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ORIGINAL ARTICLE

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Evaluation of fetal intrapartum hypoxia by middle cerebral and umbilical artery Doppler velocimetry with simultaneous cardiotocography and pulse oximetry

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Abstract *Objective:* To investigate fetal circulation at different stages of hypoxia during labor, and to study blood flow alterations in the brain and peripheral tissues, through simultaneous use of three non-invasive techniques. *Materials and methods:* Ninety two pregnant women between 38 and 41 weeks of gestation, comparable for maternal age and parity, were simultaneously monitored with cardiotocography (CTG), continuous fetal pulse oximetry and Doppler ultrasonography during the first stage of labor. In 70 cases evaluation was successful, and useful data was obtained. Doppler waveforms were obtained before and during abnormal CTG patterns, of both the umbilical (UA) and middle cerebral artery (MCA) to measure the pulsatility index (PI), resistance index (RI), and flow velocity integral (FVI). The study population was divided in three groups, according to CTG and fetal pulse oximetry tracings: 20 term fetuses with normal CTG patterns and oxygen saturation ($FSPO_2$) values >40%, 30 term fetuses with abnormal CTG patterns and $FSPO_2$ values between 30 and 40%, and 20 fetuses with abnormal CTG patterns and $FSPO_2$ values <30% for a time up to 2 min. These were studied and peripartum outcomes were compared. *Results:* Redistribution of blood flow was noted at $FSPO_2$ values of 37%, in all groups. In the presence of reduced oxygen saturation (near to or below 30%), MCA Doppler showed significantly lower PI (1.06±0.33 vs.0.74±0.39, p=0.03) and RI (0.59±0.14 vs. 0.44±0.14, p=0.03), while that of the UA showed mildly higher resistance indices (0.98±0.14 vs. 1.28±0.50, p=0.01 and 0.57±0.12 vs. 0.79±0.24, p=0.004, respectively). When an oxygen saturation value of <30% was maintained for greater than 2 min, MCA Doppler indices reversed, likely indicating morbid fetal hypoxia. Differences in fetal outcomes between groups correlated with Doppler and pulse oximetry tracings. *Conclusions:* During active labor the fetus maintains oxygen supply to the brain by redistributing blood flow. In cases of hypoxia this is feasible for only 2 min. We note a strong correlation between fetal pulse oximetry, Doppler velocimetry of the MCA and UA, and fetal morbidity.

Keywords Brain-sparing effect · Middle cerebral artery · Doppler ultrasonography · Fetal pulse oximetry

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Introduction

During labor, uterine contractions may cause compression of uterine perforating arteries or of the umbilical cord, and thereby an acute reduction in uteroplacental or umbilical circulation respectively. Both can lead to fetal hypoxia. Carditocography (CTG) is still the most widely used method for the assessment of fetal well-being during labor, although its specificity for indicating fetal compromise is relatively low [13]. Non-reassuring fetal heart rate patterns lead to operative interventions. Postpartum fetal acid-base status often proves these latter to have been unnecessary.

In the search for continuous and non-invasive methods for monitoring fetal condition that are more specific for fetal distress, reflectance pulse oximetry and Doppler ultrasonography of fetal hemodynamics offer great promise.

Fetal pulse oximetry is a new technique, still undergoing investigation [2], which allows intrapartum assessment of fetal oxygen saturation ($FSPO_2$) and has been shown to improve the assessment of fetal well-being [6, 15]. Readings of fetal SPO_2 at delivery appear to correlate with cord blood SPO_2, and dynamic changes in fetal oxygen levels can be identified using this technique [5, 11, 16].

Intrapartum Doppler velocimetry of the fetal middle cerebral (MCA) and umbilical artery (UA) allows detec-

tion of changes of the fetal circulation, in cases of fetal oxygen stress. The efficacy of monitoring fetal antepartum Doppler velocimetry for predicting adverse perinatal outcome in complicated pregnancies has been widely investigated [12, 26]. Positive correlations between cerebral Doppler data and fetal hypoxia, in cases with absent end diastolic flow in the aorta or the umbilical artery, have been demonstrated throughout pregnancy [9, 17].

The most essential role an obstetrician can play is to predict fetal distress on one hand, and to avoid unnecessary operative intervention on the other. In the present study we simultaneously compare CTG, pulse oximetry, and MCA and UA Doppler during labor. Very few studies have been performed which take fetal oxygen saturation into account [3, 11] and only one other has used these three methods simultaneously [23]. The aim of the present study is to evaluate the pathophysiology of "brain sparing effect" in an attempt to understand the physiologic changes which occur with low fetal oxygenation and/or fetal heart rate abnormalities during labor via Doppler velocimetry, and to compare the latter to CTG and fetal pulse oximetry tracings.

Materials and methods

With the approval of the institutional ethic committee, 800 Doppler flow measurements of the fetal MCA and UA were performed in 92 pregnant women between 38 and 41 weeks gestation. Study subjects were recruited from women admitted to the intrapartum unit of the 2nd Department of Obstetrics and Gynecology of the University of Athens, "Aretaieion" Hospital, between January 1999 and December 2001. Admission criteria included: cephalic fetal presentation, absence of maternal pathology (such as hypertension, gestational diabetes, etc), absence of chromosomal abnormalities or major malformations, and gestational age having been established by 1st trimester ultrasound. No medications were given to women except iron, folic acid, and vitamins.

Our study population was divided in three groups, according to CTG and $FSPO_2$ measurements:

- Group A, which was the control group, consisted of 20 subjects with normal CTG patterns and $FSPO_2$ values >40%
- Group B consisted of 30 subjects with abnormal CTG patterns and $FSPO_2$ >30%, group C consisted of 20 subjects with abnormal CTG tracings and $FSPO_2$ <30% for a periods of up to 2 min

Abnormal CTG patterns were defined, according to the American College of Obstetricians and Gynecologists criteria, as those having at least one of the following: late decelerations, decreased baseline variability (beat-to-beat variability <5 bpm for 20 min), severe variable decelerations, moderate or severe bradycardia (<100 bpm for 3 min) and tachycardia (baseline rate >160 bpm) [1].

All measurements were performed during the first stage of labor. Fetal condition was evaluated by continuous fetal heart rate monitoring (CTG), fetal pulse oximetry, and Doppler ultrasonography of the fetal MCA and UA. Signed informed consent was obtained from the mother prior to the procedure. Once the cervical dilatation was 3 cm or more, and the head station was minus 2 or lower, fetal SO_2 monitoring was carried out using the Corometrics Oxicardiotocography series 120 (USA) with a Nellcor FS-14B fetal oxygen sensor. The sensor was placed on the fetal cheek or temple, facing the maternal spine. The sensor remained in place until delivery.

Fetal UA and MCA flow velocity array waveforms were obtained before and during abnormal CTG tracings. In group A, waveforms were obtained at random, before and during contractions, in order to have data for comparison. All measurements were used for analysis.

Waveforms were measured transabdominally by the same operator (C.S.), using duplex phased Doppler ultrasound. Doppler ultrasonographic studies were carried out with an Esaote dynamic image scanner (EsaoteMedica AU5, Florence, Italy). A 3.5-Mhz convex transducer was used (in order to minimize the angle-dependence of some indices) with a 2–3 mm sample volume, wall filter of 50 Hz, pulse repetition frequency 2.5 KHz and average velocity 83.43 cm/s.

Woman was placed in a semi-recumbent position and the transducer was moved to the base of the fetal head, in order to obtain the MCA waveform. Using color flow imaging, the middle cerebral artery could be seen as a major lateral branch in the circle of Willis, running anterolaterally towards the lateral edge of the orbit. The pulse Doppler sample gate was then placed on the proximal portion of this vessel to obtain flow velocity waveforms. UA Doppler waveforms were obtained from the fetal end of the cord, as they are significantly higher than those at the placental end [18]. The convex transducer was applied to the maternal abdomen with minimal pressure, in order to avoid the effect of fetal head compression on the cerebral flow impedance [24]. Measurements were performed in the absence of gross fetal body or breathing movements, and at similar heart rate in all groups (baseline values were 135±15 bpm and drops ranged from 70 to 110 bpm). Neither pain medications, nor epidural anesthesia were used in labor. No oxytocin was given, unless parturient entered the second stage of labor. Ringer's lactated was administered to all subjects at a mean of 1±0.6 l oxygen therapy (100% with a mask) was also given in the presence of abnormal CTG patterns.

S/D ratio, FVI (flow velocity integral-mean velocity of the waveform), RI (resistance index) and PI (pulsatility index) indices were measured.

Flow velocity indices were defined as: $PI = V_1 - V_0/V_{mean}$, $RI = V_0 - V_1/V_0$, FVI = mean velocity of the waveform and S/D = mean systolic/diastolic ratio, while V_o = peak systolic velocity and V_1 = end diastolic velocity. Fetal brain sparing effect was defined as: cerebroplacental (C/P) RI ratio (RI of MCA/UA)<1. We considered an FSPO2 of 30% as the cut-off point of intrapartum hypoxia and an emergency cesarean section was decided in cases where $FSpO_2$ values were below 30% for more than 2 min [3].

Fetal outcome data collected included 5th minute Apgar score, necessity of intubation of the neonate and its transfer to a special care unit, as well as pH and base excess values from the umbilical artery, taken immediately after delivery.

Problems in our study included mother's refusal to participate to the process, interference between the Doppler waves and the external receiver of GTG and oximeter, position of the fetus, maternal movements and breathing, uterine contractions and labor pain. Satisfactory data from all measurements were obtained in 70 cases.

Mean oxygenation values were 45±20% and mean duration of pulse oximetry monitoring was 43 min, with sensor contact achieved 96% of the time. Success in obtaining an MCA waveform was achieved in 80% of cases, while UA waveforms were obtained in 100% of trials.

Statistical analysis

Differences between means were tested using the Student's *t*-test and the Fisher exact test, as appropriate. All data were analyzed using the statistical package for Windows "Statistica". A probability value of <0.05 was considered statistically significant.

Results

Mean age of subjects of group A was 26.5 years (range 22–31), of group B 27 years (range 23–30) and of group C 28.5 years (range 24–32).

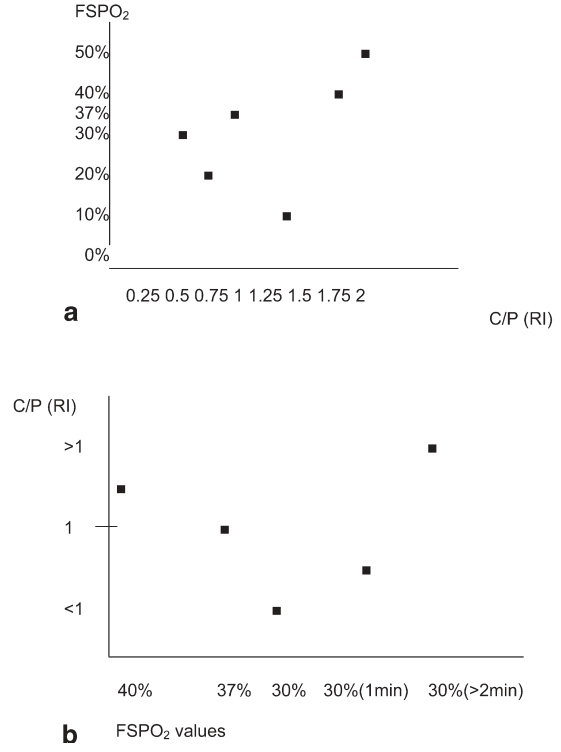

Fig. 1 **a** Relationship between cerebroplacental (*C/P*) resistance index (*RI*) ratio and oxygen saturation (*FSPO* $_2$) values during intrapartum hypoxic status. **b** Changes on C/P (RI) in $FSPO_2$ values near and below 30%

Isolated fetal incidents, such as transient or early decelerations lasting for up to 15 s, occurred in 76% of subjects studied and had no effect on fetal oxygenation. In those cases $FSpO_2$ values varied from 35 to 58%. Decelerations of longer duration were less common. Transient drops of $FSpO_2$ <30% were observed not only in group C, but in group A (34% of measurements) and group B (42% of measurements) as well, lasting for up to 20 s.

In groups B and C, the phenomenon of redistribution (brain sparing effect –C/P<1) was noted to start at $FSPO_2$ values of 37% (Fig. 1a) during uterine contraction. This effect proceeded from an initial fall in resistance indices in the MCA, to an increase in MCA flow velocity and a rise in UA resistance indices (Table 1). Changes in oxygen saturation values in both groups were observed when brain sparing effect lasted for over 10 s.

In Table 2 we present the variation in indices in the three groups during labor for each of the two vessels. In Figs. 2 and 3 we present scatter plots and mean values for the studied indices (PI, RI, FVI) in both vessels, in the study groups. These values are those obtained at the time of maximal change from baseline in each case.

In group B, PI in the MCA was significantly lower and RI in the UA was significantly higher during abnormal CTG tracings, compared to baseline heart rate values (Fig. 2).

In group C, both resistance indices in the MCA were significantly lower, while those in the UA were significantly higher during the abnormal heart rate (Fig. 3).

Table 1 Changes on fetal arteries indices during uterine contraction (brain-sparing effect). *RI* resistance index, *FVI* flow velocity integral, *PI* pulsatility index, *MCA* middle cerebral artery, *UA* umbilical artery

	MCA I→II	UA I→II
RI	↓↓	↑
FVI	↑↑	↓/–
PI	↓↓	–/↑

I: non-hypoxic status, II: pre- or hypoxic status

Table 2 Results of our measurements in the three groups (A, B, and C), before (*B*) and during (*S*) uterine contraction for the PI, RI and FVI indices of the two vessels

	Mean ± SD	*p*
APIUAB vs. APIUAS*	0.94±0.49 vs. 0.84±0.20	0.41
APIMCAB vs. APIMCAS*	1.21±0.37 vs. 1.22±0.42	0.92
ARIUAB vs. ARIUAS	0.58±0.25 vs. 0.52±0.08	0.29
ARIMCAB vs. ARIMCAS	0.63±0.19 vs. 0.62±0.12	0.94
AFVIUAB vs. AFVIUAS	0.26±0.10 vs. 0.27±0.09	0.82
AFVIMCAB vs. AFVIMCAS	0.29±0.31 vs. 0.17±0.06	0.10
BPIUAB vs. BPIUAS	0.93±0.31 vs. 0.85±0.07	0.52
BPIMCAB vs. BPIMCAS	1.26±0.26 vs. 1.00±0.21	0.02**
BRIUAB vs. BRIUAS	0.46±0.12 vs. 0.59±0.05	0.02**
BRIMCAB vs. BRIMCAS	0.57±0.16 vs. 0.58±0.14	0.93
BFVIUAB vs. BFVIUAS	0.42±0.31vs. 0.37±0.32	0.71
BFVIMCAB vs. BFVIMCAS	0.30±0.25 vs. 0.30±0.33	0.99
CPIUAB vs. CPIUAS	0.98±0.14 vs. 1.28±0.50	0.01**
CPIMCAB vs. CPIMCAS	1.06±0.33 vs. 0.74±0.39	0.03**
CRIUAB vs. CRIUAS	0.57±0.12 vs. 0.79±0.24	0.004**
CRIMCAB vs. CRIMCAS	0.59±0.14 vs. 0.44±0.14	0.03**
CFVIUAB vs. CFVIUAS	0.23±0.08 vs. 0.18±0.05	0.11
CFVIMCAB vs. CFVIMCAS	0.28±0.27 vs. 0.25±0.05	0.74

*APIUAB vs. APIUAS = PI of UA of group A before the uterine contraction vs. same index during the contraction
APIMCAB vs. APIMCAS = PI of MCA of group A before the uterine contraction vs. same index during the contraction
**p<0.05 is statistically significant

In group C, redistribution depended on the time that elapse with $FSPO_2$ <30%. When this drop lasted for 1 min, UA resistance indices started to rise (C/P<1). During the 2nd minute MCA velocity started to fall and resistance indices to rise (C/P>1), followed by a very high rise of UA FVI with no further alterations in resistance indices (Fig. 1b). In these cases, an emergency cesarean section was performed.

These differences in arterial flow indices were associated with differences in labor outcomes as presented in Table 3. Incidence of low Apgar scores (<7) at 5 min in group C was 40% as opposed to only 10% in group B and none in group A. The need for intubation or transfer of the neonate to a special care unit were also significantly higher in group C than in the other groups (10% vs. none respectively). Mean cord pH of newborns of group A was 7.42±0.18, of group B 7.36±0.20, and of group C 7.28±0.19, while mean base excess was −8.2±2.5, −9.5±3.2, and −10.1±3.1 respectively. Cord pH <7.2 and base excess ≥12 meq/l of the blood taken after delivery were 25% in group C, while in group B was only 3.33% and in group A none.

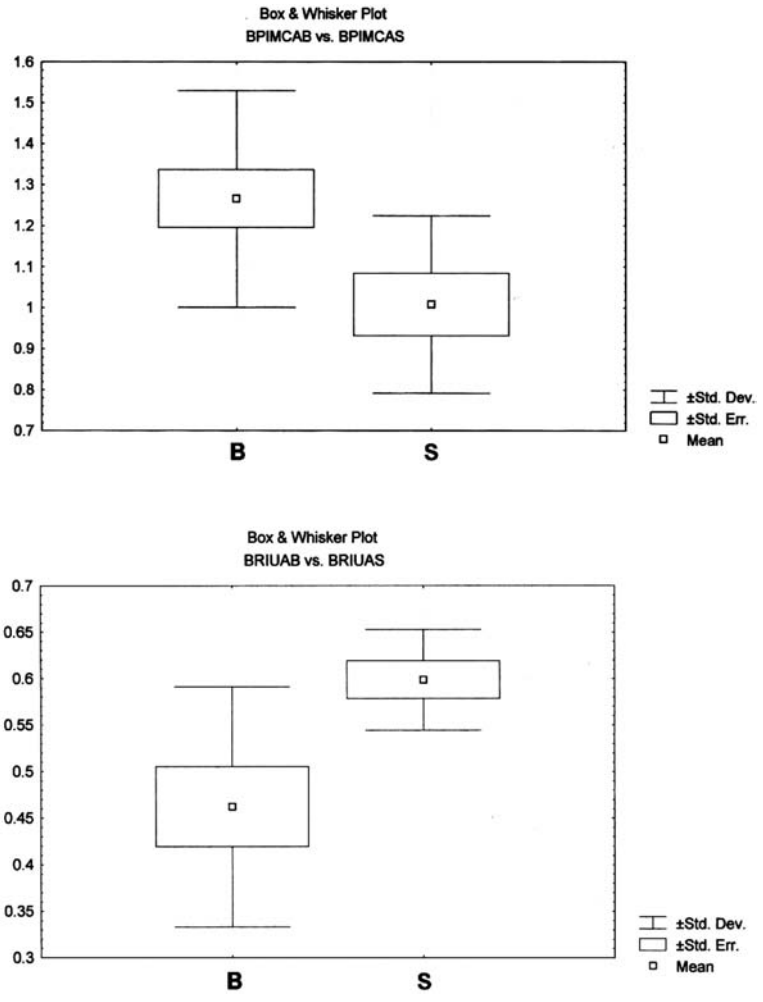

Fig. 2 Distributions and means middle cerebral artery (MCA) pulsatility index PI and umbilical artery (UA) RI indices of group B before (*B*) and during (*S*) cardiotocography (CTG) abnormalities

Table 3 Fetal outcome in the three groups

	Group A (n=20)	Group B (n=30)	Group C (n=20)
Apgar-score 5' <7	0	3 (10%)*	8 (40%)*
Intubation of the neonate—transfer to neonatal unit	0	0	2 (10%)*
Cord pH <7.2	0	1	5 (25%)*
Base excess ≥12	3.33%*		

*p<0.05 = statistically significant between groups

Discussion

Using a combination of CTG, continuous fetal pulse oximetry, and Doppler velocimetry, we attempted to evaluate the reaction of the fetal vessels to varying fetal arterial oxygen saturation during the first stage of labor. We found that brain-sparing effect began at an $FSPO_2$ value of 37%, and failed at $FSPO_2$ values below 30%. Varying reactions in the cerebral and umbilical vessels were also noted, depending on the degree of hypoxia. The present study is part of an ongoing research protocol in progress in our hospital under the heading of "Intrapartum Surveillance". As the optimal method for identifying fetal distress has not been determined,we present a preliminary investigation into a possibly superior new screening modality.

Fetal pulse oximetry is one of the most reliable methods for predicting fetal distress during labor [7]. We selected an oxygen saturation value of 30% as the limit, below which fetal compromise becomes increasingly likely [11, 19]. A time lapse of 2 min at such low arterial oxygen saturations was decided upon as the limit beyond which adverse fetal outcome became likely [3] and operative delivery should be performed.

Doppler sonographic measurements have been shown to predict fetal compromise throughout pregnancy [12], as well as during labor [10]. We decided specifically to study the UA and MCA, because they reflect most accurately fetal blood flow, and consequently oxygenation, of the periphery and the cerebrum respectively [17].

Few data have been published on dynamic changes of the fetal circulation during labor and only one study has been published using these three methods simultaneously [23]. This makes direct comparison of our results quite difficult.

Animal studies on the effects of hypoxia have provided evidence of redistribution of cardiac output to favor

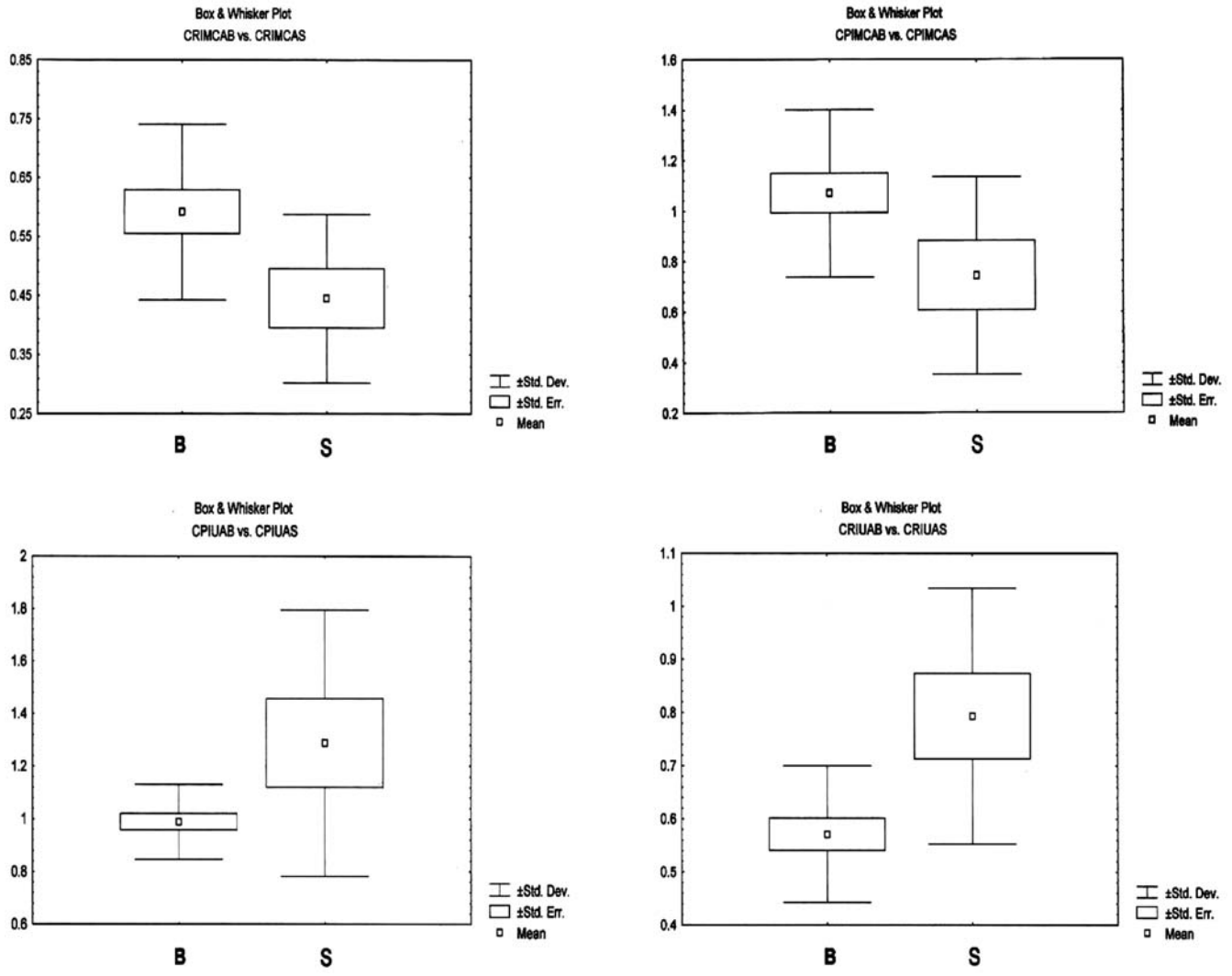

Fig. 3 Distributions and means MCA and UA PI, RI indices of group C before (*B*) and during (*S*) CTG abnormalities

perfusion of the fetal heart, adrenals and brain, at the expense of the musculoskeletal system, gut, and kidneys [22]. We know that this "brain sparing effect" is activated during intrauterine life in cases of chronic hypoxia, as during pregnancy induced fetomaternal pathology, and during the active phase of labor when the fetus experiences transient changes in blood oxygen saturation. In the present study, we observed that this dynamic process, defined by a cerebroplacental RI ratio of ≤ 1 [8], starts at an oxygen saturation value of 37%. This active vasomotor response, leads to cerebral vasodilatation, and peripheral vasoconstriction, effectively maintaining blood supply to the brain.

When oxygen saturation drops to 30% and remains at this level or lower for at least 2 min, this mechanism apparently reaches its limit. Beyond this we believe that the fetal body is no longer able to maintain increased peripheral vascular tone, likely secondary to the advent of anaerobic metabolism and a consequent drop in pH. The ensuing acidosis provokes uncontrollable vasodilatation and the breakdown of normal fetal autonomic physiology, including failure of this "brain sparing" reflex. In these situations, we observed a rise in cerebral impedance indices in parallel with a rather peculiar rise in the blood flow velocity of the umbilical artery, associated with decrease in UA resistance (C/P>1). A possible explanation for this rise of cerebral impedance indices might be the development of brain edema, something that has been speculated in antepartum studies [24]. The accompanying rise in blood flow velocity in the periphery might reflect a reaction to the changes in flow in the umbilical vessel.

Other authors, using CTG and pulse oximetry, have come to similar conclusions about cerebral blood flow as we present here. Some employing more limited techniques [3, 11], and others the identical combination of 3 methods that we present here [23]. According to Hecher et al., [12] when the compensatory mechanism of arterial blood flow redistribution has reached its limits, characteristic changes in venous flow velocity waveforms occur.

This phenomenon was not investigated in the present study.

With regards to the umbilical circulation, our results were quite different than those presented in other studies. We found increased resistance indices in the umbilical circulation as the oxygen saturation fell from 37% to levels near or below 30%. We theorize that this reflects a reaction by which the fetus keeps blood in its own vascular space, even if poorly oxygenated, and takes advantage of the higher affinity of fetal hemoglobin for oxygen. Contradictory opinions on this point [20, 23] are based on the differing relative cerebral and umbilical Doppler indices found by various investigators [14].

During our measurements, transient drops of $FSPO_2$ <30%, were noted in all groups (in approximately 35% of all cases studied), lasting up to 20 s, but not affecting fetal outcome parameters. Bloom has noticed similar drops in more than half of fetuses studied. He, however, concluded that $FSpO_2$ <30% was not a predictor of fetal compromise, and gave little value to the duration of saturation below 30%.

No fetal death occurred in the present study. With the exception of three cases, fetal outcome was never seriously impaired. The fetus tolerated drops in SPO_2 <30% for a period of 2 min, without an evident effect on well being. Apgar-scores <7 at the 5th minute, the need for intubation or transfer of the neonate to a special care unit, as well as cord pH <7.2 and base excess $\geq$12 meq/l at delivery, were more profound at $FSPO_2$ values below 30%, however, implying that pulse oximetry combined with elapsed hypoxic time is a good predictor of perinatal outcome. Similar conclusions are reached in some studies [4, 17, 23], while others differ [11, 21].

Given the data presented, we would also propose the combination of these three methods for diagnosing intrapartum fetal hypoxia, in cases of abnormal CTG tracings. We show here that this method can lead to recognition of fetal deterioration and predict fetal compromise earlier than other parameters do. Further investigation is needed, however, before this method might be recommended as the standard of care.

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