IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 15, Issue 12 Ver. IX (December. 2016), PP 47-51
www.iosrjournals.org

Comparitive Study of The Effect of Lignocaine Hydrochloride With Adrenaline And Its Combination With Alkali Solution on Supraclavicular Brachial Plexus Block.

Dr.P.Chalapathy[1],Dr. D.Sangeetha Rao[2]
[1](Department Of Anaesthesiology, RIMS Medical College, Ongole/Dr.NTR University Of Health Sciences, India)
[2](Department Of Anaesthesiology, RIMS Medical College, Ongole/Dr.NTR University Of Health Sciences, India)

Abstract: *Alkalinization of 1.5% lignocaine hydrochloride with adrenaline leads to alternation in its onset of action and duration of action.60 patients requiring upper limb surgery between the age group of 20-60 years of either sex, falling under ASA grade I and II and were randomly allocated into two groups. Group A received 32ml of lignocaine hydrochloride with adrenaline (1:200000) solution (pH=4.37).Group B received 32ml of alkalinised standard solution of lignocaine hydrochloride with adrenaline (1:200000) prepared by addition of 2ml 7.5% (w/v) sodium bicarbonate (pH=7.24). Supraclavicular brachial plexus block technique was used for administration of drug. Onset of sensory block, onset of motor block, quality of block and duration of block and duration of analgesia were assessed using standard parameters. It was observed that raising the pH of solution from 4.37 to 7.24 produced a reduction in latency of sensory block (13.09±2.83 to 6.32±1.68 minutes), a reduction of latency of motor block (22.62±5.9 to 10.89±2.05 minutes) and increase in the duration of block and duration of analgesia. Alakalinization of lignocaine with adrenaline increased the frequency of complete block from 63% to 93%.*

Keywords: *Adrenaline, Alkalinization, Brachial plexus block, Lignocaine hydro chloride, Sodium bicarbonate 7.5*

I. Introduction

Brachial plexus block is preferentially used technique for anaesthesia of upper limb due to its easy accessibility and simplicity with predictable land marks. Prolonged latency following brachial plexus anaesthesia presents a potential problem in the busy clinical setting. Efforts to improve onset time have centered on the choice and modification of local anaesthetic solutions.

Alkalinization has been shown to improve neural blockade in animal models[1], and research involving alkalinization of different local anaesthetic solutions has been undertaken in various clinical settings in man: brachial plexus [2-7], epidural anaesthesia [8-10], and various others [11,12]. The results produced have been conflicting, ranging from no effect to improvement in both onset and duration of anaesthesia. Although the pharmacology is well understood, the topic remains one of controversy. Technique, agent specificity, the addition of adrenaline, and the degree of change in pH have been suggested as possible explanations [1, 7].

The choice of local anaesthetic agent is also important in determining the latency of neural blockade. Traditionally, lignocaine has been used during brachial plexus anaesthesia to produce a relatively rapid onset time. However, the duration of anaesthesia is often less than with other local anaesthetic agents. This study has aimed to investigate the effect of alkalinization on latency, quality and duration of anaesthesia.

II. Materials And Methods

The present study was conducted in the department of anaesthesiology, Rajiv Gandhi Institute of Medical Sciences, Ongole, Prakasam District. After approval from the institutional ethical committee and informed and written consent were taken from patients. 60 patients who required upper limb surgery were selected for this study. The age of patients ranged from 20 to 60 years of either sex. Prior to selection a thorough pre-anaesthetic clinical evaluation was carried out and only ASA grade I and II patients were included in this study. Exclusion criteria included progressive neurological disorder, severe liver or kidney disease, patients with bilateral upper limb surgery or any history of previous adverse reaction to local anaesthetic drug. Patient who weighed less than 50 kg or more than 70kg were also excluded from the study as a fixed amount of local anaesthetic was used. Patients having contralateral pneumothorax or collapsed or partially collapsed lungs were also excluded from the study.

The patients were randomly allocated into two groups A and B according to the drug received. For Each Group 30 patients were allotted .Group A patients received 32 ml of 1.5% lignocaine hydrochloride with adrenaline (30 ml lignocaine with adrenaline (1:200000)(pH=4.37) +2ml distilled water) and Group B patients received 32ml of freshly prepared alkalinised solution of lignocaine hydro chloride with adrenaline (1:200000) (30 ml of 1.5% lignocaine hydrochloride with adrenaline+2ml of 7.5% sodium bicarbonate(w/v) (pH=7.24)). Alkalinization of solution was done just prio to use by adding 2 ml of sodium bicarbonate 7.5%(w/v) to 30 ml of lignocaine hydrochloride with adrenaline. Mixture was inverted, without shaking, 30 times over a period of 45to 60 seconds. pH of the solution was estimated by an electronic pH meter.

Brachial plexus block was given by conventional supraclavicular approach. Onset of sensory and motor block were tested at every one minute interval for a maximum of 30 minutes. Onset of sensory block was determined by loss of pinprick sensation. Onset of motor block was judged by loss of finger movements. Latency of sensory blockade is calculated from the time of injection to loss of pinprick sensation. Latency in motor blockade is determined from the time of injection to loss of finger movements. Duration of blockade is calculated from loss of pinprick to reappearance of pinprick sensation. Duration of motor block being the time taken from loss of finger movements till the patients started moving their fingers. Duration of analgesia is calculated from loss of pinprick sensation to first requirement of analgesia by the patient in the post operative ward. Quality of block was assessed by the time taken to achieve sensory and motor block. When all the parameters were achieved within 30 minutes it was a complete block. When any of the parameters could not be achieved except pinprick within 30 minutes, it was incomplete and failed when none of the parameters were achieved.

Routine monitoring of patients was done both preoperatively, after injection and postoperatively. All patients were kept under observation for 24 hours.

3. Observations and Results

Data is analyzed and tabulated and subjected to statistical analysis by using student "t" test (two tailed distribution and unpaired) to find the significance between two groups and tabulated below (level of significance $t>2$ and $p< 0.05$).

Table-1: Age and sex distribution of the patients

Age (in years)	No. of patients					
	Group A			Group B		
	M	F	Total	M	F	Total
20-30	8	6	14	4	3	7
31-40	4	1	5	7	2	9
41-50	3	1	4	4	4	8
51-60	5	2	7	4	2	6
Total	20	10	30	19	11	30

Table-2: Distribution of the patients according to the surgical procedures

Surgical procedures	Group A	Group B
Radial plating	3	6
Radial head excision	1	1
Ulnar nailing	3	6
Tension band wiring of elbow	1	-
Dynamic compression plating of humerus	4	3
Radial plating and ulnar nailing	4	2
Debridement	3	3
Amputation	3	-
Excision of swelling	2	4
Tendon repair	2	1
Post traumatic contracture release and split skin grafting	2	2
K wire fixation	2	2
Total	30	30

Table-3: Onset of block (minutes)				
Parameters	**Group A**	**Group B**	**t value**	**p value**
Sensory block (Loss of pinprick sensation)	13.09±2.83	6.32±1.68	11.2	<0.001
Motor block (Loss of finger movements)	22.62±5.91	10.89±2.05	10.27	< 0.001

Table-4: Duration of block (minutes)				
Parameters	**Group A**	**Group B**	**t value**	**p value**
Sensory block	95.43±34.19	125.16±27.75	3.7	<0.001
Motor block	103.50±33.31	134.96±29.83	3.85	<0.001
Analgesia	128.76±38.01	187.83±39.34	5.86	<0.001

Table-5: Quality of block							
Group	**Complete**	**%**	**Incomplete**	**%**	**Failure**	**%**	**Total**
A	19	63%	10	33%	1	1%	30
B	28	93%	2	7%	0	0%	30
Total	47		12		1		60

III. Discussion

The present study is an attempt to compare relative efficacy of lignocaine hydrochloride with different pH values with regard to Brachial plexus block by supraclavicular approach. A local anaesthetic is a drug which reversibly blocks the transmission of peripheral nerve impulses. Local anaesthetics are usually injected as acidic solutions of the hydrochloride salt (pH<5). In this form amine group is ionized and the drug becomes soluble in water and therefore suitable for injection . After injection tissue buffering raises the pH and a percentage of the drug dissociates to become free bases, the amount depending upon 'dissociation constant' of the individual drug .Being lipid soluble, the free base is able to penetrate both the nerve coverings and the lipid cell membrane to reach the interior of the axon where a portion re-ionizes. The re- ionized portion enters the sodium channels and plug s these channels so that sodium ions cannot enter the cell. As a result action potentials are neither generated nor propagated and conduction block occurs.

On the other hand alkalinization (carbonation) of lignocaine hydrochloride lead to higher pH>6 and thus it is less depending on tissue buffering capacity of the tissues (pH=7.4). On injection of this alkalinized solution the free base liberated, carbondioxide rapidly diffuses into the axon interior and the pH falls ,which forces dissociation of local anaesthetic to the active cationic form .This effect results in 'ion-traping' further favouring the rapid movement of the local anaesthetic into the axon.

Catchlov et al[13] demonstrated in vitro studies that the exposure of axons to equal amounts of carbonate or hydrochloride lignocaine ,resulted in a tenfold increase in the degree of block for the carbonate salt compared to the hydrochloride salt .The effect of carbondioxide on local anaesthetic action in vitro depends on these probable mechanisms.

1) Axonplasmic acidification with ion traping of local anaesthetic
2) A general reduction in the safety margin for nerve impulse conduction induced by carbondioxide
3) Species specific increase in sodium channel binding by local anaesthetic

Usually among all the sensory modalities of temperature, touch, pinprick ,pressure sensation the first sensory parameter to be lost was temperature which was followed by touch,pinprick and lastly pressure sensation. In the present study we took only pin prick sensation for estimating the latency of sensory block . In our study onset of sensory and motor block in group B was earlier compared to group A. Duration of sensory, motor and analgesia was prolonged in group B compared to group A.

The 50.25% reduction in latency of sensory block observed in our study with group B is almost nearer to 45% reduction noted in the study done by Radha Sukhani et al [14]. In their study they compared 1% lidocaine hydrochloride with 1.1% lidocaine carbonate. The reduction in the onset of time they attributed was due to greater rapidity of spread, tissue penetration and intraneural diffusion more with the carbonate than with hydrochloride salt. In our study we used 1.5% lignocaine hydrochloride solution with pH 7.24 which was near to physiologic pH and reduced the buffering pH period of the tissues. So, this may be the reason for 50% reduction in latency in sensory block. Schulte-Steinberg et al[15] when in their study compared 1.73% carbonated lignocaine (pH=6.5) with 2% lignocaine hydro chloride (pH=4) they found latency for complete analgesia to be averaged 4-5 minutes for carbonated solutions and 4-20 minutes for hydrochloride solutions. Gormly et al[16] demonstrated in their study using 1.5% lignocaine with adrenaline (pH=4.2) and alkalinised

1.5% lidocaine with adrenaline (pH=7.2) on axillary plexus block that there was a significant reduction in time to useful anaesthesia and reduced requirements for the adjuvents in the alkalinised group.

In our study onset of motor block is significantly reduced from 22.62±5.91 to 10.89±2.05. these findings are similar to the study of Radha Sukhani et al[14] who observed that the onset of paralysis was significantly faster with the carbonated lidocaine(pH=6.8) than with the hydrochloride (pH=6.5). Quinlan et al[7]while working with alkalinized Mepivacaine on axillary block concluded that no significant difference in time to onset of paresis was noted but it significantly shortened the time to onset of both proximal and distal paralysis. Chow et al[17] showed in their study that alkalinized lidocaine(pH=7.15) did not improve the onset of motor block after axillary brachial plexus anaesthesia.

Quality of block was graded into three-complete, incomplete and failed. In present study 63% and 93% cases had complete block respectively in group A and B. incomplete block was seen in present study to be 33% in group A and 7% in group B. Schulte-Steinberg et al[15] found 44% patients had a complete block when carbonated solution of lignocaine was used(pH=6.5) but only 36.3% had such blockade when hydro chloride solution was used (pH=4). Chow et al[17] compared lidocaine with adrenaline solution (pH=6.24) with alkalinised lidocaine with adrenaline solution (pH=7.15) for axillary plexes. They concluded that difference in the overall success did not reach statistical significance between the two groups.

In the present study mean duration of sensory blockade in group A cases was 95.43±34.19 minutes and in group B it was 125.16±27.75 min. Mean duration of motor block in group A was 103.50±33.31 in minutes and in group B it was 134.96±29.83 minutes. Mean duration of analgesia in group A was 128.76±38.01 and in group B it was 187.83±39.34. Our study demonstrated that alkalinization of lignocaine hydrochloride improved the duration of sensory, motor and duration of analgesia significantly (p value< 0.001).

Ririe DG et al[18] performed median nerve blocks on 10 volunteers to compare the efficacy of 1% plain lidocaine with 1% lidocaine mixed with sodium bicarbonate o.1mmol/lit. their data suggested that addition of bicarbonate to lidocaine for median nerve block significantly increased the rate of motor block. Similarly Difazio et al[8] compared pH adjusted lidocaine solution for epidural anaesthesia and demonstrated that the degree of improvement in time to onset and duration is directly related to extend of difference in the pH of the solutions.

IV. Complications

Out of sixty patients in our study arterial puncture was encountered in 5 patients and tachycardia in 3 patients. We did not encounter any case of pneumothorax, phrenic nerve palsy and toxicity of drug.

V. Conclusion

In conclusion we can say that raising the pH of 1.5% lignocaine hydrochloride with adrenaline solution from 4.37 to 7.24 produced a definite reduction in the latency of sensory as well as motor block. It was also observed that alkalinization of 1.5% lignocaine hydrochloride improved the duration of motor and sensory block. Also greater frequency of complete block was seen on increasing the pH from 4.37 to 7.24. Duration of analgesia also significantly increased in alkalinized lignocaine hydrochloride group.

Therefore alkalinized lignocaine hydrochloride solution provides a significant advantage over non alkalinized lignocaine hydrochloride in terms of quicker onset, duration and quality of block.

References

[1]. Strobel GE, Bianchi CP. The effects of pH gradients on the action of procaine and lidocaine in intact and desheathed sciatic nerves. The Journal of Pharmacology and Experimental Therapeutics 1970; 1-17.

[2]. Hglier M.Alkalinization of bupivacaine for brachial plexus block. Regional Anaesthesia 1985; 10; 59-61.

[3]. Bedder MD,Koozdy R, Craig DB.Comparision of bupivacaine and alkalinized bupivacaine in brachial plexus anaesthesia. Anaesthesia and Analgesia 1988;67: 48-52.

[4]. Berman S, Rosenquist RW, Finucane BT. Alkalinization of mepivacaine for axillary brachial plexus block . Canadian journal of Anaesthesia 1988;35(suppl); 93.

[5]. Tetzlaff JE, Yoon HJ, O'hara J, Reaney J, Stein D ,Grimes-Rice M. Alkalinization of mepivacaine accelerates onset of interscalene block for shoulder surgery.Regional anaesthesia 1990; 15; 242-4

[6]. Buttner J, Klose R. Alkalisierung von mepivacain zuraxillaren katheterplexunaesthesie.Regional A

[7]. naesthesia 1991; 14 : 17-24.

[8]. Quinlan JJ, Oleksey K, MURPHY FL. Alkalinization of mepivacaine for axillary block . Anaesthesia and Analgesia 1992; 74: 371-4

[9]. Difazio CA, Carron H, Grosslight KR , MOscicki JC, Bolding WR, John RA . Comparision of pH-adjusted solutions for epidural anaesthesia. Anaesthesia and Analgesia 1986; 65: 760-4

[10]. Benzon HT, Toleikis JR, Dixit P, Goodman I, Hill JA. Onset, intensity of blockade and somatosensory evoked potential changes of the lumbosacral dermatomes after epidural anaesthesia with alkalinized lidocain. Anaesthesia and Analgesia 1993; 76: 328-32.

[11]. Capogna G, Celleno D, Costantino P , Mratori F, Sebastiani M. Alkalinization improves the quality of lidocaine fentanyl epidural anaesthesia for caesarean section . Canadian journal of Anaesthesia 1993; 40: 425-30

[12]. Mericq O, Guerot A, Boussaton E, Rigaud J ,Colombani A ,Pech C,Sans P ,Lareng L , Gay R ,Mansat M. Alkalinization de la bupivacain hyper bare en rachianesthesie. Cahier d' Anaesthsiogie 1988; 36: 371-4.
[13]. Cventry DM ,Todd JG . Alkalinization of bupivacaine for sciatic nerve blockade. Anaesthesia 1988;36: 271-4.
[14]. Catchlov, Richard FH: The influence of CO2 and pH on local anaesthetic actions .J pharmacol. Exp. Ther. 1972; 181:298.
[15]. Sukhani Radha and Winnie AP : Clinical pharmacokinetics of carbonated local anaesthetics I :Subclavian perivascular brachial block model. Anaesthesia Analg 1987; 66: 739-45.
[16]. Schulte-Steinberg, Hartmuth J, Schutt L : Carbon dioxide salts of lignocaine in brachial plexus block. Anaesthesia; 1970; 25(2): 191.
[17]. AV Gormley WF, Hill DA, Murray JM, Fee JP : The effect of alkalinization of lignocaine on axillary brachial plexus anaesthesia. Anaesthesia 1996; 51(2): 185-88.
[18]. Mark Y.H. Chow, Alex T.H. Sia, C.K. Koay, Y.W. Chan : Alkalinization of lidocaine does not hasten the onset of axillary brachial plexus block. Anaesth Analg 1998; 86: 566-68.
[19]. Ririe DG, Walker FO, James RL, Butterworth J : Effect of alkalinization of lidocaine on median nerve block. Br J Anaesth 2000; 84(2): 163-