

# Fetal Cerebral Monitoring During the Second Stage of Labor

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*The moment of perinatal hypoxic injury is still difficult to be identified by current monitoring techniques. Recent studies highlights that the effectiveness of therapy in hypoxic ischemic encephalopathy, such as therapeutic hypothermia and antioxidant agents, is determined by the time elapsed from the moment of injury to the beginning of intervention. Twenty six term newborns were analyzed, 13 from vaginal delivery and 13 extracted by cesarean section. The group selection criteria were: term pregnancy (gestation age  $\geq$  37 weeks), normal labor, cranial presentation, without fetal malformations and normal neonatal transition. We believe that additional fetal brain monitoring (NIRS and/or aEEG) can predict fetal brain events due to severe prepartum acidosis. Intrauterine fetal cerebral saturation is at the lower limit of postnatal neonatal cerebral saturation. FTOE is maximum during vaginal or cesarian section delivery compared to those in the first 10 min of life. The mode of delivery does not significantly affect FTOE or placental oxygen blood supply. Because the hypoxic - ischemic injury has occurred during late decelerations, consider it necessary to identify hypoxic markers prior to detection of this typ of FHR .*

**Keywords:** second stage of labor, fetal cerebral oxygen saturation ( $rSO_2$ ), fractional cerebral tissue oxygen extraction (cFTOE), fetal hypoxia, newborn

The moment of perinatal hypoxic injury is still difficult to be identified by current monitoring techniques. Recent studies highlights that the effectiveness of therapy in hypoxic ischemic encephalopathy, such as therapeutic hypothermia and antioxidant agents, is determined by the time elapsed from the moment of injury to the beginning of intervention [1,2]. Animal studies had established the optimal time to initiate therapy within the first 6 h of hypoxic ischemic injury [3]. The applicability within the first 6 hours in human studies has been extrapolated but often this does not coincide with the moment of injury. Near Infrared Spectroscopy (NIRS) and amplitude-integrated electroencephalography (aEEG) monitoring during neonatal transition have the potential to bring informations about fetal distress and help early tratment, but often the results are inconclusive [4]. An explanation for these results could be the late initiation of terapy in late-stage post injury even in the presence of immediate postnatal monitoring. To sum up, we believe that additional fetal brain monitoring (NIRS and/or aEEG) can predict fetal brain events due to severe prepartum acidosis.

## Experimental part

### Material and method

Twenty six term newborns were analyzed, 13 from vaginal delivery and 13 extracted by cesarean section. The group selection criteria were: term pregnancy (gestation age  $\geq$  37 weeks), normal labor, cranial presentation, without fetal malformations and normal neonatal transition. Vaginal delivered newborns were monitorised for cerebral saturation during delivery and immediately postpartum. At those extracted by cesarean section the cerebral oxygenation was evaluated straight away. The blood gas analysis was performed in all patients from the sample within the umbilical artery. Cerebral blood oxygenation was evaluated by  $SO_2$  and  $PaO_2$ .

NIRS measurements were performed with INVOS 5 100 C (Somametics, Covidien) by placing the sensor consisting

of one diode and two optodes located at 3.5 cm and respectively 4 cm from the diode.

The neonatal sensor of NIRS has sizes of 50 x 20 x 1.5 mm, its sterile, disposable and placed under sterile conditions by an obstetrician at pregnant woman after the amniotic membrane rupture and the dilation of cervix  $\geq$  5 cm. The sensor is attached to the fetal scalp by the pressure of maternal tissues adjacent to the fetal head. Due to the NIRS penetration to the cerebral cortex, the exact position of the sensor is not critical, but the face and ears should be avoided. This should be checked by the obstetrician. Recorded NIRS values has been individually analyzed and only those with full SSI (Signal Strength Indicator) signal has been validated. In all the cases studied, prior to antenatal and postnatal measurements, the written and verbal agreement was obtained with the informed consent of each pregnant woman. There were no reported difficulties in placing the sensors on the fetal scalp. Pregnant women did not report discomfort during fetal NIRS monitoring. Procedures required for this monitoring have not interfered with routine care required during labor or delivery. No lesions due to transvaginal insertion, fetal head application, or removal of the NIRS sensor occurred in both maternal tissues and fetal tissues. Also, the light emitted by NIRS is below the standard internationally approved safety limits for exposure to LASER light of the eyes or tegument. Thus, NIRS monitoring is free of fetal tissue damage.

### Difficulties in NIRS monitoring at the fetus and potential causes of errors

Inevitably, in the case of direct fetal monitoring and especially during contractions, we will encounter difficulties due to indirect and limited access, the movement of sensors applied to the fetal scalp and the important pressures exerted during the contractions of the uterus. In our study, they cause measurement difficulties and increased incidence of artifacts. An important source

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All the authors had the same contribution to this article

of this technical failure is the weak contact between the optodes and the fetal scalp due to either rapid head descent or maternal position change. A weak contact can be detected by observing steep changes in the value of the SSI signal. Regarding the accuracy of the displayed NIRS value, it may vary by the distance between optodes and the diode can be read as variations of the chromophores (OxiHb and HHb) or the pressure exerted on them. The NIRS-INVOS program is set to provide a constant wavelength. Changing diode and optode distances may occur during contractions. INVOS 5100 C can not detect and attenuate very small and gradual changes in the position of the optodes that are accompanied and the change in the total optical wavelength. Some researchers have observed in a dead fetus similar changes in fetal cerebral  $rSO_2$  and dynamics to that of a living fetus [5]. These changes are explained by the optodes motion artifacts and / or the pressure on them during contractions, but the use of newer NIRS techniques allows the attenuation of variations. NIRS with modulated intensity highlights variations in wavelengths during contractions of up to 6%, which with current techniques become insignificant to be sources of error [6,7].

## Results and discussions

A prospective observer study has been conducted in the labor room in pregnant women without associated pathology. We studied 13 human fetuses at term in the second stage of labor, with an average of 30 min antenatal (10-100 min antenatal) in the Polizu Clinical Hospital. Their anthropometric data after birth were (table 1): mean age of gestation (GA) 39 weeks, mean BW 3474 g, equal proportions between male and female. The evaluation of

the acid-base balance at birth (table 2.) in the umbilical artery from the umbilical cord ( $pH = 7.22$ ,  $BE = -8.2$  mmol / L,  $pO_2 = 20$  mmHg,  $pCO_2 = 53$  mmHg, lactate = 4.9 mg / dL) shows the absence of severe cases of fetal suffering in the analyzed cases. In one case, the birth was instrumentalised by vacuum aspiration, and the blood gas analysis revealed moderate acidosis ( $pH = 7.08$ ,  $BE = -14.6$  mmol / L, lactate = 7.5 mmol / L). In one case, Nitrous oxide ( $N_2O$ ) mixed with  $O_2$  - an inhaled anesthetic gas consisting of two equal proportions of  $N_2O$  and  $O_2$ , was used as a method of maternal analgesia in labor, at which fetal brain  $rSO_2$  values were observed during and between contractions, and they were similar to cases without maternal analgesia.

During stage II contractions, cerebral oxygenation was on average 15.8 (15-18), and between contractions  $rSO_2$  rises sharply (1-3 min) to a peak of 53.3 (42-56). During delivery, fetal cerebral  $rSO_2$  decreases to the minimum values of 16 (15-19) similar to the values measured during contractions.

At rest, the intrauterine pressure is approximately 10 mmHg, which is also transmitted to the intervillous space (10 mmHg). During contraction, intramyometrial pressure (IMP) increases and flow through the spiral arteries decreases from 70 mmHg to zero when the intrauterine pressure exceeds 35 mmHg [8,9]. With the cessation of contraction the flow through the uterine spiral arteries, it resumes and returns to normal at the end of the contraction [9,10]. This is reflected in the fetal cerebral circulation by a decreased fetal  $rSO_2$  at rest to an average of 53.3% (42-62%), to a value of 16.3% (15-21%) during the complete contraction, and returning to the initial value at the end of contraction (fig.1).

**Table 1**  
THE GROUP'S CHARACTERISTICS

	Vaginal Delivery (no. 13)	C-section delivery (no. 13)	$p < 0.05$
Gestational Age (weeks)	39 (37-40)	38 (37-40)	n.s
Birth Weight (g)	3474 (2900-3900)	3270 (2900-4300)	n.s
Male/Female	6/7	8/5	n.s
Apgar Score (SA)	8.6(6-9)	8.4(7-9)	n. s.

**Table 2**  
ACID-BASE BALANCE

Characteristic	Vaginal Delivery (no. 13)	C-section delivery (no. 13)	$p < 0.05$
pH	7.22 (7.08-7.34)	7.34 (7.17-7.45)	0.039
BE (mmol/L)	-8.2 ((-1.5) - (-14.6))	-2.6 (-7-0.7)	0.005
$pO_2$ (mmHg)	18.4 (8-29)	21.1 (10.5-28.1)	0.24
$pCO_2$ (mmHg)	54.2 (39-65)	43.6 (32.7-62.9)	0.043
Lactat (mmol/L)	5.0 (3-7.5)	2.4 (0.7-4.6)	0.013

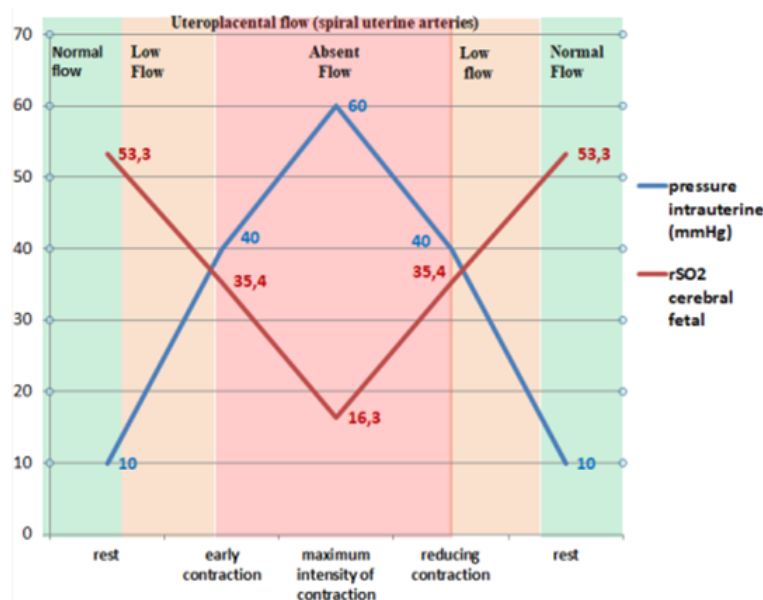


Fig.1. The dynamics of fetal brain oxygenation during uterine contractions

	Vaginal delivery (nr. 13)	Cesarian section (nr. 13)	p value	All patients
rSO <sub>2</sub> cerebral (%)	17.2 (15-19)	16.8 (15-30)	0.33	16.9 (15-30)
FTOE cerebral (%)	0.73 (0.61-0.75)	0.71 (0.33-0.78)	0.33	0.72 (0.57-0.81)
ctO <sub>2</sub> (mL/dL)	7.9 (2-12.2)	7.01 (1.6-13.6)	0.36	(1.6-13.6) 7.5
SaO <sub>2</sub> (%)	35.1 (10.8-60)	40.0% (8.6-69.1)	0.18	37.2 (8.6-69.1)

**Table 3**  
CEREBRAL OXYGENATION AND  
CEREBRAL FRACTIONAL TISSUE  
OXYGEN EXTRACTION (cFTOE) IN  
VAGINAL VERSUS CESARIAN SECTION  
DELIVERY

It is noted that intrauterine fetal cerebral rSO<sub>2</sub> between contraction (at rest) is approximately equal to the normal range of postnatal neonatal cerebral saturation. The flow of the spinal arteries that supply the placenta is stopped during maximum contraction (intraamniotic pressure 40-60 mmHg) similar to what happens during the delivery, a confirmed phenomenon by the rSO<sub>2</sub> that has a minimum value of 16.3% (15-21%) during contraction, delivery and immediately postpartum. Fetal cerebral saturation (rSO<sub>2</sub>) is mainly determined by cerebral blood flow and oxygen intake.

Special vascular organization in the uterine wall with the perpendicular disposition of the spiral arteries and parallel of the veins, facilitates vein closure during contraction preventing the extortion of blood from the intervillous space [8]. Thus, even at intrauterine pressures above 60 mmHg, the diastolic notch at the level of the umbilical vessels does not occur, and their resistance indices remain stable in labor and allow a permanent fetoplacental exchange [11]. We can say that rSO<sub>2</sub> decreases based on the reduced oxygen intake to the fetus that is interrupted with each contraction, but this is a common phenomenon for a normal fetus (such as breathing interruption for a few seconds). If contractions become too frequent, they can lower the fetal oxygen supply needed for the following contractions and can put the fetus in danger of hypoxia. The same risk of hypoxia is experienced by fetuses who already have significant pathologies that affect their oxygen resources and will not normally tolerate frequency, duration or intensity of normal contractions.

A group of 13 neonates extracted via C-section (table 2) has been studied comparatively with the above group, and in the last one rSO<sub>2</sub> was been measured immediately after cesarean delivery and after premature clamping of umbilical cord. This group includes clinically-assessed newborns with good intrauterine and postnatal development and by the blood gas analysis from the arterial vessel of the umbilical cord (table 2). The anthropometric and biochemical characteristics are without significant differences between the two groups. Thus, it was observed that cerebral oxygenation in the first minute of life, in this category of newborns, has values of 16.8 (15-30) similar to the values measured in delivery during natural birth (table 3). This suggests that cesarean section procedures, such as uterine incision and fetal pull-out procedures have fetal cerebral haemodynamic effects equivalent to those during a contraction during natural delivery. Some studies show that in the case of extraction by cesarean with labor the pH is similar (pH = 7.25) but pCO<sub>2</sub> is higher and pO<sub>2</sub> is lower compared to natural birth [12]. Repeated contractions in normal labor prior to cesarean delivery result in significant metabolic stress on the fetus. In the absence of labor prior to cesarean extractions, the blood gas analysis in the umbilical artery may be close to those of the mother (higher pH, pO<sub>2</sub>, BE and bicarbonate, and

respectively lower pCO<sub>2</sub>) [13,14]. Our results show significantly higher pH in the case of C-sections and this is due to the absence of labor. In the case of natural births the right shift of the O<sub>2</sub>Hb dissociation curve, the low pH works by lowering the affinity of the oxygen allowing it to increase its release to the tissue or cerebral level during metabolic acid stress during contractions. Extraction time is too short for biochemical changes to occur, so biochemical parameters (e.g. pH) remain unchanged but cerebral hemodynamic changes occur that explain similar values of cerebral oxygenation during delivery or cesarian section extraction although pH is different in the two conditions. Cerebral fractional cerebral tissue oxygen extraction (cFTOE) is slightly increased in the case of natural births 0.65 vs. 0.62 but insignificantly statistically. The ctO<sub>2</sub> does not statistically influence the FTOE, which indicates the placental and fetal blood oxygen reserve are sufficient for normal labor or cesarian section (table 3). Therefore the mode of delivery does not influence cerebral blood flow status.

When measuring fetal cerebral rcSO<sub>2</sub> in the second stage of labor, in the case of vaginal delivery, the term newborns showed a gradual decrease over the last 30 minutes until delivery with a minimum in delivery, and a gradual increase after birth in the first 10 min. Previous studies show good protection of the fetal head during contractions and deliveries by direct measurements of intracranial and intraamniotic pressure on human fetuses with hydrocephalus incompatible with life [15,16], and indirect measurements like cerebral flow, cerebral oxygenation and cerebral electrical activity of the fetus. Thereby in stage II of labor, the cerebral blood volume increases [16] and the EEG route is maintained [17,18] within normal limits during contractions or delivery. This decrease of fetal rcSO<sub>2</sub> in vaginal delivery is normal and is not caused by mechanical factors of fetal cephalic compression or the effects of acidic and basic biochemical factors on cerebral flow. In conclusion, it remains to be studied utero-placental oxygen intake using fetal pulse oximetry and cerebral metabolism.

There is little data to show the link between FHR (fetal heart rate) and cerebral fetal oxygenation. From the three main types of FHR deceleration (early, variable and late), late ones have the greatest clinical significance in predicting hypoxia and fetal acidosis. Previous stage II labor studies shows that the O<sub>2</sub>Hb, deoxyHb, and total haemoglobin dynamics are the same during contraction in the two groups studied (normal-control and late-deceleration contractions). However, in the time after the contraction there was a significant decrease in O<sub>2</sub>Hb and a important increase in deoxyhaemoglobine in the second group, as well as the collapse of FHR [19,20]. This increase in deoxyHb associated with low FHR is believed to be due to a deoxygenated placental blood bolus coming from the umbilical vein [21].



## Conclusions

Cerebral oxygenation decreases during contractions due to low oxygen in the utero-placental blood supply, while placental-fetal perfusion is permanently maintained. This is a physiological phenomenon of fetal tissue hypoxic preconditioning to support delivery. Under natural labor conditions, placental oxygen supplies during contraction are sufficient to prevent anaerobic metabolism and the occurrence of lactic (metabolic) acidosis.

Intrauterine fetal cerebral saturation is at the lower limit of postnatal neonatal cerebral saturation. FTOE is maximum during vaginal or cesarian section delivery compared to those in the first 10 min of life. The mode of delivery does not significantly affect FTOE or placental oxygen blood supply.

Because the hypoxic - ischemic injury has occurred during late decelerations, consider it necessary to identify hypoxic markers prior to detection of this type of FHR .

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