Prolongation Of Action Of Lignocaine By Addition Of Dextran 40 In Brachial Plexus Block. A Randomised Controlled Trial

R Puri, A Saxena, D Kumar, T Chand, A Agarwal, A Mittal, A Mittal, M Sarkar, A Gupta, V Badada, N Mittal

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Abstract

Adjuncts to local anesthetics for peripheral plexus blockade have been proposed to enhance the quality and duration of anesthesia and postoperative analgesia. The effect of lignocaine can be prolonged by adding adrenaline, by alkalinization of solution, by adding opioids like fentanyl and also by addition of alpha2 adrenergic agonist. Lignocaine has been found to be a drug with very short latency period. Dextrans are complex, branched polysaccharides consisting of chains of individual glucose units, and are synthesised from sucrose by the action of certain lactic-acid bacteria. It is hypothesised that dextrans may form water-soluble complexes with LAs that remain at the site of injection longer than the unbound drug, dextran also alters the pH of the injected LA solution, and this alkalinisation may contribute to the prolongation of action8. The present study is designed to asses the prolongation of action of lignocaine by addition of dextran 40 in brachial plexus block.

INTRODUCTION

Brachial plexus block may be more advantageous for routine as well as emergency surgery in upper limb. Brachial plexus comprises cervical roots of C5,6,7,8 and T1 nerves. These supply the entire motor and almost the entire sensory nerve supply of the upper limb. Brachial plexus block can be performed with any local anaesthetic drug like bupivacaine, lignocaine etc. But most commonly used 15-20ml of 1-2% lignocaine with or without adrenaline in the nerve trunk forming brachial plexus either by supraclavicular or axillary approach. It provides good analgesia with good surgical condition for approximate one hour duration. In our study we used dextran 40 with lignocaine to assess the efficacy, safety and duration of brachial plexus block.

Lignocaine is a tertiary amide, a derivative of the acetanilide synthetic nitrogenous compound. This drug is used for spinal anaesthesia, epidural anaesthesia, and peripheral nerve block, as well as topical and infiltrative anaesthesia. It is excreted through kidney. The maximum safest dose of lignocaine is 5mg/kg body wt. without adrenaline and 7mg/kg body wt. with adrenaline. The middle trunk of the brachial plexus (C7) represented by the palmer distribution of the median nerve was found to be slow to develop analgesia1. In supraclavicular brachial plexus blockade lignocaine 1% with adrenaline2, found a mean total latency of 14.07 ± 3.76 minutes in major nerve blocks.

The addition of dextran as an adjunct to LAs was first studied in 1960 in an attempt to prolong the duration of action of the sensory blockade. Since that time there have been conflicting results from numerous other studies, with some demonstrating a prolongation of analgesia3,4 while others have been unable to show any conclusive benefit5,6. The mechanism of action is unclear, but it is hypothesised that dextrans may form water-soluble complexes with LAs that remain at the site of injection longer than the unbound drug, due to an increase in viscosity with reduced diffusion of the complex7. Alternatively, the addition of dextran alters the pH of the injected LA solution, and this alkalinisation may contribute to the prolongation of action8. Dextrans have been shown to decrease the systemic absorption of lidocaine, offering benefits in decreasing the incidence of toxicity. The present study is designed to asses the prolongation of action of lignocaine by addition of dextran 40 in brachial plexus block.

AIMS OF STUDY

To see the efficacy and safety of brachial plexus block after injecting lignocaine with dextran 40

1. Onset and duration of analgesia produced by lignocaine with Dextran 40 in brachial plexus block
2. Degree of sensory and motor block achieved
3. Incidence of side effect and complication
4. Failure rates

MATERIAL AND METHODS

This study was conducted after the approval of the institutional ethical committee. It was carried out on 60 patients of either sex, ASA grade I-II between 15–65 years undergoing upper limb surgery and the supraclavicular brachial plexus block was given. Written informed consent was taken from the patients before their selection for the study. The patients were randomly divided in two groups (n=30). The local anaesthetic drug used 15 ml of 2% lignocaine hydrochloride with 5ml of normal saline (Group I) or 15 ml of 2 % lignocaine hydrochloride with 5 ml of dextran 40 (Group II). The patient having neurological signs and symptoms other than those due to trauma were excluded from this study.

A proper history along with detailed physical examination was done. The procedure of the brachial plexus block was explained to the patient. Sensitivity test for local anaesthetic agent was done. Intravenous access was established in the nonoperative upper limb and standard monitoring was attached (noninvasive blood pressure, electrocardiography, and pulseoximetry).

The patient was made to lie with a pillow longitudinally between the shoulder blades in line with the spine. The arm in which the block is to be given is kept at the side. The shoulder of the side to be blocked was depressed and the head turned to the opposite side. The patient was then asked to reach for his knee with the hand on the side to be blocked, in order to lower the shoulder. After scrubbing up and wearing gown and gloves, the supraclavicular region of the patient prepared with savlon, sprit and iodine. The area was then properly draped. The patient was held by an assistant in the position mentioned before. The patient was then instructed to say ‘yes’ and not to move as soon as he felt tingling, shooting or burning pain (like electric shock), radiating down the arm or hand. He was also advised not to the move during the procedure. The subclavian artery palpated with the thumb and then the artery is retracted downwards and medially. Skin weal was raised with 24 gauge needle and local anaesthetic injected at a point immediately lateral to the finger palpating the subclavian artery.

Then a 22 gauge needle attached with the 10 cc syringe containing local anaesthetic was inserted through the skin wheal close to the tip of the finger. The needle was then advanced gently and gradually downwards, backwards and slightly medially, towards the first rib. In case the patient reported the parasthesiae as instructed to him, the needle was fixed at that point. The aspiration test was done for blood to avoid the intravascular injection of drug. The required volume of the drug was injected at this point in all the directions by rotating the needle. The area massaged to enhance the spread of the analgesic solution in the sheath of the plexus. Analgesia was assessed by pin prick using a 22 gauge hypodermic needle every 2 minutes and compared with the corresponding areas of the other arm. Pulse and blood pressure recorded preoperatively and immediately after giving the block. Thereafter pulse and blood pressure were recorded every 10 min during the operation and properly recorded till the effect of local anaesthetic drug weaned off completely..

The time of onset of analgesia were noted. The active movement of hand and arm were sought for, to asses the motor function and recorded. The regression of block was similarly observed till complete recovery. Side effects and complication during injection, during operation and postoperatively were properly recorded and treated accordingly.

RESULTS

In our study 60 surgical and orthopaedic cases of upper limb surgery, both elective as well as emergency, were conducted under supraclavicular brachial plexus block. The following observation and result were noted.

Table 1

Operative Procedures

S. No.	Operative Procedure	No. Of Patients			
		Group I		Group II	
		No.	%	No.	%
1.	Reduction of the colles fracture	6	20	6	20
2.	Square nailing of radius & ulna	3	10	6	20
3.	Debridement of the fore arm & hand	6	20	3	10
4.	External fixation of the fingers and thumb	3	10	6	20
5.	K – wire fixation of the fracture radius and ulna & thumb	6	20	6	20
6.	Tension band wiring of the olecranon fracture	6	20	3	10
Total		30		30	

Table 2

Patient measurements

		Group I	Group II	P value	Inference
		Mean ± SD	Mean ± SD		
Age; years		36.3 ± 10.54	33.25 ± 9.52		
Mean pulse rate		80±1.73 (64-100)	80.33±2.35 (66-100)	>0.05	Insignificant
Change in blood pressure	SBP	130.66 ± 4.55	128.5 ± 4.51	>0.05	Insignificant
	DBP	80.33 ± 3.53	83.83 ± 3.60		

Table 3

Time of onset of sensory block

Time (in minutes)	No. Of Cases			
	Group I		Group II	
	No.	%	No.	%
0 – 5	1	3	0	0
6 – 10	6	20	1	4
11 – 15	15	50	4	14
16 – 20	3	10	8	27
21 – 25	3	10	10	34
26 – 30	2	7	7	21
Total Cases	27+3*	100	28+2*	100
Mean	14.15		21.00	
S. D.	3.78		4.49	
T	2.61		6.39	
P	<0.0001		Extremely significant	

*Cases of patchy / partial analgesia and failures.

The comparison of mean time in min for onset of analgesia was 14.15 ± 3.78 in group I and 21 ± 4.49 in group II were statistically significant. As seen 60% of cases in group I have onset time between 11-20 min and 80% cases in group II have onset between 16-30 min.

Table 4

Time of onset motor block

Time (in minutes)	No. Of Cases			
	Group I		Group II	
	No.	%	No.	%
0 – 5	0	0	0	0
6 – 10	1	4	1	3
11 – 15	4	14	2	6
16 – 20	8	27	1	3
21 – 25	10	34	3	10
26 – 30	7	21	8	27
31 – 35	0	0	8	27
36 – 40	0	0	7	24
Total Cases	27+3*	100	28+2*	100
Mean	21.00		29.16	
S. D.	5.88		7.14	
T	0.05		4.832	
P	<0.0001		Extremely significant	

*Cases of patchy / partial analgesia and failures.

The comparision of mean time in min for onset of motor

block was 21 ± 5.88 in group I and 29.16 ± 7.14 in group II were statistically significant. As seen 75% of cases in group I have onset time between 11-25 min and 88% cases in group II have onset between 16-40 min.

Table 5

Time of regression of sensory block

Time (in minutes)	No. Of Cases			
	Group I		Group II	
	No.	%	No.	%
25 – 50	2	6	1	4
51 – 75	2	7	2	6
76 – 100	3	10	1	4
101 – 125	6	20	1	4
126 – 150	3	10	6	20
151 – 175	9	30	10	32
176 – 200	5	17	9	30
201 - 225	0	0	0	0
Total Cases	30	100	28+2*	100
Mean	132.13		150.48	
S. D.	29.81		31.31	
T	=		2.325	
P	= 0.236		Considered significant	

*Cases of patchy / partial analgesia and failures.

The mean regression time in min for sensory block was 132 ± 29.81 in group I and 150.48 ± 31.31 in group II.

Table 6

Time of regression of motor block

Time (in minutes)	No. Of Cases			
	Group I		Group II	
	No.	%	No.	%
25 – 50	2	7	1	3
51 – 75	3	10	3	10
76 – 100	10	33	4	14
101 – 125	8	27	6	20
126 – 150	3	10	3	10
151 – 175	1	3	8	26
176 – 200	3	10	5	17
201 - 225	0	0	0	0
Total Cases	30	100	28+2*	100
Mean	106.3		130.48	
S. D.	31.21		29.72	
T	= 3.073			
P	= 0.0032		Very significant	

*Cases of patchy / partial analgesia and failures.

The comparison of mean regression time n min for motor block was 106 ± 31.21 in group I and 130.48 ± 29.72 in group II were statistically very significant.

Table 7

Time of complete recovery of sensations

Time (in minutes)	No. Of Cases			
	Group I		Group II	
	No.	%	No.	%
100 – 150	5	17	4	13
151 – 200	15	50	14	47
201 – 250	9	30	8	27
251 – 300	0	0	0	
301 – 350	0	0	0	
351 – 400	0	0	0	
401 – 450	1	3	3	10
451 – 500	0	0	1	3
Total Cases	30	100	28+2*	100
Mean	190.41		217.1	
S. D.	82.05		73.19	
T	= 3.073			
P	= 0.0032		Very significant	

The comparison of mean time in min of complete recovery of sensations was 190.41 ± 82.05 in group I and 217.1 ± 73.19 in group II were statistically very significant

Table 8

Change in Pulse Rate

Stage (in minutes)	Pulse Rate in Range	
	Group I	Group II
0	66 – 100	70 – 100
30	64 – 90	70 – 90
60	68 – 92	68 – 90
120	72 – 90	70 – 88
180	70 – 86	72 – 92
240	74 – 88	66 – 88
Mean	80.0	80.33
S. D.	1.73	2.35
T = 0.277	p > 0.05	In significant

The patients in group I are having pulse rate per min between 64-100 with a mean of 80 ± 1.73 and patients in group II were having pulse rate between 66-100 with a mean of 80.33 ±2.35 were statistically insignificant.

Table 9

Change in systolic and diastolic blood pressure

Stage	Group I		Group II	
	Systolic	Diastolic	Systolic	Diastolic
0	110 – 150	70 - 92	110 – 130	74 – 94
30	110 – 150	68 – 90	118 – 150	76 – 90
60	114 – 140	72 – 92	120 – 146	78 – 88
120	122 – 150	76 – 92	122 – 148	80 – 90
180	120 – 146	70 – 90	112 – 140	76 – 88
240	116 – 140	72 – 86	110 – 136	80 – 92
Mean	130.66	80.33	128.5	83.83
S. D.	4.55	3.53	4.51	3.60
T = 0.277		p > 0.05	In significant	

The patients in group I are having mean blood pressure (mmHg) 130.66 and 80.33patients in group II were having blood pressure with a mean of 128.5 and 83.83 were statistically insignificant.

Table 10

Complications

Complications	Group I		Group II	
	No.	%	No.	%
During Injection				
1. Arterial Puncture	3	10	2	7
2. Chest Pain			1	3
3. Flushing of face				
During Operation				
1.Headache / giddiness				
Postoperative				
1. Haematoma	2	7	1	3
2. Neurological Complications	1	3		
3. Drowsiness				

DISCUSSION

Many times for the surgical procedure due to any reason anaesthesia may be required for more than one hour, which can not be obtained by brachial plexus block by lignocaine alone. Our study conducted is to evaluate the longer duration of the action of lignocaine with the addition of dextran 40 in brachial plexus block.

Loder9 in 1960 proved the prolongation of action of lignocaine by the addition of dextran 40 with adrenaline. This has been demonstrated by several other investigators before loder`s work. In our study the mean time for onset of analgesia was 14.15 ± 3.78 minutes for the lignocaine and 21 ± 4.49 minutes for lignocaine with dextran group. Moore et al, 197010 obtained a complete onset of analgesia in 10 minutes 40 ml of 1% lignocaine with 1: 200,000 adrenaline. It has been also reported a similar range of 4-22 min with a mean complete onset time of 12 min using 2 % lignocaine with adrenaline11.

The mean onset time for motor block was 21.00 ± 5.88 minutes and 29.16 ± 7.14 minutes for lignocaine and lignocaine with dextran group. It has been reported that onset of motor block follows the sensory block. It has been noted the mean onset of time of analgesia between 11.2 to 20.2 minutes and mean onset time for anaesthesia between 23.3 and 48.2 minutes for bupivacaine and ropivacaine in brachial plexus block, which clearly shows that onset of motor block follows sensory block14.

The regression time for sensory block in the lignocaine

group in our study varied from 45 -200 minutes with mean 132.13 ± 29.81 minutes. It was found that 1% lignocaine with adrenaline and reported longer regression time ranging between 220-260 min with mean of 240 min10. It was observed that total duration of analgesia for 296 min12.

In the present study the time for sensory and motor block is significantly increases in the lignocaine dextran group as compared to plain lignocaine group. The field blocks of the inguinal region in two groups of patients, in one group using a mixture of 0.5% bupivacaine, 1:200 000 adrenaline and dextran 110 and in the second group using a mixture of 0.5% bupivacaine, 1:200 000 adrenaline and dextran 150 the study being performed in a double-blind fashion. The dextran 150 mixture produced a highly significant increase ($p < 0.01$) in the duration of the block when compared with the dextran 110 mixture. The authors concluded that the use of dextran of increasing molecular weight increases the duration of action of local anaesthetics correspondingly13. The recovery of motor functions starts and completed before the sensory block. It was observed that the mean duration of analgesia to be 9.2 to 13 hrs and mean duration of motor block to be 5 -10.2 hrs for bupivacaine in brachial plexus block14. In the present study also the regression of motor block was found to be earlier than that of sensory block. Failure rate of the brachial plexus block was 5 out of 60 cases in the present study. 3 cases found to be in the lignocaine series and 2 cases were in lignocaine dextran group.

In the present study only mild to moderate changes in pulse rate and blood pressure was observed the patients in group I are having pulse rate between 64-100 with a mean of 80 ± 1.73 and patients in group II were having pulse rate between 66-100 with a mean of 80.33 ±2.35.in this way insignificant changes occur in pulse rate in both group I and II, this is confirmed by t test ($t = 0.277$, $p > 0.05$).

Mild to moderate changes in blood pressure were noticed patients in group I are having mean blood pressure 130.66 and 80.33 patients in group II were having blood pressure with a mean of 128.5 and 83.83 which is statistically insignificant.

In the present study, accidental arterial puncture was encountered in 5 cases out of which 3 patients had haematoma formation. The haematomas resolved with treatment within a week in all the cases without any sequelae. This complication has been discovered by most of author. Harley and Gjessing (1969) had arterial puncture as a complication in about 33% cases15. It has been observed that haematoma formation following arterial puncture in 2 out of 230 cases16.

In the present study no case of pneumothorax was observed. Hartmuth et al (1970) 17 and Steinberg et al (1970) 11 also found zero incidence of pneumothorax in their studies.

In the one case, however the patient had chest pain during the injection of drug. The chest pain in this case would have been due to irritation of nerve to serratus anterior.18

In this study Horner syndrome was not observed. Many authors Steinberg et al (1970) 11 and Brand and Papper (1961) 16 reported Horner syndrome as a complication of brachial plexus block In the lignocaine group one case had numbness of the whole upper limb till about 24 hrs. In this case tourniquets applied, the probable cause of this seems to be compression of nerves due to tourniquet.

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Author Information

Rajeev Puri, Lecturer
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Arpita Saxena, Lecturer
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Durgesh Kumar, Assistant Professor
Department of Physiology, Rama Medical College hospital and research centre
Kanpur. India

Trilok Chand, Professor
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Apurva Agarwal, Assistant Professor
Department of Anesthesia and Critical Care Medicine, G.S.V.M Medical College
Kanpur, India

Awak Mittal, Lecturer
Department of Orthopaedics ,S N Medical College
Agra. India

Apurva Mittal, Lecturer
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Mita Sarkar, Post graduate student
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Amit Gupta, Post graduate student
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Vivek Badada, Senior Resident
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India

Nitika Mittal, Post graduate student
Department of Anesthesia and Critical Care Medicine, S. N. Medical College
Agra. India