# **Doppler Findings in Intrapartum Fetal Distress**

Laleh Eslamian and Khatereh Tooba

Department of Obstetrics and Gynecology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

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Abstract- The umbilical vein (UV) has a non pulsating and even pattern in normal fetuses. Pulsation of UV has been described in severely growth restricted fetuses with chronic hypoxia. We wanted to see whether UV pulsations could also be seen in fetuses with heart deceleration during labor, as an adjunctive measure to assess the intra partum hypoxia. In a prospective study Doppler examination was performed on 34 fetuses with normal cardiotocography (CGT) and 26 fetuses with abnormal CTGs (GA>37w and cervical dilatation>3cm). Perinatal outcome was assessed according to presence or absence of UV pulsations. The 2 groups were similar regarding gestational age, cervical dilatation, Umbilical artery blood pH, S/D ratio, Pulsatility Index(PI) and Resistance Index (RI). Intraabdominal UV pulsation were present in 6 (23.1%) of abnormal CTG group but no case were seen in normal CTG group (P=0.005). Five of 6 (83.3%) fetuses with UV pulsation underwent cesarean delivery. The rate of cesarean delivery was 90% in abnormal CTG group without pulsation and 14.7% in normal CTG group. The frequency of Apgar score <7 was more in fetuses with UV pulsations (16.7% vs 5%) although not statistically significant. NICU admission was considerably more in UV pulsation group (33% vs 5%, P= 0.123). After exclusion of LBW fetuses the UV pulsation was present in 4 (19%) of abnormal CTG group, who 3 of them underwent cesarean section. Neither umbilical artery pH<7 nor Apger score <7 or NICU admission were seen in these 4 neonates. Pulsation in UV was seen in 23% of fetuses with abnormal CTG during intra partum period. Cesarean delivery and NICU admission was increased in fetuses with UV pulsations, although not statistically significant. When LBW fetuses were excluded no case of UA pH<7, Apgar sore <7or NICU admission were seen.

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# Introduction

Examination of venous Doppler waveform in fetus is reported in early 1980 (1,2). Umbilical vein (UV) Doppler assessment for acid-base prediction is performed in FGR fetuses (3,4). Umbilical vein waveform is pulsatile in early pregnancy, but it resolves between 8-13 weeks (5). The UV has a non pulsatile and even pattern in normal fetuses.

Linginan *et al.* described pulsatile UV in fetuses with imminent asphyxia (6) and later Gundmundsson *et al.* in fetuses with non immune hydrops (7). In serverly ill growth restricted fetuses, the UV has a pulsatile pattern due to increased central venous pressure in hypoxia and in hydropic fetuses in response to heart failure (8,9). Several authors have reported pulsatile UV in fetus with chronic hypoxia (10,11). Ghosh *et al.* have recently performed studies on UV during labor (12). They found pulsations in UV in 30.8% of fetuses with pathological cardiotocography (CTG), which increased the operative delivery for fetal distress. In this study we wanted to examine pulsation in the UV in the fetuses with abnormal CTG and whether it could affect the delivery mode.

## **Materials and Methods**

A prospective double blind study was designed at Shariati hospital after approval by Tehran University of Medical Sciences ethic committee.

The study included pregnant women with single cephalic fetuses and gestational weeks >37 weeks in active phase of labor (cervical dilation>3cm). Exclusion criteria were second stage of labor and when immediate intervention did not allow Doppler study. Informed consent was obtained from all these pregnant women.

Corresponding Author: Laleh Eslamian

Department of Obstetrics and Gynecology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

Tel:+98 21 84902415, Fax: +98 21 88633039, Email: leslamian@tums.ac.ir

Doppler study was performed on 34 pregnant women with normal and 26 with abnormal CTG. Abnormal CTG was defined as: repeated late decelerations, prolonged deceleration, repeated variable decelerations with a duration more than 60 seconds or deceleration > 70 bpm and tachycardia with pathological variability.

Through a 2D color Doppler technique, umbilical artery (UA), UV and middle cerebral artery (MCA) was assessed by an Acuson sequoia 512 machine in supine position. Doppler study was performed on both intra abdominal portion and free loop of UV between contractions in the absence of fetal movement. At least 10 heart cycle were included and the angle of insonation was less than 30 degree. The presence or absence of pulsations were recorded. The maximum blood velocity was reduced 15% or more when UV pulsations were present. No one was aware of Doppler results except the sonographist who was not the managing obstetrician.

The UV Doppler results were compared to the perinatal outcome: UA blood pH, Apgar score, birth weight, delivery mode and NICU admission. Data analysis was done using SPSS version 11.5. Continuous data was compared by Mann Whitney U test and categorical data by chi-square or Fisher's exact test when appropriate. A P-value of <0.05 was considered significant.

#### Results

The study included 60 pregnant women, 34 with normal CTG and 26 with abnormal CTG. The clinical characteristics of the two groups were similar (Table 1).

Although premature rupture of membrane (PROM) and thick meconium were more frequent in abnormal CTG group, but their difference were not statistically significant (Table 1).

More women were induced by misoprostol in normal CTG group (41% vs 11.5% P = 0.041) (Table 1).

Pulsation in the intra abdominal UV were present in 6 (23.1%) of abnormal CTG group and none of normal CTG group (P= 0.005). No pulsation was seen in free loop of UV. Five of six (88.3%) women with pulsation in the intra-abdominal UV underwent cesarean, 2 had late, 2 had repetitive variable deceleration and one had tachycardia with absence of variability.

The rate of cesarean was significantly more in abnormal CTG group in comparison with normal CTG group (88.5% vs 14.7%, P=0.001). The rate of cesarean was similar in abnormal CTG group with or without UV pulsation (83.3% vs 40%, P=1) (Table 2).

Although the birth weight was significantly less in abnormal CTG group but there was no statistically significant difference between birth weight in abnormal CTG group with or without UV pulsation (P=0.128) (Table 2).

NICU admission was similar in normal and abnormal CTG group (9.1% vs 11.5%, P=1). The rate of NICU admission in abnormal CTG group was considerably more in women with UV pulsation than in women without although not statistically significant (33% vs 5%, P=0.123), (Table 2).

Analysis was repeated after excluding LBW neonates. (One in normal CTG and 5 in abnormal CTG group, P = 0.076) (Table 3,4). Now, pulsation in UV was present in 4 (19%) of abnormal CTG group. Three of them under went cesarean delivery, 1 had late, 1 had repeated variable deceleration and one tachycardia with no variability. No case of umbilical artery PH <7, Apgar score <7 or NICU admission were seen in these four neonates (Table 3).

	Normal CTG	Abnormal CTG	· · · · · · · · · · · · · · · · · · ·	I CTG	Р	
	(n =34)	(n = 26)			UV Pulsations (n = 6)	
Gestational week	39.5 (37.0-41.4)	39.1 (37.0-40.4)	0.149	38.9(37.3-40.3)	39.9(37.0-40.4)	0.428
Cervical dilatation	3 (3-9)	3 (3-8)	0.539	3 (3-5)	3 (3-8)	1
PI	0.82 (0.64-1.20)*	0.91 (0.69-1.17)**	0.408	0.92 (0.69-1.17)	0.89 (0.80-0.96)	0.720
RI	$0.57 (0.48 - 0.68)^{*}$	0.58 (0.48-0.71)**	0.407	0.59 (0.48-0.71)	0.58 (0.55-0.62)	0.857
S/D	2.27 (1.57-3.70)	2.44 (1.92-3.61)***	0.224	2.38 (1.92-3.42)	2.63 (195-3.61)	0.158
PROM	4(11.8)	6 (23.1)	0.305	6 (30.0)	0	0.280
Thick meconium	1 (2.9)	4 (15.4)	0.156	3 (15.0)	1 (16.7)	1
Induction by:			0.041			0.673
Oxytocin	8 (23.5)	9 (34.6)	0.156	6 (30.0)	3 (50.0)	
Misoprostol	14 (41.2)	3 (11.5)	0.156	3 (15.0)	0	

Data presented as Median (range) or n (%)\*, measured in 25\*\*, measured in 21\*\*\*, measured in 22 women

	Normal CTG	Abnormal CTG	P Abnormal CTG		nal CTG	Р
	(n=34)	(n=26)		No UV Pulsations (n = 20)	UV Pulsations (n = 6)	
Umbilical artery PH	7.23 (6.96-7.35)	7.26 (7.02 – 7.37)	0.477	7.28 (7.02-7.37)	7.19 (7.02-7.33)	0.211
Umbilical artery	3 (9.1)	4 (15.4)	0.688	3 (15)	1 (16.7)	1
PH<7.1						
Apgar Score	9 (6-10)	9 (4 -10)	0.031	9 (6-10)	9 (4 - 9)	0.185
Apgar Score <7	1 (2.9)	2 (7.7)	0.574	1 (5.0)	1 (16.7)	0.415
Birth weight	3200 (2400-4000)	3000 (2000-3600)	0.036	3025 (2280-3600)	2710 (2000-3250)	0.128
Cesarean section	5 (14.7)	23 (88.5)	< 0.001	18 (90.0)	5 (83.3)	1
NICU admission	3 (9.1)	3 (11.5)	1	1(5.0)	2 (33.3)	0.123

Data presented as Median (range) or n (%)

Table 3. Clinical characteristics after exclusion of LBW	neonates
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	Normal CTG (n =33)	<b>Abnormal CTG</b>	Р	Abnormal CTG		P s
		(n = 21)		UV Pulsations (n=4)		
Gestational week	39.5 (37.0-41.4)	39.0 (37.0-40.4)	0.324	38.7 (37.3-40.3)	40.0 (37.0-40.4)	0.242
Cervical dilatation	3 (3-9)	3 (3-8)	0.809	3 (3-5)	3 (3-8)	0.764
PI	$0.84(0.64-1.20)^{*}$	0.91 (0.69-1.17)**	0.843	0.92 (0.69-1.17)	0.87 (0.80-0.91)	0.659
RI	$0.57 \left(0.48 \text{-} 0.68\right)^{*}$	0.58 (0.48-0.71)**	0.832	0.59 (0.48-0.71)	0.58 (0.55-0.58)	0.704
S/D	2.27 (1.57-3.70)	2.37 (1.92-3.42)**	0.682	2.34 (1.92-3.42)	2.62 (1.95-2.68)	0.614
PROM	4(12.1)	6 (28.6)	0.162	6 (35.3)	0	0.281
Thick me conium	1 (3.0)	3 (14.3)	0.287	2 (11.8)	1 (25.0)	0.489
Induction by:			0.092			0.581
Oxytocin	7 (21.2)	9 (28.6)		4 (23.5)	2 (50.0)	
Misoprostol	14 (42.2)	3 (14.3)		3(17.6)	0	

Data presented as Median (range) or n (%)<sup>\*</sup>, measured in 24<sup>\*\*</sup>, measured in 17 women.

	Normal CTG	<b>Abnormal CTG</b>	P Abnormal		nal CTG	Р
	(n=33)	(n=21)		No UV Pulsations (n=17)	UV Pulsations (n=4)	
Umbilical artery PH	7.23 (6.96-7.35)	7.28 (7.07-7.37)	0.109	7.29 (7.07-7.37)	7.24 (7.19-7.33)	0.501
Umbilical artery PH<7.1	3 (9.4)	4 (4.8)	1	1 (5.9)	0	1
Apgar Score	9 (6-10)	9 (8-10)	0.202	9 (8-10)	9 (9-9)	1
Apgar Score <7	1 (3.0)	0	1	0	0	-
Birth weight	3200 (2500-4000)	3100 (2650-3600)	0.226	3100 (2750-3600)	2935 (2650-3250)	0.243
Cesarean section	5 (12.1)	18 (85.7)	< 0.001	15 (88.2)	3 (75.0)	0.489
NICU admission	3 (9.4)	0	0.269	0	0	-

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## Discussion

The UV has a non pulsating and even pattern in normal fetuses. Pulsatile UV is seen in fetuses with imminent asphyxia and chronic hypoxia (6,10,11).

We saw intra abdominal UV pulsation in 23.1% of fetuses with abnormal CTG and in 19% when LBW fetuses were excluded. No case of UV pulsation was seen in fetuses with normal CTG. No pulsation was seen in free loop of UV.

Ghosh et al have seen pulsating UV in 30.8% of fetuses with pathological CTG during labor (12).

PROM and thick meconium was more in abnormal CTG group, but PROM, thick meconium and induction of labor was less in abnormal CTG group with UV pulsation although not significant.

The rate of cesarean was significantly more in abnormal CTG group in comparison with normal CTG group but the rate of cesarean was similar in abnormal CTG group with or without UV pulsation (83. 3% vs 90%).

Detection of fetal hypoxia during labor is based on CTG in our delivery unit, (considering clinical condition of mother and fetus). CTG has a risk of overestimation of fetal hypoxia which may lead to unnecessary operative intervention. We considered using adjunctive technique during labor would result in lower cesarean delivery rate or aid in earlier detection of fetal distress.

The cesarean delivery rate was similar in pathologic CTG group with or with out pulsating UV. The rate of Apgar score <7 was more in pulsating UV group than non pulsation group, although not significant. After exclusion of LBW fetuses no difference was seen.

NICU admission was more in pulsating UV group than non pulsating group (although not significant), but when LBW fetuses were excluded NICU admission was similar in both group, there fore it seems UV Doppler exam could be more efficient in detecting hypoxia during labor in LBW babies.

As a conclusion pulsation in UV is seen in 23% of fetuses with abnormal CTG during labor. Venous Doppler study could be a useful tool for fetal surveillance in LBW fetuses during labor.

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