

## Original Research

### To compare the effect of epidural analgesia with parenteral analgesia on neonatal acid base status in dystocia requiring augmentation of labour

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#### ABSTRACT:

**Introduction:** The vicious cycle of dystocia and its augmentation leads to increase in maternal anxiety, physical exertion, metabolic demand for uterine contractions and release of cortisol and catecholamines. Increased pain and anxiety cause hyperventilation and respiratory alkalosis thereby leading to leftward shift of oxy-hemoglobin dissociation curve, decreased tissue oxygen transfer, metabolic acidosis and consequently uterine vasoconstriction, decreased placental blood flow and fetal acidosis. As there is scarcity of studies which specifically has looked into the effects of labour analgesia. It would be prudent to measure the impact of epidural analgesia in parturient requiring augmentation of labour on neonatal acid base parameters. **Materials and Method:** 60 labouring women with partographic evidence of slow progress of labour requiring augmentation in active phase of labour were enrolled in the present study. In Group 1 (epidural group), epidural analgesia was established and in group 2 (Intramuscular Tramadol group), patients received IM tramadol hydrochloride 1 mg/kg (with max. dose 400 mg / 24 hours) every 4 hours. Tramadol was discontinued when cervical dilatation was >8 cm. The neonatal acid base was assessed from the placenta by drawing 1 ml of arterial blood and the sample thus obtained was sent for analysis of acid base parameters. **Results:** Apgar scores of neonates at one minute were significantly better in epidural group compared to tramadol group ( $p=0.018$ ). Apgar scores at 5 minutes were similar. More than 97% of neonates in epidural group had Apgar scores >7 as compared to 80% in tramadol group. Cord pH values were significantly better in epidural group with all of the neonates in epidural group having pH >7.1 as compared to 8% of neonates in tramadol group. ( $p=0.018$ ). The mean cord pH between the group was statistically significant ( $p=0.05$ ). Neonates in epidural group had significantly higher base excess values as compared to that in tramadol group. **Conclusion:** To conclude, epidural analgesia led to better neonatal acid base parameters and had no significant adverse effects on either mother or neonates. Our result suggests that use of epidural analgesia in diagnosed case of dystocia reduces the risk of metabolic acidosis in neonates. However, one needs to be very vigilant when mothers are on epidural analgesia.

**Keywords:** Dystocia; Epidural analgesia; Fetal acidosis; Tramadol

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#### INTRODUCTION

Caring for women with dystocia is a major challenge in maternity care. Dystocia refers to prolonged or slowly progressing labour. In dystocia mothers are more anxious as they experience intense pain due to uncoordinated uterine contraction and contraction without cervical dilatation.<sup>1</sup> Intense labour pain by

itself retards progression of labour by increasing sympathetic activity. Increased sympathetic activity as well as stress associated with prolonged labour leads to catecholamine surge which inhibits endogenous oxytocin release that in turn worsens uterine contractions and prolongs labour.<sup>2</sup> Inhibition of endogenous oxytocin again restrains the activity of

oxytocin neurons in the brain impairing its ability to mediate anti stress profile as oxytocin participates in raising pain threshold, activation of vasovagal reflex, anxiolysis and sedation.<sup>3,4</sup>

Since 1940s, dystocia in absence of cephalo-pelvic disproportion has mostly been treated with oxytocin augmentation.<sup>5</sup> Its augmentation further increases pain without increasing the maternal pain threshold as exogenously administered oxytocin does not cross blood brain barrier unlike endogenous oxytocin.

This vicious cycle of dystocia and its augmentation leads to increase in maternal anxiety, physical exertion, metabolic demand for uterine contractions and release of cortisol and catecholamines. Increased pain and anxiety cause hyperventilation and respiratory alkalosis thereby leading to leftward shift of oxy-hemoglobin dissociation curve, decreased tissue oxygen transfer, metabolic acidosis and consequently uterine vasoconstriction, decreased placental blood flow and fetal acidosis.<sup>6</sup>

Studies by Pearson et al and Thalme et al on effect of continuous epidural analgesia upon fetal acid base status in both first and second stages of labour reported increased cord pH and base excess suggesting that epidural analgesia was associated with reduced fetal acidosis.<sup>7,8,9</sup> Zador et al<sup>10</sup> showed that epidural analgesia reduced both maternal and fetal acidosis even during prolonged second stage and also appeared to protect the fetus from the detrimental effects of prolonged second stage.<sup>10</sup> Reynolds et al<sup>11</sup> reported a meta-analysis comparing epidural with systemic opioids analgesia to determine the effect of these anaesthetic interventions on acid base status at birth and concluded that epidural analgesia was associated with improvement in base excess, suggesting that placental exchange was well preserved in association with this technique.<sup>11</sup>

As there is no study which specifically has looked into the effects of labour analgesia, it would be prudent to measure the impact of epidural analgesia in parturients requiring augmentation of labour on neonatal acid base parameters.

## MATERIALS AND METHOD

Sixty labouring women with partograph evidence of slow progress of labour requiring augmentation in active phase of labour were enrolled in this prospective randomized controlled study. A written informed consent was taken from all participating women before intervention. Inclusion criteria comprised of women in spontaneous and/or induced labour, vertex presentation, gestational age 36 to 41 weeks and patients who had slow progress with cervical dilatation (1cm/hrs) or arrest in cervical dilatation over 4 hours in active phase of labour and decision was taken to augment labour or to continue oxytocin in escalated dose in induced labour. Exclusion Criteria comprised of patient's refusal, any contraindication to regional technique or iv analgesia, congenital malformation of fetus, abruptio placentae

or placenta previa i.e. antepartum hemorrhage, coagulopathy/thrombocytopenia (platelets count <75000/uL), fetal distress, intrauterine infection, any allergy to bupivacaine or tramadol, multiple pregnancy, intrauterine fetal death, severe intrauterine growth restriction, severe preeclampsia, antepartum eclampsia, severe maternal medical disorder like heart diseases, insulin dependent diabetes, severe anemia, cervical dilatation > 9 cm, women who had received opioid analgesia within 4 hours, women who had already received epidural analgesia before start of study, malpresentation, women with evidence of obstructed labour or cephalo-pelvic disproportion.

The enrolled women were randomly allocated into two groups; group 1 or group 2 to receive analgesia using a computer generated random number table. The sequentially numbered sealed envelopes containing random number were opened just before initiation of analgesia by an investigator not further involved in the study.

In Group 1 (epidural group), epidural analgesia was established according to guidelines of department of Anesthesia and intensive care, PGIMER. and in group 2 (Intramuscular Tramadol group), patients in group 2 received IM tramadol hydrochloride 1 mg/kg (with max. dose 400 mg / 24 hours) every 4 hours. If additional doses were required, half of the initial dose was given. However, if required after 4 hours the same dose as the initial bolus was given with a maximum dose not exceeding 400 mg in 24 hours. Tramadol was discontinued when cervical dilatation was >8 cm. If patients complain of nausea or vomiting, antiemetic Ondansetron 4mg IV was given and recorded. Total dose of tramadol was recorded. Information regarding maternal history of present pregnancy, relevant past history, family history, obstetric history and routine investigations was taken and recorded. Onset of labour was defined as presence of regular painful uterine contractions i.e. 3 regular painful contractions over 10 min, together with at least one of the following features: mucoid or blood show, cervical dilatation of  $\geq 3$ cm, or spontaneous rupture of membrane.

Slow progress of first stage was defined as < 1cm of dilatation of cervix per hour after 3 cm dilatation of cervix. Maternal vitals were checked and every woman received intravenous infusion of 500ml of lactated Ringer solution 20 minutes before the start of the study in both groups. Afterwards intravenous maintenance fluid was given at the rate of 8-10 drops per minute in both groups. Oxytocin augmentation was done with similar oxytocin augmentation protocol in both groups as per PGIMER, labour room protocol. Low dose protocol was used using infusion pump. Thirty units of oxytocin was dissolved in 500 ml of normal saline. Infusion was started at the rate of 3 mU per minute (mU/min) and was increased at the rate of 3mU/min every 30min reaching maximum dose till moderate uterine contraction (3-4 contraction

in 10 minute lasting 30-40 second each) or maximum dose of 72mU/min was achieved. At the time of establishment of epidural analgesia maternal monitoring was done using electrocardiography (ECG), non-invasive blood pressure (NIBP) and Pulse oximetry. (CSI Criticare, Waukesha, Wisconsin) NIBP was recorded every 10 minutes initially for one hour after test drug administration, thereafter at regular intervals every hourly. Women in group 2 were monitored with hourly pulse, BP and respiratory rate. In both the groups pain was assessed by a 10 cms long marked visual analogue scale and numerical rating scale (NRS). All participating women were explained about the Visual Analogue Score and Numerical rating scores (VAS/NRS) where 0 means no pain and 10 means worst, unimaginable pain. Pain was scored at one hourly interval throughout the study period. Sedation was assessed by attending obstetrician on a S-point sedation score every hourly. No subsequent analgesia was given if sedation score was  $>2$ , Progress of labour was assessed by partogram maintained for each women Maternal satisfaction was recorded within 24 hours of delivery by the attending obstetrician. Point descriptive score of excellent, very good, good, fair or poor. Maternal hypotension was defined as systolic BP  $< 90$  mmHg or reduction in arterial blood pressure  $> 30$  mmHg from base line. For treatment of hypotension, boluses of phenylephrine 50ug was used. In both the groups labour monitoring was done by using WHO partograph. This was done using cardiotocography (CTG) (Huntleigh Health Care monitor, United Kingdom) or intermittent auscultation. Intermittent auscultation was done every 15 min in first stage and

every 10 min in second stage. Fetal heart rate was recorded immediately after contraction. Cardiotocography was performed for at least half hour after each dose of analgesia. In case of any fetal heart rate abnormalities and suspected fetal distress continuous cardiotocographic monitoring was done along with another method for fetal rescue i.e left lateral position, hydration, oxygen supplementation or omitting oxytocin were taken till heart rate normalizes. If CTG showed evidence of continued fetal heart rate abnormality suggesting hypoxia appropriate measure was taken to deliver the fetus.

### ASSESSMENT OF NEONATAL ACID BASE

Assessment of neonatal acid base was done by following method:

Immediately at birth one loop of cord was isolated after triply clamping the cord from the placenta. After identifying umbilical artery in that loop of cord 1 ml of arterial blood was drawn in heparinized syringe and care was taken to avoid air entry into the syringe. The sample thus obtained was sent for analysis of acid base parameters. Analysis was done using Cobas B 121 Analyzer. Umbilical cord Arterial blood gas (ABG) (pH  $< 7.1$  and base deficit  $> 8$  mcg was defined as fetal/neonatal acidosis). All the women were monitored for 24 to 48 hours postpartum period for complication like hypotension, motor weakness, urinary retention, sedation, nausea or vomiting, allergy fever. The neonates were followed up till discharge from hospital. The mother was advised to visit gynecology emergency if any problem persisted. They were followed up to 4 weeks postpartum by means of contact numbers/address.

## RESULTS

**Table 1: Age Distribution in both groups**

Age years	Epidural No. (%)	Tramadol No. (%)	Total No. (%)
$\leq 20$	2(6.7%)	1(3.3%)	3(5.0%)
21-25	11(36.7%)	12(40.0%)	23(38.3%)
26-30	14(46.6%)	7(23.4%)	21(35.0%)
31-35	3(10.0%)	6(20.0%)	9(15.0%)
$> 36$	0(0.0%)	4(13.3%)	4(6.7%)
Total	30	30	60
p $>0.05$ (Pearson Chi-Square 0.057(0.103))			

Age distribution of women enrolled in study is as shown in table 1. Of the 60 women more than 50% were in their third decade of life. Twenty three (38.3%) women were between 20-25 years and twenty

one between 26-30 years. The mean age of women in epidural group was  $26.20 \pm 3.41$  years and in tramadol group was  $28.00 \pm 15.16$  years which was comparable in both groups.

**Table 2: Cord pH values between both groups**

pH	Epidural No. (%)	Tramadol no. (%)	Total
$< 7.1$	0 (0%)	1 (3.3%)	1 (1.7%)
7.1-7.2	10 (34.5%)	4 (13.3%)	14 (23.7%)
7.21-7.3	15 (51.7%)	25 (83.4%)	40 (67.8%)
$> 7.3$	4 (13.8%)	0 (0%)	4 (6.8%)
Total			

Only one neonate in tramadol group had a PH $<7.1$ . This neonate from tramadol group had birth asphyxia

and had apgar scores 4 and 9 at 1 min and 5 min respectively, Cord pH was 7.05 and base excess was-

15.4. The neonate had fetal heart rate deceleration 80 bpm for 15 second during second stage and was delivered by normal vaginal delivery with episiotomy quickly. The augmentation delivery time was 3 hrs. This neonate quickly responded to initial resuscitative measures. The mean cord pH was  $7.26 \pm 0.63$  in

epidural group and  $7.23 \pm 0.057$  in tramadol group. This was statistically significant ( $p=0.05$ ). All the neonates of epidural group had PH  $\geq 7.1$ . One woman in epidural group had intrauterine fetal demise and has been excluded from the results.

**Table 3: Base excess values between groups**

Base excess	Epidural No.(%)	Tramadol No.(%)	Total No.(%)	P value
<-10	3(10.3%)	14(46.7%)	17(28.8%)	0.002
-8 to -9.9	4(13.8%)	2(6.7%)	6(10.2%)	0.667
-6 to -7.9	12(41.4%)	10(33.3%)	22(37.3%)	0.182
-4 to -5.9	5(17.2%)	3(10.0%)	8(13.6%)	0.500
>-4	5(17.2%)	1(3.3%)	6(10.2%)	0.102
Total	29	30	59	
$p < 0.05$ (Pearson Chi-Square=0.025)				

The base excess was found to be  $\geq -4$  in 5 neonates in epidural group and only one neonates in tramadol group. In the epidural group total of 41.4% of neonates had base excess between 6 to 7.9 as compared to 33.3% in tramadol group. Significantly higher number of neonates (47%) were born with metabolic acidosis (base excess 8 or below) in tramadol group in comparison to epidural group (10%). Mean base excess was  $7.02 \pm 3.10$  in epidural group and  $9.327 \pm 3.35$  in tramadol group respectively. The difference between the mean was statistically significant ( $p=0.008$ ). There were six neonates in the tramadol group with base excess of  $\leq 12$  where as 2

neonates in epidural group were born with base excess  $< 12$  ( $p=0.254$ ) (table 3). The mean cord pH in epidural group after including the neonates of one woman who received epidural from tramadol group was  $7.265 \pm 0.06$  which was comparable with tramadol group with the mean pH of  $7.23 \pm 0.057$ . The difference in mean was not statistically significant. The mean base excess value in epidural group after including the neonate of one woman was  $-7.06 \pm 3.06$  which was again significantly better than in tramadol group with mean base excess of  $-9.35 \pm 3.41$ . The difference in mean was again statistically significant. (table 4)

**Table 4: Cord pH and base excess based on the analgesic regimen women received during labour**

	Group	Number	Mean	Std. Deviation	Std. Error Mean	P value
pH	Epidural	30	7.26553	0.062423	0.011397	0.69
	Tramadol	29	7.23300	0.57204	0.010622	
Base excess	Epidural	30	-7.067	3.0628	0.5592	0.008
	Tramadol	29	-9.359	3.4115	0.6335	

The distribution of Apgar scores with relation to cord pH values has been shown in table 5. In this study out of 44 neonates having cord pH values more than 7.2. Five neonates had Apgar score at 1 min  $< 7$ . All of these 5 women were from tramadol group. One woman had Apgar scores of 1 at one min and 7 at 5 minutes. This baby had birth asphyxia and required naloxone. The baby developed jaundice and received phototherapy for three days and was discharged on day 4 of life. The augmentation delivery time was 20

hours and was delivered by caesarean section for fetal bradycardia. This neonate did not require NICU care. Base excess in this neonate was 7.4. Two neonates were born with Apgar score of 6, 8 and 6, 9 at 1 and 5 minutes respectively. One of these neonates was delivered by outlet forceps delivery for prolonged second stage and another by caesarean delivery for non progress of labour. Augmentation delivery interval in these women were 16 hrs and 10 hrs respectively.

**Table 5: Comparison of cord pH with Apgar Scores**

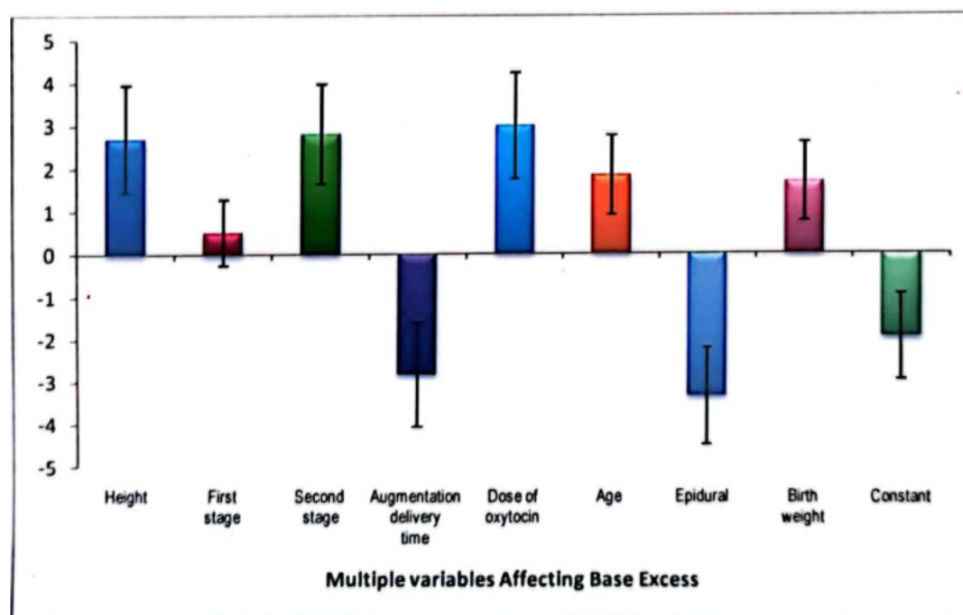
Cord pH	Apgar Score			Total
	<7	7	>7	
<7.1	1 (16.7%)	0 (0.0%)	0 (0.0%)	1 (1.7%)
7.1-7.2	0 (0.0%)	0 (0.0%)	14 (26%)	14 (23.7%)
7.21-7.3	5 (83.3%)	0 (0.0%)	35 (67.3%)	40 (67.8%)
>7.3	0 (0.0%)	1 (100.0%)	3 (5.8%)	4 (6.8%)
Total	6	1	52	59
$p < 0.05$ (Pearson Chi-square=0.000)				

Multiple maternal as well as intrapartum factors which were likely to affect base excess in neonates were taken in to account in logistic regression analysis to find out which factors significantly increase the risk of low base excess. i.e.  $< 8$ . In epidural group odds ratio (OR) for base excess less than 8 was 0.036 which was statistically significant ( $p=0.004$ ) indicating low chances of acidosis due to epidural analgesia. If height was less than 152 cm OR ratio for base excess  $< 8$  was 14.87. When first stage of labour was more than 12 hours then OR for base excess  $< 8$  was 1.7. When second stage was more than 2 hours then OR for base excess  $< 8$  was 16.57 which was statistically significant indicating that prolonged second stage affect base excess value significantly. When augmentation delivery time was less than 10 hours OR for base excess  $< 8$  was 0.060 which

was statistically significant indicating that chances of acidosis was less when augmentation delivery time was less than 10 hours. When the dose of oxytocin used was more than 45 mU/min then OR for base excess  $< 8$  was 20 which was statistically significant indicating that dose of oxytocin significantly affects base excess value. When the age of the mother was more than 27 OR for base excess  $< 8$  was 6.30. When birth weight was less than 3 kg then OR for base excess was 5.39. Among compared variables epidural analgesia, second stage less than 2 hours, height more than 152 cms, dose of oxytocin less than 45mU/min, augmentation delivery time less than 10 hours had positive correlation with base excess. Logistic regression analysis could not be applied for cord pH as numbers of neonates having cord pH less than 7.1 were very few (table 6, figure 1)

**Table 6: Multiple variables affecting base excess (intention to treat basis)**

Variable	B	S.E.	Wald	df	Sig	Exp (B)	95% CI for EXP (B)	
							Lower	Upper
Height	2.699	1.250	4.659	1	.031	14.868	1.282	172.454
First Stage	.517	.765	.458	1	.499	1.678	.375	7.512
Second Stage	2.808	1.166	5.801	1	.016	16.570	1.687	162.769
Augmentation Delivery Time	-2.808	1.234	5.170	1	.023	.060	.005	.679
Dose of oxytocin	2.997	1.238	5.862	1	.015	20.31	1.770	226.703
Age	1.840	.929	3.924	1	.048	6.296	1.020	38.868
Epidural	-3.324	1.142	8.480	1	.004	.036	.004	.337
Birth Weight	1.685	.922	3.342	1	.068	5.393	.886	32.845
Constant	-1.943	1.005	3.736	1	.053	.143		



**Figure 1: Multiple variables affecting base excess**

## DISCUSSION

Cord blood gas analysis is the gold standard for assessing fetal acid-base status and utero-placental function at birth. Umbilical artery pH and base excess reflect fetal and immediate neonatal condition whereas umbilical vein values reflect maternal acid-

base status and placental function. We analyzed these neonatal parameters to study the effects of the analgesics on neonatal outcome.

In our study umbilical arterial blood was used for cord blood gas analysis. In our study population severe acidosis was uncommon. In the present study

all of neonates in epidural group had pH  $\geq 7.1$  as compared to 97% of neonates in tramadol group.

Normal values vary depending on the definition of normality and the influence of factors like altitude, parity, breech vaginal delivery and duration of labour on the population studied. The normal umbilical artery pH is said to be  $>7.2$  and base excess - 10 to 0 mmol/l.<sup>12</sup>

Helwig et al retrospectively examined the records of 15,000 vigorous newborns with a five minutes Apgar scores of  $>7$ . Mean umbilical artery values, with the 2.5th percentile value in parentheses, were pH 7.26 (7.10) and base excess 4 mmol/l (11 mmol/l). The means  $\pm 2$  standard deviations were similar. The generally accepted lowest limit of normal umbilical artery pH extends to 7.10 and base excess to 12 mmol/l.<sup>13</sup> In our study we took pH  $<7.1$  and base excess  $<8$  to define acidosis in neonates.<sup>14</sup>

Base excess values is of greater usefulness than pH values because base excess does not change significantly with respiratory acidosis and demonstrates linear, rather than logarithmic, correlation to the degree of metabolic acidosis. Umbilical artery base excess is the most direct measure of fetal metabolic acidosis.<sup>15</sup>

In our study 24% of neonates had base excess of less than 8 in epidural group as compared to 53% of neonates in tramadol group. In the present study these neonatal parameters were better in epidural group probably as a correlation to better pain relief and less maternal exhaustion than in tramadol group. Our results with the neonatal parameters were consistent with previous studies.<sup>16,17,18,19</sup>

In a randomised study Throp et al reported better cord pH in epidural group compared to opioid group though the difference was not statically significant ( $p>0.05$ ). The mean pH values were  $7.26\pm0.06$  and  $7.24\pm0.06$  in epidural and opioids groups respectively.<sup>20</sup> Ramin et al<sup>21</sup> also in their study did not find any difference in cord pH, base excess or Apgar scores in women who received epidural analgesia as compared to opioid analgesia.<sup>21</sup> Both these studies included women in spontaneous labour and bupivacaine was used in higher concentration. Though they reported increased analgesics efficacy in epidural group, they also reported increased in caesarean delivery rates which probably had masked the beneficial effects of epidural analgesia in these studies.

In contrast Clark et al<sup>19</sup> in a randomized study, reported that the neonates of mothers receiving epidural were less likely to have cord pH  $<7.15$  (RR0.33, 95%CI,  $P<0.05$ ). The mean cord pH was  $7.24\pm0.05$  and  $7.23\pm 0.08$  in epidural and opioid groups respectively. The epidural group also had significantly better Apgar scores at 1 and 5 mins ( $p<0.05$ ) as compared to opioids. However, no comments were made on base excess values. They included women in spontaneous labour and used

higher dose of bupivacaine. They also had increased rate of caesarean deliveries in epidural group.

Another non randomized study by Zador and Nilsson reported that umbilical artery pH and base excess were better in the epidural group as compared to opioids ( $p=0.05$ ). Better cord pH values were found even when second stage was prolonged in epidural group.<sup>10</sup>

Reynolds et al concluded that fetal pH and base excess was higher in epidural group suggesting that placental exchange was well preserved in association with maternal sympathetic blockage and good analgesia. Though epidural analgesia caused few potential adverse effects they appeared to be outweighed by the benefits to neonatal acid-base status.<sup>22</sup>

Intrapartum hypoxia is reflected in the acid base parameters of neonates. Apgar score is a clinical assessment and might not have strong correlation with long term neonatal outcome. However, when combined with acid base parameters the correlation is better. Based on these findings it can be said that higher cord pH and base excess predict better long term neonatal outcome.

All the neonates in epidural group had Apgar scores at 1min and 5 min more than seven as compared to 80% neonates in tramadol group. Only one neonate in epidural group needed resuscitation by bag and mask for 30 seconds. These neonates had Apgar score of 7 at one minute. The neonate was delivered by normal vaginal delivery; augmentation delivery time was 4 hours only and did not require any special care. The mother had received epidural analgesia 4 hours prior to delivery and no fetal heart rate abnormality was noted after administration of drugs. The neonate had cord pH of 7.3 and base excess of 3.4.

In yet another randomised study comparing effects of analgesia on labour outcome, Sharma et al<sup>18</sup> reported significantly better Apgar scores at 1 min and cord pH in epidural group ( $p<0.001$ ). Mean pH values were  $7.26\pm0.07$  in epidural group and  $7.24\pm0.08$  in meperidine group. They reported that use of naloxone was significantly higher in opioid group ( $p=0.001$ ). Lower base excess values and abnormal CO<sub>2</sub> tension ( $>65$ mm of Hg) were significantly more common in the meperidine analgesia group ( $p<0.019$ ). The mean base excess values were 4.6 and 5.76 in epidural and meperidine groups respectively. However, NICU admissions were similar. Similar to our study they had included only nulliparous women. In our study the findings corroborate with that noted by Sharma et al.<sup>18</sup> In our study there was a trend for the neonates to be born with better pH, base excess and better Apgar scores when mother had received epidural analgesia. Halper et al in a meta-analysis which included 10 randomised trials also reported better Apgar scores (Apgar 1 min  $<7$  OR 0.54, 95%CI,  $p=0.001$ ), better cord pH values (OR0.76, 95%CI,  $p=0.04$ ) and lesser need for naloxone (OR0.24, 95% CI,  $p<0.001$ ) in epidural group as compared to opioid group.<sup>12</sup>

Silverman et al in their study of clinical significance of Apgar scores reported that when the pH was below 7.05 there was a statistically significant increase in Apgar scores below 7 at five minutes ( $p < 0.001$ ). The incidence of low one minutes Apgar scores was greater with an umbilical artery pH below 7.2, where 70% of neonates who had pH < 7.2 had Apgar < 7. They also reported that in clinical use a low Apgar may be regarded as more significantly related to long term outcome although the actual relationship is modest.<sup>23</sup> In our study 5 neonates in tramadol group were born with APGAR score < 7 at one min and received naloxone and pH and base excess in this was 7.05 and 15.5 respectively. In our study correlation between Apgar score and metabolic acidosis was modest at the best.

Victory et al in another study reported that term infants with acidosis at birth had higher risk of Apgar less than 7 at 5 minutes, NICU admission, and need for assisted ventilation. Low Apgars, NICU admission and assisted neonatal ventilation had significant inverse relationships with both umbilical artery and umbilical vein pH and base excess (all  $p < 0.001$ ).<sup>24</sup>

In our study we correlated base excess with method of analgesia used, age of mother, height of mother, dose of oxytocin used, duration of first and second stage of labour, oxytocin to delivery time and birth weight of neonates Among compared variables epidural analgesia, second stage less than 2 hours, height more than 152 cms, dose of oxytocin less than 45mU/min, augmentation delivery time less than 10 hours had positive correlation with base excess. Maternal height less than 150cm has been noted to increase the risk of dystocia and thus may increase risk of fetal acidosis as was seen in our study. In general duration of labour particularly that of second stage is widely held as risk factor of asphyxia.<sup>25</sup> However, in a large study duration of second stage of labour has not been found to correlate with low Apgar score at 5 minute, neonatal seizure, admission to neonatal intensive care unit provided fetal heart trace were normal.<sup>26</sup> Monitoring of labour, use of epidural analgesia risk of cephalopelvic disproportion in a particular patient population are important factors which need to be considered before allowing a patient to be in prolonged second stage (2hour).

We in our study noted significantly increased risk of fetal acidosis and fetal death when second stage was prolonged.

## CONCLUSION

To conclude, in our study Apgar scores of neonates at one minute were significantly better in epidural group compared to tramadol group ( $p = 0.018$ ). Apgar scores at 5 minutes were similar. More than 97% of neonates in epidural group had Apgar scores > 7 as compared to 80% in tramadol group. Cord pH values were significantly better in epidural group with all of the neonates in epidural group having pH > 7.1 as

compared to 8% of neonates in tramadol group. ( $p = 0.018$ ). The mean cord pH between the group was statistically significant ( $p = 0.05$ ). Neonates in epidural group had significantly higher base excess values as compared to that in tramadol group. In logistic regression analysis epidural analgesia, second stage less than 2 hours, height more than 152 cms, dose of oxytocin less than 45mU/min, augmentation delivery time less than 10 hours had positive correlation with base excess.

Epidural analgesia led to better neonatal acid base parameters and had no significant adverse effects on either mother or neonates. Our result suggests that use of epidural analgesia in diagnosed case of dystocia reduces the risk of metabolic acidosis in neonates as well as dystocia related caesarean section rate. However, one needs to be very vigilant when mothers are on epidural analgesia. The sample size in our study was small and so our results need to be corroborated in larger patient.

## REFERENCES

1. Hess PE, Pratt SD, Soni AK, Sara MC, Oriol NE. An association between severe labour pain and cesarean delivery. *Anesth Analgesia*. 2000;90:881-6.
2. Lederman R, Lederman E, Work Jr B, McCann D. The relationship of maternal anxiety, plasma catecholamines, and plasma cortisol to progress in labour. *Am J Obstet Gynecol*. 1978;132:495-500.
3. Petersson M, Alster P, Lundeborg T, Uvnäs-Moberg K. Oxytocin increases nociceptive thresholds in a long-term perspective in female and male rats. *Neurosci Lett*. 1996;212:87-90.
4. Uvnäs-Moberg K, Ahlenius S, Hillegaard V, Alster P. High doses of oxytocin cause sedation and low doses cause an anxiolytic-like effect in male rats. *Pharmacol Biochem Behav*. 1994;49:101-6.
5. Oscarsson ME, Amer-Wählin I, Rydhstroem H, Källén K. Outcome in obstetric care related to oxytocin use. A population-based study. *Acta obstetrica et gynecologica Scandinavica*. 2006 Sep;85(9):1094-8.
6. Sangoul F, Fox GS, Houle GL. Effect of regional analgesia on maternal oxygen consumption during the first stage of labor. *American Journal of Obstetrics and Gynecology*. 1975 Apr 1;121(8):1080-3.
7. Pearson JF, Davies P. The effect of continuous lumbar epidural analgesia upon fetal acid-base status during the second stage of labour. *BJOG: An International Journal of Obstetrics & Gynaecology*. 1974 Dec;81(12):975-9.
8. Pearson JF, Davies P. The effect of continuous lumbar epidural analgesia on the acid-base status of maternal arterial blood during the first stage of labour. *BJOG: An International Journal of Obstetrics & Gynaecology*. 1973 Mar;80(3):218-24.
9. Thalme B, Belfrage P, Raabe N. Lumbar epidural analgesia in labour: I. Acid-base balance and clinical condition of mother, fetus and newborn child. *Acta obstetrica et gynecologica Scandinavica*. 1974 Jan;53(1):27-35.
10. Zador G, Nilsson BA. Low Dose Intermittent Epidural Anaesthesia with Lidocaine for Vaginal Delivery II: Influence on labour and foetal acid-base status. *Acta*

- ObstetriciaetGynecologica Scandinavica. 1974 Jan;53(S34):17-30.
11. Reynolds F, Sharma SK, Seed PT. Analgesia in labour and fetal acid–base balance: a meta-analysis comparing epidural with systemic opioid analgesia. BJOG: an international journal of obstetrics and gynaecology. 2002 Dec 1;109(12):1344-53.
12. Halpern SH, Leighton BL, Ohlsson A, Barrett JF, Rice A. Effect of epidural vs parenteral opioid analgesia on the progress of labor: a meta-analysis. Jama. 1998 Dec 23;280(24):2105-10.
13. Helwig JT, Parer JT, Kilpatrick SJ, Laros RK. Umbilical cord blood acid-base state: what is normal? Am J Obstet Gynecol. 1996;174:1807-14.
14. Scott PH, Warton BA. Biochemical values in newborn. In: NRC Robertson, editor. Textbook of neonatology. Edinburg: Churchill Livingstone; 1986:839-41.
15. Littleford J. Effects on the fetus and newborn of maternal analgesia and anesthesia: a review. Can J Anesth. 2004;51:586-609.
16. Ramin SM, Gambling DR, Lucas MJ, Sharma SK, Sidawi JE, Leveno KJ. Randomized trial of epidural versus intravenous analgesia during labor. Obstetrics & Gynecology. 1995 Nov 1;86(5):783-9.
17. Thorp JA, Hu DH, Albin RM, McNitt J, Meyer BA, Cohen GR, Yeast JD. The effect of intrapartum epidural analgesia on nulliparous labor: a randomized, controlled, prospective trial. American journal of obstetrics and gynecology. 1993 Oct 1;169(4):851-8.
18. Sharma SK, Alexander JM, Messick G, Bloom SL, McIntire DD, Wiley J, Leveno KJ. Cesarean Delivery: A Randomized Trial of Epidural Analgesia versus Intravenous Meperidine Analgesia during Labor in Nulliparous Women. The Journal of the American Society of Anesthesiologists. 2002 Mar 1;96(3):546-51.
19. Clark A, Carr D, Loyd G, Cook V, Spinnato J. The influence of epidural analgesia on cesarean delivery rates: a randomized, prospective clinical trial. American journal of obstetrics and gynecology. 1998 Dec 1;179(6):1527-33.
20. Newton ER, Schroeder BC, Knape KG, Bennett BL. Epidural analgesia and uterine function. Obstetrics & Gynecology. 1995 May 1;85(5):749-55.
21. Jain S, Arya VK, Gopalan S, Jain V. Analgesic efficacy of intramuscular opioids versus epidural analgesia in labour. Int J Gynaecol Obstet. 2003;83:19-27.
22. Reynolds F, Russell R, Porter J, Smeeton N. Does the use of low dose bupivacaine/opioid epidural infusion increase the normal delivery rate?. International Journal of Obstetric Anesthesia. 2003 Jul 1;12(3):156-63.
23. Silverman F, Suidan J, Wasserman J, Antoine C, Young BK. The Apgar score: is it enough? Obstet Gynecol. 1985;66:331-6.
24. Victory R, Penava D, da Silva O, Natale R, Richardson B. Umbilical cord pH and base excess values in relation to adverse outcome events for infants delivering at term. <sup>[1]</sup><sub>SEP</sub>Am J Obstet Gynecol. 2004;191:2021-8.
25. Le Goueff F, Garite TJ. 14. American College of Obstetricians and Gynecologists. Dystocia and augmentation of labor. Washington (DC): The College; 2003. ACOG Practice Bulletin no.: 49. 15. Goffinet F, Langer B, Carbonne B, Berkane N, Tardif D.
26. Menticoglou SM, Manning F, Harman C, Morrison I. Perinatal outcome in relation to second-stage duration. Am J Obstet Gynecol. 1995; 173:906-12.