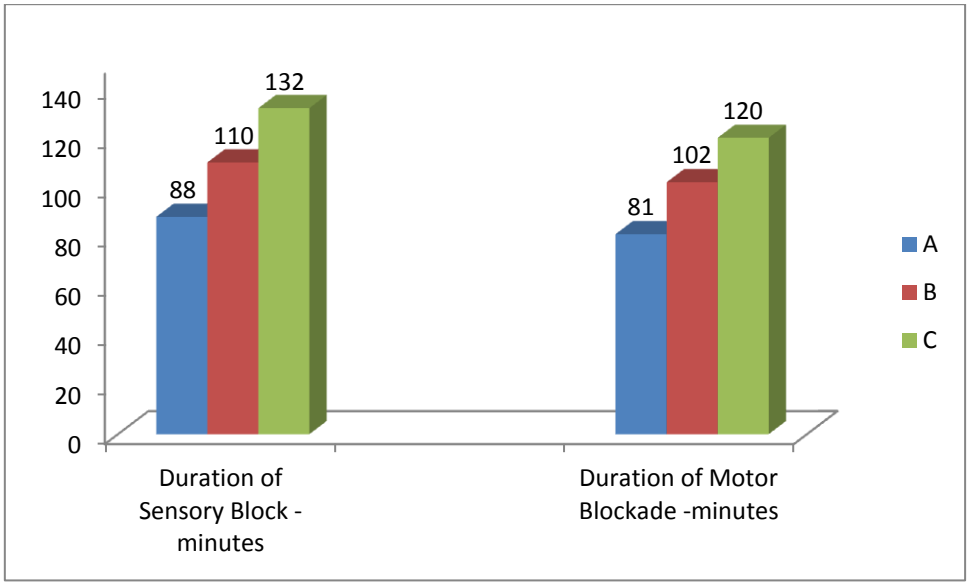
Table 10 Complications during surgery

Complication Group	Group A No of patients	Group B No of patients	Group C No of patients
Hypertension	-	-	-
Tachycardia	3	1	
Arrythmia	-	-	-
Dizziness	1	-	-
Pneumothorax	-	-	-

Table 11 p H of the solution

Group	Solution	p H
A	Lignocaine 1.5% with adrenaline (1:200000)	5.90
B	Lignocaine 1.5% with adrenaline 1:200000+ 2 ml sodium bicarbonate	6.72
C	Lignocaine 1.5% + Potassium chloride 0.2mmol	5.89

Fig 1 .Comparison of Duration of sensory and motor blockade between three groups.



Discussion

William Halsted in 1885 introduced the concept and use of nerve block and infiltration anaesthesia by injection of cocaine. One important aspect of regional anaesthesia relatively slow onset and limited duration of action. Various measures have been tried to improve the nerve blockade of local anaesthetics like use of rapidly acting agents like chlorprocaine, longer acting agents like etidocaine, bupivacaine, addition of hyaluronidase, addition of dextran enzymes, oils, glycols, vasoconstrictors, compounding of local anaesthetics, warming of local anaesthetic solution addition of clonidine, fentanyl, addition of potassium chloride and alkalinisation of local anaesthetic solution.[1,2,3,4,5,6,7 ,8,9,10,11,12]

Adrenaline is the commonest adjuvant used to prolong the duration of local anaesthetics. It acts by causing vasoconstriction and thereby delays the absorption of local anaesthetic from the site of injection. But it is associated with potentially toxic effects like tachycardia, arrhythmia and hypertension [11]. Hence a physiological adjuvant which can the duration of action holds tremendous appeal as a safe alternative to adrenaline.

We conducted this study to evaluate the effects of addition of potassium chloride and sodium bicarbonate to lignocaine 1.5% on its onset and duration of action.

Effects of addition of Sodium bicarbonate

In our study , the p H of lignocaine 1.5% with adrenaline was adjusted from 5.91 to 6.72 by the addition of 2 ml of sodium bicarbonate .Group B receiving alkalinised solution had a mean onset time of 4.10±0.9 mins as compared to control Group A which had a mean onset time of 9.10±1.5 mins. This difference is statistically significant p<0.01. Similar results have shown by Bromage et al et al by using carbonated salt of lignocaine as compared to hydrochloride salt of lignocaine. [13] DiFasio et al adjusted the p H of 1..5% lignocaine with adrenaline from 6 to 7 by addition of 2 mEq of sodium bicarbonate .The mean onset time of alkalized solution was 2.68 mins and was significantly shorter than the lignocaine with adrenaline group.[3]

Sukhani Segura and Winnie obtained a faster onset of brachial plexus blockade using lignocaine 1.1% solutions with pH in the alkaline range. They postulated that a higher p H required less buffering by tissues which facilitates the liberation of free base which diffuses rapildly into nerve fibres.[14]. Capoga G has shown that concerning brachial plexua block, effects of alkalization were more evident with lidocaine .[15]

Duration of Analgesia

The duration of analgesia (sensory block) in the group receiving alkalised lignocaine Group B was 110±5.0 mins and motor block of 102±4.2 mins as compared to the group lignocaine with adrenaline Group A 88±8.2 mins and 81±7.5mins respectively. This difference is statistically significant p<0.01. Thus alkalization prolongs the duration of action of lignocaine. Hinger found alkalinisation of bupivacaine resulted in a significant prolongation of duration of action. Mc Morland G H Douglas M J using pH adjusted bupivacaine in parturients observed that duration of action was significantly prolonged from 79.4 mins in the control group to 96.5 mins in the alkalised group.[16] Similar results were obtained other studies.[2,10]

Intensity or quality of blocakde

Another mechanism that could explain favourable effect of fresh alkalinisation on quality of motor block acquired is size of fibres involved in motor transmission. The larger the nerve fibre, higher the concentration of local anaesthtic required to interrupt transmission of conduction. During the finite interval after injection, when in vivo buffering occurs with plain solutions, diffusion and admixture with extracellular fluid occurs. Alkalinised solutions would become intracellular at higher concentration and theoretically this would result in an improved block. Rapidity of action with carbonated solution can be used to diagnose failure to block any one particular nerve at a much earlier time and to start supplementary blocking procedures. There was no need to wait for long time as was often when hydrochloride salts of local anaesthetic were used. Another possible advantage when alkaliseed solutions are used is the increased analgesic potency of block – without increased plasma concentration of local anaesthetic .[15]

Mode of action

An increase in pH leading to an increase in the unchanged lipid soluble base form. This readily penetrates the nerve and in a relatively acidic intraneural environment is converted to the active form. This perhaps is the mechanism for the increased potency of alkalised solution.

Effects of addition of KCl

The time to onset of analgesia in the group receiving lignocaine +KCl combination (Group C) was 9.25 ±1.5 mins .This slightly slower than mean onset time of 9.1 ±1.5 mins in the group receiving lignocaine with adrenaline (Group A).This difference in mean onset time however is statistically insignificant. p>0.01. These findings are consistent with other studies.[5, 17] Our results however differ from those of Bromage and Burfoot and Paris et al who reported faster onset of analgesia.[17,18]

The study group C receiving lignocaine + KCl had a mean duration of analgesia (sensory block) of 132 ±6.5 mins and motor block of 120±6.7mins. This was significantly longer than 88 ±8.2 mins and81±7.5 mins of the control Group A receiving Lignocaine +Adrenaline p<0.01.This is in accordance with the results obtained by other workers.[17] These observations can be explained by the mode of action local anaesthetics and the changes in the ionic milieu during resting and active membrane potential. Huxley and Stampfli have shown A nerve impulse can be effectively blocked by the accumulation of potassium ions outside the neuron .From this experimental concept, it is assumed that the blockade produced by increased K+ concentration outside the nerve membrane [19]. Successive equal increments in the extracellular potassium will have a geometrically decreasing effect on the membrane potential due to an altered Donnan equilibrium - Em α log (K+)O/log (K+) l where Membrane potential, K+ I Intracellular potassium concentration, K+O extracellular potassium concentration. The membrane Em will decrease as the extracellular potassium K+O increases[5] .Also inhibition of ionic fluxes across the membrane may play a part[20]. High concentrations of extracellular potassium efflux from the cell and the altered ionic relationships may affect the orientation of lipoproteins in the cell membrane in such a way that the membrane lattice becomes less permeable to lipid soluble substances such as local anaesthetics. Also Swetha et al reported a significant prolongation of sensory block of the order of 205 mins and motor blockade of 467 mins with bupivacaine and potassium chloride 0.2 mmol. However in our study, adding potassium to Lignocaine rather than Bupivacaine prolongs the duration of sensory blockade of order of 132 mins for sensory blockade and motor blockade of 120 mins which suits most upper limb surgeries with to unique and without unduly prolonging the motor blockade in the post operative period.

Complications /Side effects - The expected toxic reactions due to Lignocaine and Potassium chloride were arrhythmias, convulsions, tremors, palpitations. These were not observed in our study. Inadvertent intravascular injection of the drug was prevented by careful aspiration before drug injection and careful and continuous monitoring of vital parameters .Thus from this study it can be said that alkalinisation of local by the addition of sodium bicarbonate is an effective way to enhance the onset time of the commercially available local anaesthetic solutions. Physiological concentrations of potassium can be safely used prolong the duration of local anaesthetics.

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