# UNIVERSITÀ DEGLI STUDI DI MILANO

SCUOLA DI DOTTORATO IN

SCIENZE FISIOPATOLOGICHE, NEUROPSICOBIOLOGICHE E ASSISTENZIALI DEL CICLO DELLA VITA

Ciclo XXV

DIPARTIMENTO

*Scienze della Salute*

TESI DI DOTTORATO DI RICERCA

# NOVEL APPROACH FOR INTERPRETAZTION OF FETAL HEART RATE IN THE SECOND STAGE OF LABOR

**MED/40**

Dottoranda

Mona Mansour

TUTOR

Prof.ssa: Anna Maria Marconi

DIRETTORE DELLA SCUOLA:

Prof.: Roberto L. Weinstein

Anno Accademico 2013/2014

# Novel approach for interpreting the fetal heart rate in the II stage of labor

## Introduction

Fetal surveillance in labor is performed to identify fetuses at risk of hypoxia with the aim to reduce neonatal morbidity and mortality by prompt intervention. Since its introduction in the 1960s, cardiotocography ( CTG) has been widely used for fetal surveillance during labor and normal and abnormal CTGs patterns in the first stage have been identified and classified (1-2-3).

On the contrary, the classification of CTG patterns in the second stage of labor has been often neglected from a cultural and clinical viewpoint because of its usual short duration and because considered the final stage after conclusion of the more prolonged high risk phase of cervical dilatation.

There have been some previous studies attempting to determine the characteristics of an abnormal second stage CTGs and its relation to neonatal status.

Fetal bradycardia, either persistent or progressive, has been shown to be strongly suggestive of fetal hypoxemia and Apgar score <7 at 5 minutes (4). Similarly, fetal tachycardia has been shown to be associated with low Apgar scores at five minutes and with neonatal acidemia (pH <7.10 ) in up to 20% of neonates (5). Also, absent CTG variability has been shown to be associated with an increased risk of neonatal acidemia , even in the presence of an otherwise normal CTG (6-7). The presence of bradycardia preceded by decreased baseline variability strongly predicts the development of pathologic neonatal acidemia thus indicating the need for prompt delivery. (8)

Accelerations were noted not to be commonly present in the second stage; decelerations were more common, and seen in more than 70 percent of second stage heart rate traces (5). Early decelerations did not appear to increase the risk of low 5 minute Apgar score and, as in the first stage, should be viewed as benign, regardless of baseline heart rate (4). Variable decelerations were much common and seen in approximately half of the second stage CTGs patterns. If the baseline heart rate was normal, mild variable decelerations appeared to have little influence on the incidence of low Apgar scores (4). However, variable decelerations with a drop in fetal rate of >70 beats per minute have been shown to be associated with an increased risk of metabolic acidosis (7). Late decelerations, although relatively uncommon in the second stage of labor, markedly increased the risk of low 5 minute Apgar scores, regardless of baseline heart rate (4).

Thus far, the only thoroughly classification of the fetal heart rate in the second stage of labor, is that performed by Melchior and Bernard (9) and modified by Piquard et al in 1988 (10) .

However, this classification was performed analyzing the tracings of a small number of pregnant women (145 primiparous and 89 multiparous) and has not been validated since then.
We performed this study to revise the classification of Piquard of the CTG patterns in the II stage of labor in a larger number of low risk pregnancies with the aim to identify patterns at high risk of neonatal acidemia at birth. In addition, to verify whether this classification might be applicable and useful also in high risk pregnancies we analyzed also tracings recorded in women with pre pregnancy or pregnancy related complications.

To do so, we analyzed the data with three main objectives:

- reanalysis of the classification of Piquard in a large number of nulliparous and multiparous women and in low and high risk pregnancies;
- relationship between the CTG tracing recorded in the II stage of labor to that recorded in the final 60 minutes of the I stage;
- analysis of the results according to the presence of neonatal academia defined as umbilical cord arterial pH $\leq$ 7.10 at the time of delivery.

**Patients and Methods**

This retrospective study was performed at the Department of Obstetrics and Gynecology of the San Paolo Hospital Medical School of Milan (Italy) from January 1ST 2012 until October 30st 2013.

Only women who delivered vaginally in this time period were eligible. Inclusion criteria were: singleton pregnancy, vertex presentation, at least 60 minutes of a technically interpretable tracing recorded in the final part of the first stage and preceding the second stage (first stage CTG), a technically interpretable tracing recorded continuously in the second stage, a second stage of labor lasting at least 10 minutes and umbilical arterial oxygenation and acid base balance measured at the time of delivery. Only live births with normal karyotype and without malformations were included.

Data collected regarded demographic and obstetric characteristics including maternal age, parity, pre pregnancy BMI ($kg/m^2$), smoking and information on the course of actual and previous pregnancies.

At the time of delivery we collected gestational age (which was determined by the last menstrual period and confirmed by an ultrasound examination performed within the 20$^{th}$ week), length of the second stage, type of labor (spontaneous, induced or augmented), mode of delivery (spontaneous or operative vaginal), neonatal sex and weight, Apgar score at 5 minutes. Growth centiles were calculated according to the Italian birth weight gestational age standards (11). Information on early neonatal morbidity (need for resuscitation and NICU admission) were also collected.

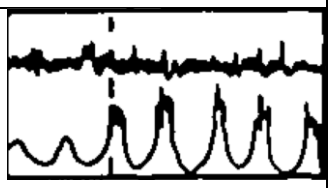
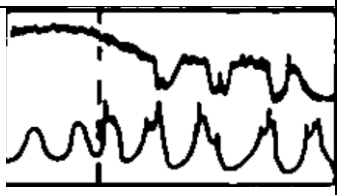
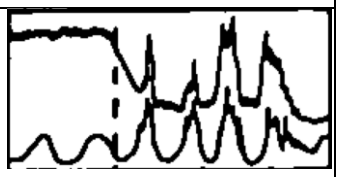
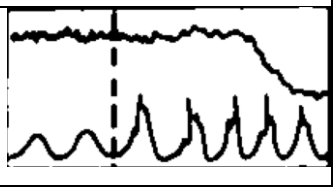
Fetal heart rate and uterine contractions were recorded using a universal cardiotocography device (Philips series 50 IP) with a paper speed set at 1cm/min.
CTGs were extracted prospectively and analyzed retrospectively blind to clinical information by a single reviewer trained in FHR interpretation.
Fetal heart rate tracings recorded continuously for last 60 minutes of the first stage of labor were analyzed according to the NICHD classification (Macones et al 2008) (12) but we simplified this classification for practical analysis as:

- category I (normal base line heart rate between 110-160, variability 6-25, presence of accelerations and absence of decelerations);
- category IIA (base line heart rate characterized by tachycardia)
- category IIB (repetitive variable decelerations)
- category IIC (variability ≤ 5 bpm )
- Category III not found because usually it prompted immediate caesarean delivery.

The beginning of the second stage was set with the beginning of the voluntary bearing-down efforts and midwives were instructed to report the time on the CTG paper.

The CTG patterns in the second stage of labor were analyzed according to the classification of Piquard et al (10) as depicted in Table 1.

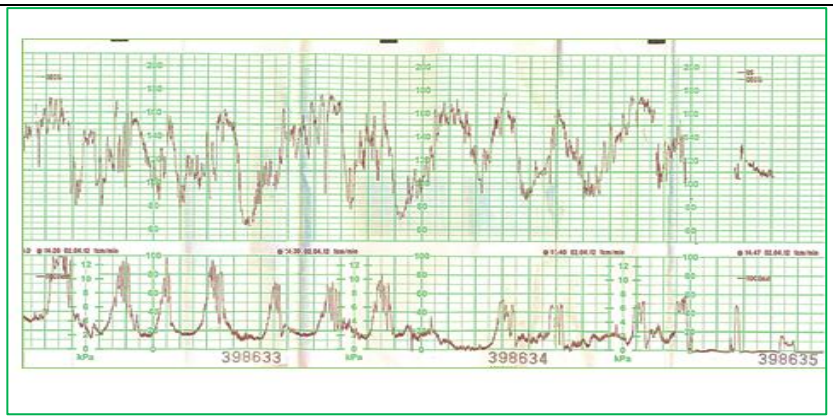
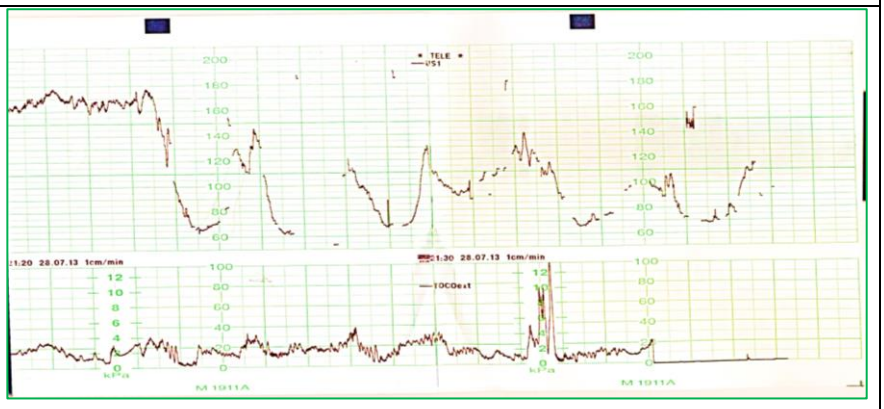
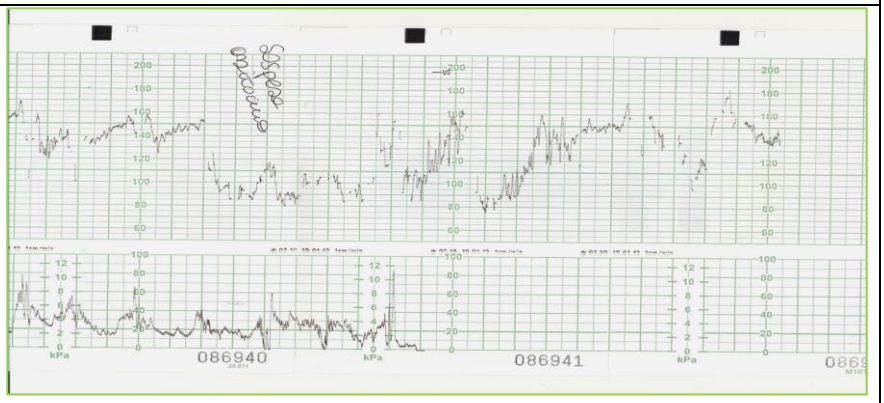
**Table 1**. The six types of CTG patterns in the classification of Piquard

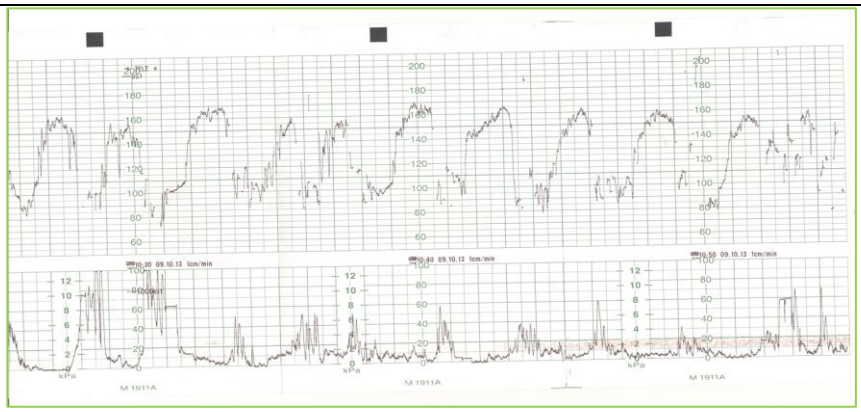
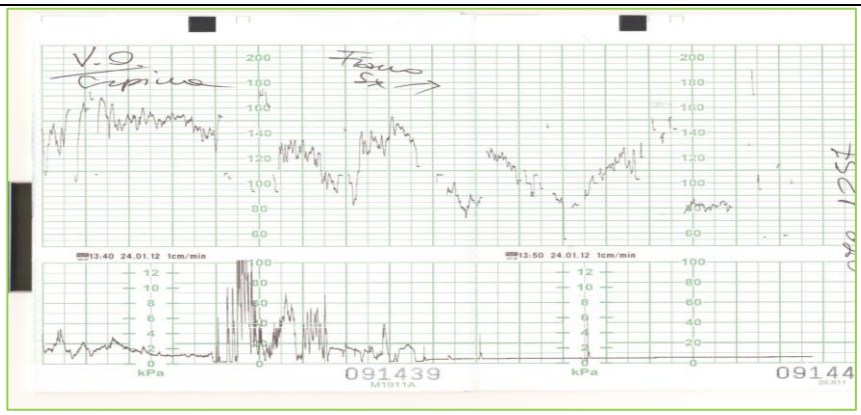
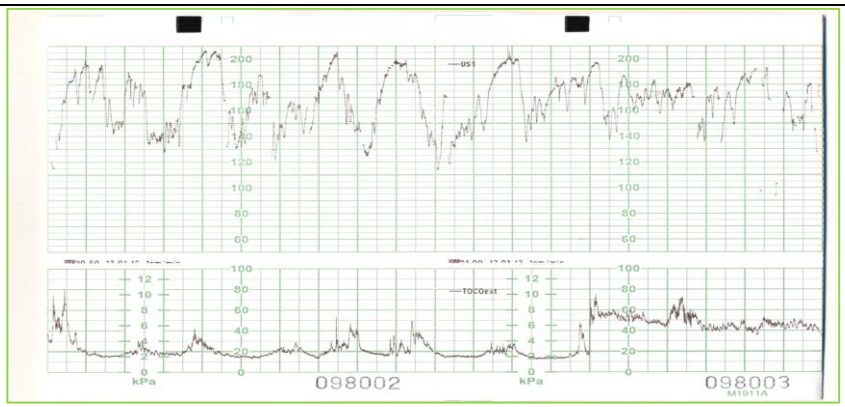
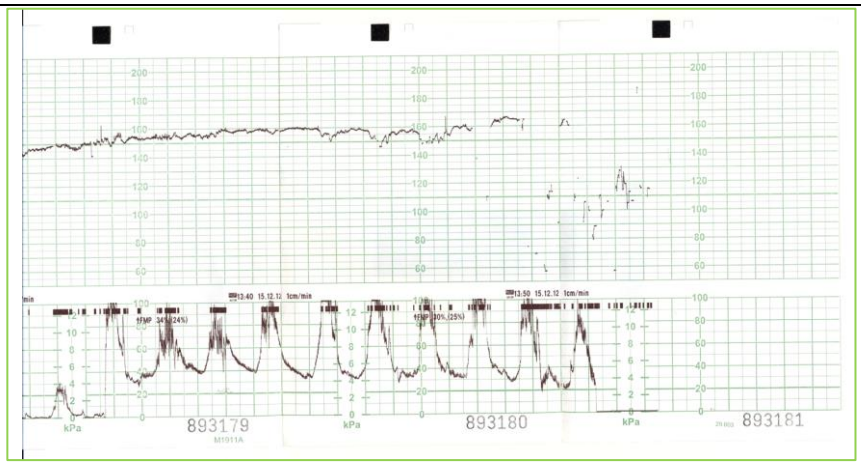
| Type | Description | |
|---|---|---|
| 0 | Stable heart rate |  |
| 1 | Deceleration at each contraction and maintenance of a normal baseline between contractions |  |
| 2A | Decreased baseline between 90-120 beats per minute, with decelerations often occurring during the contractions |  |
| 2B | Decreased baseline under 90 BPM, with frequent decreased variability |  |
| 3 | Severe fall of the baseline associated with marked accelerations during the contractions |  |
| 4 | Fall of the base line under 90 BPM, but occurring much later, practically at the extreme end of labor |  |

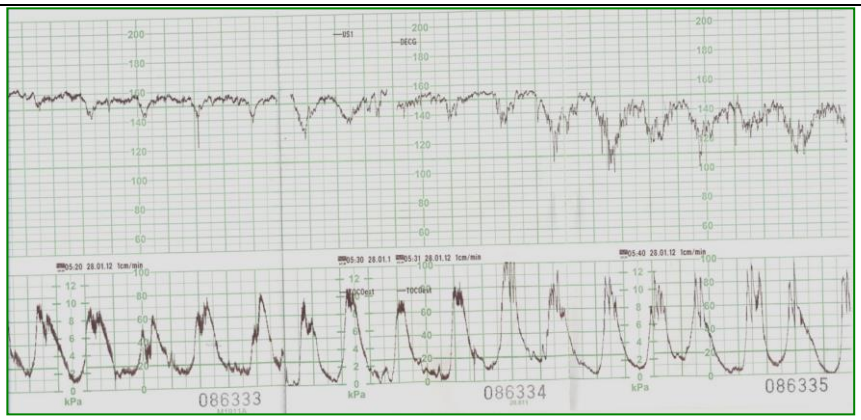
The vertical dotted line depicted in the figures represents the beginning of the II stage.

As the description of type 1 CTG is very little detailed in terms of the characteristics of the decelerations , we observed specific criteria of such decelerations which was present either alone or combined with other criteria as described and showed in (table 2)

Table 2:

| Description | |
|---|---|
| 1. repetitive decelerations (≥ 3 in10 minutes); |  |
| 2. deep decelerations (≥ 70 bpm); |  |
| 3. duration (>2 minutes); |  |

| | |
|---|---|
| 4. interval between decelerations (<2 min); |  |
| 5. lack of return to the baseline; |  |
| 6. baseline characterized by tachycardia (> 160 bpm) and repeated decelerations |  |
| 7. reduced variability (<6 bpm). |  |

| | |
|---|---|
| 8. reduced variability and reapeted decelerations |  |

Umbilical arterial blood was collected into heparinized syringes from a doubly clamped segment of the umbilical cord and complete Blood Gas Analysis was measured using a universal radiometer (ABL 700, Autocheck)

Non universal accordance has been established for the definition of acidemia . We had as a reference pH $\leq$ 7.10 (13)

**Statistical analysis**

Data are presented as mean ± SD. The significance of the differences between groups was calculated with the Student's t test or the chi-square test as appropriate. Odds ratios and corresponding 95% confidence intervals (lower confidence limit (LCI) - upper confidence limit (UCI)) were calculated through multiple logistic regression models. This model was used to investigate the independent contributions of obstetric factors to neonatal acidemia at birth.

Included factors were: pattern of CTG in the II stage; pattern of CTG in the I stage; parity; gestational age; presence of obstetric pathologies; duration of the II stage; induction/augmentation of labor; spontaneous vs vacuum assisted delivery; birth weight. Adjusting variables were selected from available literature on the topic and using the likelihood ratio test for model comparison. $P < 0.05$ was considered statistically significant. Analyses were performed using the R statistical software.

**Results**

Out of 2954 vaginal deliveries occurred in the study period, 2296 (77%) fulfilled the inclusion criteria and were included in the analysis.

Table 1 presents the clinical characteristics of the study patients. 1307 (56.9%) women were at their first delivery whereas 989 (43.1%) had had at least one previous vaginal delivery

**Table 3**

| | Nulliparous N= 1307 | Multiparous N= 989 | p |
|---|---|---|---|
| Maternal age, years | 29.9 ± 5.8 | 32.8 ± 5.4 | 0.001 |
| Maternal height, cm | 163.4 ± 6.3 | 162.9 ± 5.9 | 0.07 |
| Maternal pre pregnancy weight, kg | 60.8 ± 11.1 | 63 ± 12.7 | 0.001 |
| Maternal pre pregnancy body mass index, $kg/m^2$ | 22.8 ± 4 | 23.7 ± 4.4 | 0.001 |
| $<18.5\ kg/m^2$ | 114 (8.7) | 60 (6.1) | 0.02 |
| $\geq 30\ kg/m^2$ | 71 (5.4) | 92 (9.3) | 0.001 |
| Weight increase, kg | 12.7 ± 4.6 | 11.6 ± 4.7 | 0.001 |
| Gestational age, weeks | 39.4 ± 1.4 | 39.5 ± 1.2 | 0.8 |
| ≤34 weeks | 3 (0.2) | 1 (0.1) | 0.6 |
| Duration of II stage, minutes | 48 ± 34 | 26 ± 20.4 | 0.001 |
| Neonatal weight, grams | 3250 ± 426 | 3384 ± 437 | 0.001 |
| <2500 grams | 46 (3.5) | 20 (2.02) | 0.04 |
| Male fetus (%) | 668 (51.1) | 525 (53.1) | 0.3 |
| Apgar score at 5’ | 9.9 ± 0.4 | 9.9 ± 0.3 | 0.03 |
| Apgar score ≤7 at 5’ (%) | 8 (0.6) | 2 (0.2) | 0.2 |
| Umbilical artery pH | 7.255 ± 0.08 | 7.275 ± 0.08 | 0.001 |
| pH ≤7.10 (%) | 34 (2.6) | 14 (1.4) | 0.055 |
| Umbilical artery BD, mmol | -4.4 ± 3.6 | -3.2 ± 3.2 | 0.001 |
| BD ≥-12 (%) | 12 (0.9) | 3 (0.3) | 0.1 |
| Complicated pregnancy (%) | 130 (9.9) | 76 (7.7) | 0.07 |
| Smoker (%) | 126 (9.6) | 89 (9) | 0.6 |
| Vacuum extraction (%) | 212 (16.2) | 27 (2.7) | 0.001 |

As expected maternal age, pre pregnancy weight and BMI were significantly higher in multiparous ($p<0.001$); on the contrary, there was no difference in the rate of pregnancy complications and smoking habit.

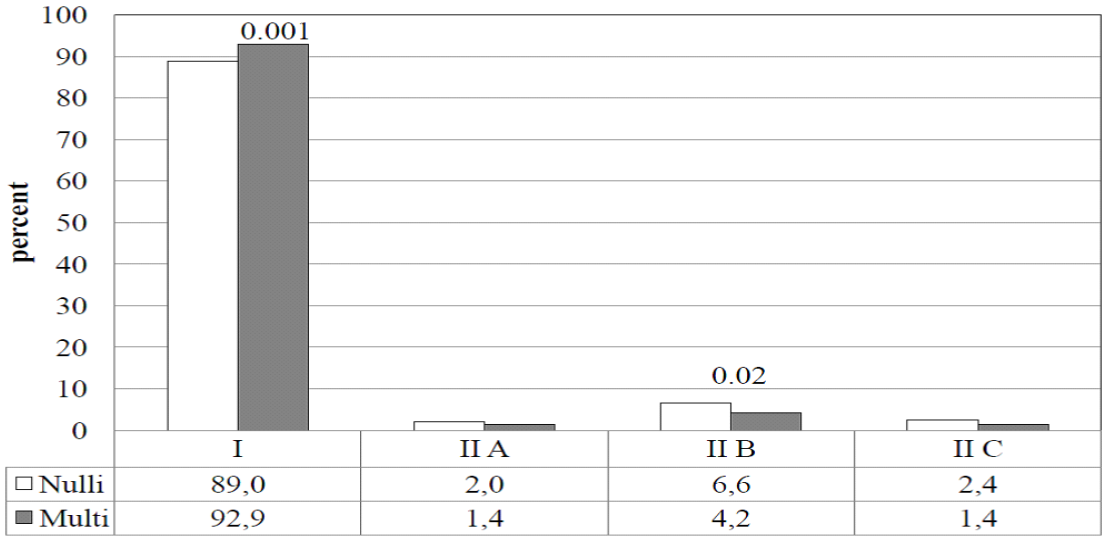
The duration of the second stage was almost doubled in nulliparous when compared to multiparous; operative vaginal delivery occurred in 16.2% nulliparous and 2.7% multiparous ($p<0.001$).

Neonatal weight was significantly higher in multiparous, although gestational age was similar in the two groups. At delivery 48 neonates had acidemia: of these, only 6 had umbilical arterial pH values <7.00 and they were all born from nulliparous: ten had a BD ≥-12 mmol/L.

Analysis of the CTG in the I stage of labour

The analysis of the FHR in the final 60 minutes of the first stage showed that 1163 (89%) nulliparous and 919 (92.9%) multiparous had a type I CTG ($p<0.001$); type II CTG was significantly more frequent in nulliparous (11% vs 7.1%; $p<0.001$) and this was mainly due to an increase frequency of type IIB tracings in this group (Figure 1).

**Figure 1**.

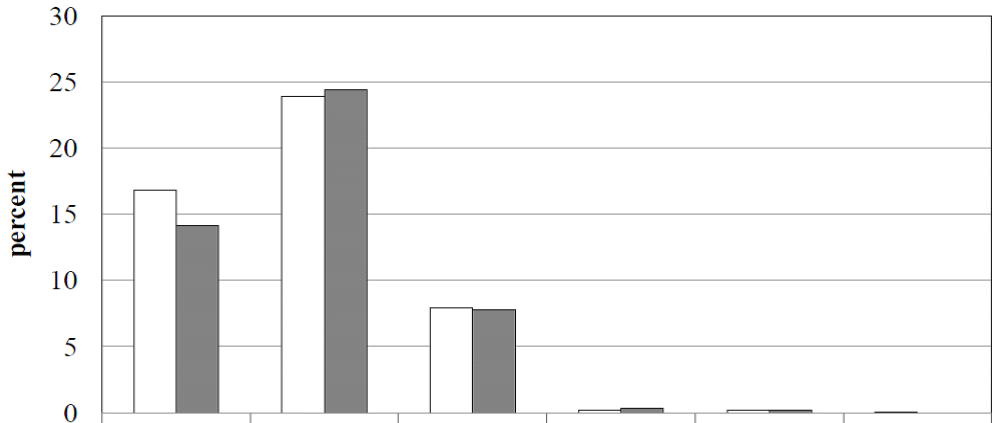



| | I | II A | II B | II C |
|---|---|---|---|---|
| Nulli | 89,0 | 2,0 | 6,6 | 2,4 |
| Multi | 92,9 | 1,4 | 4,2 | 1,4 |

Analysis of the FHR in the II stage of labor

When we analyzed the CTGs recorded in the II stage, surprisingly we found that slightly less than 50% of the tracings (49.2% in nulliparous and 46.7% in multiparous) could be classified according to the original six types of the FHR patterns proposed by Piquard et al. In addition, types 2B, 3 and 4 were very rare accounting only for 0.5% of tracings both in nulliparous and multiparous (Figure 2). The remaining tracings were actually a combination of patterns.

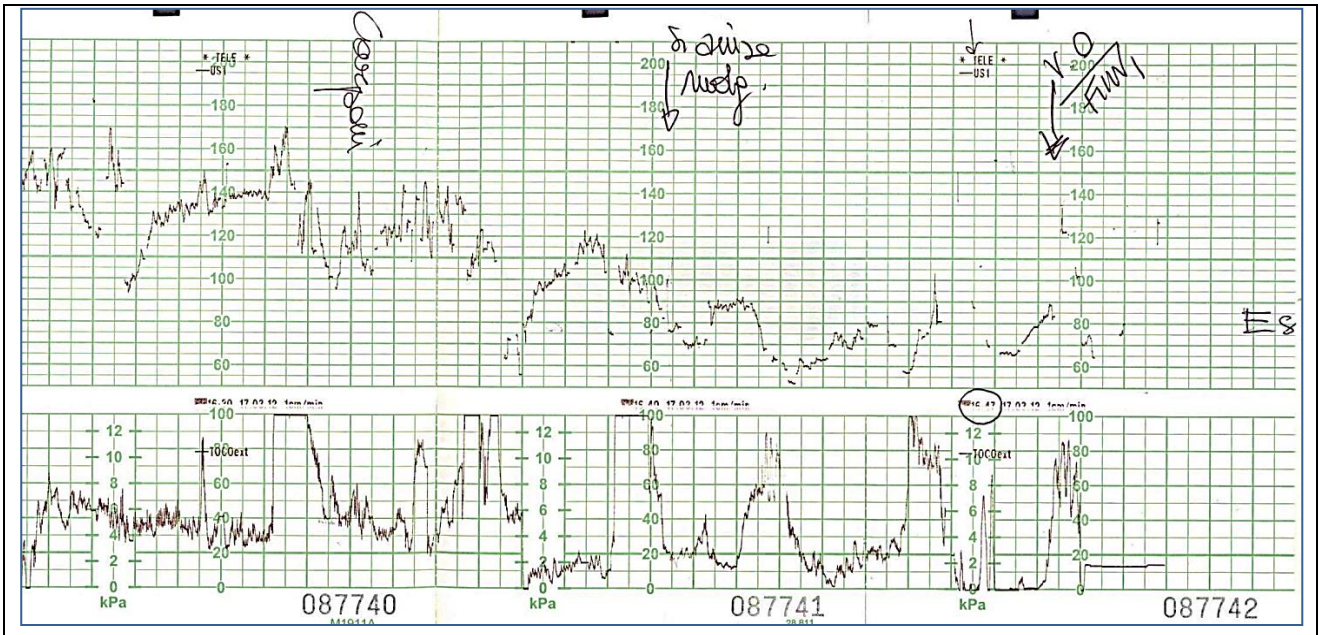
**Figure 2**.



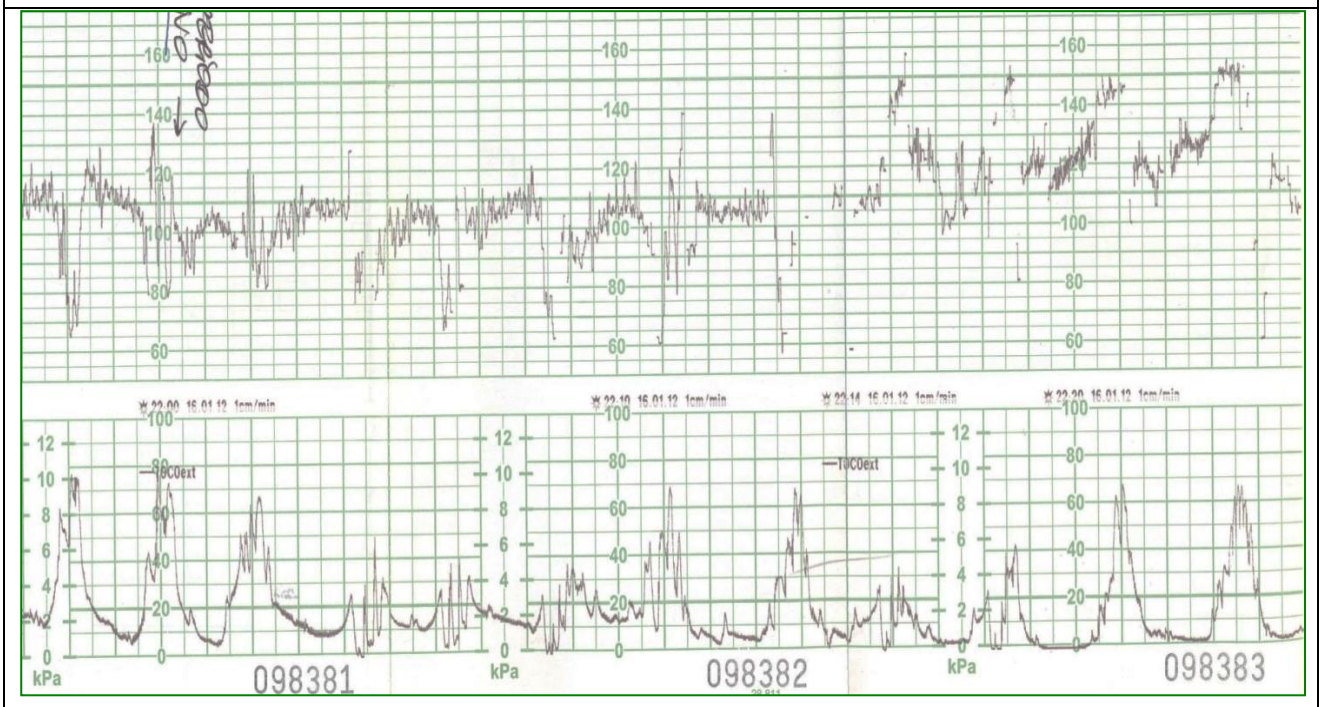

| | 0 | 1 | 2A | 2B | 3 | 4 |
|---|---|---|---|---|---|---|
| Nulli | 16,8 | 23,9 | 7,9 | 0,2 | 0,2 | 0,1 |
| Multi | 14,1 | 24,4 | 7,8 | 0,3 | 0,2 | 0,0 |

The following CTGs are examples of combined CTGs secondary to Piquard classification:

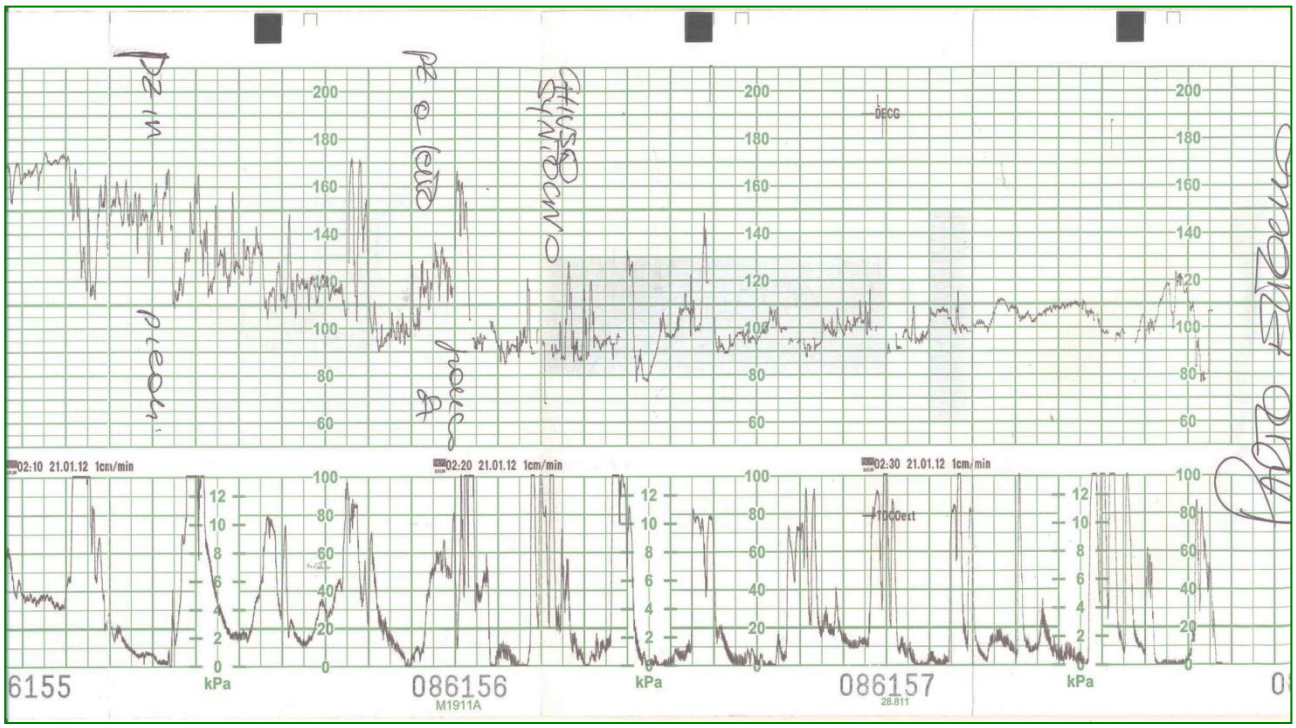
Piquard :1-2A-4



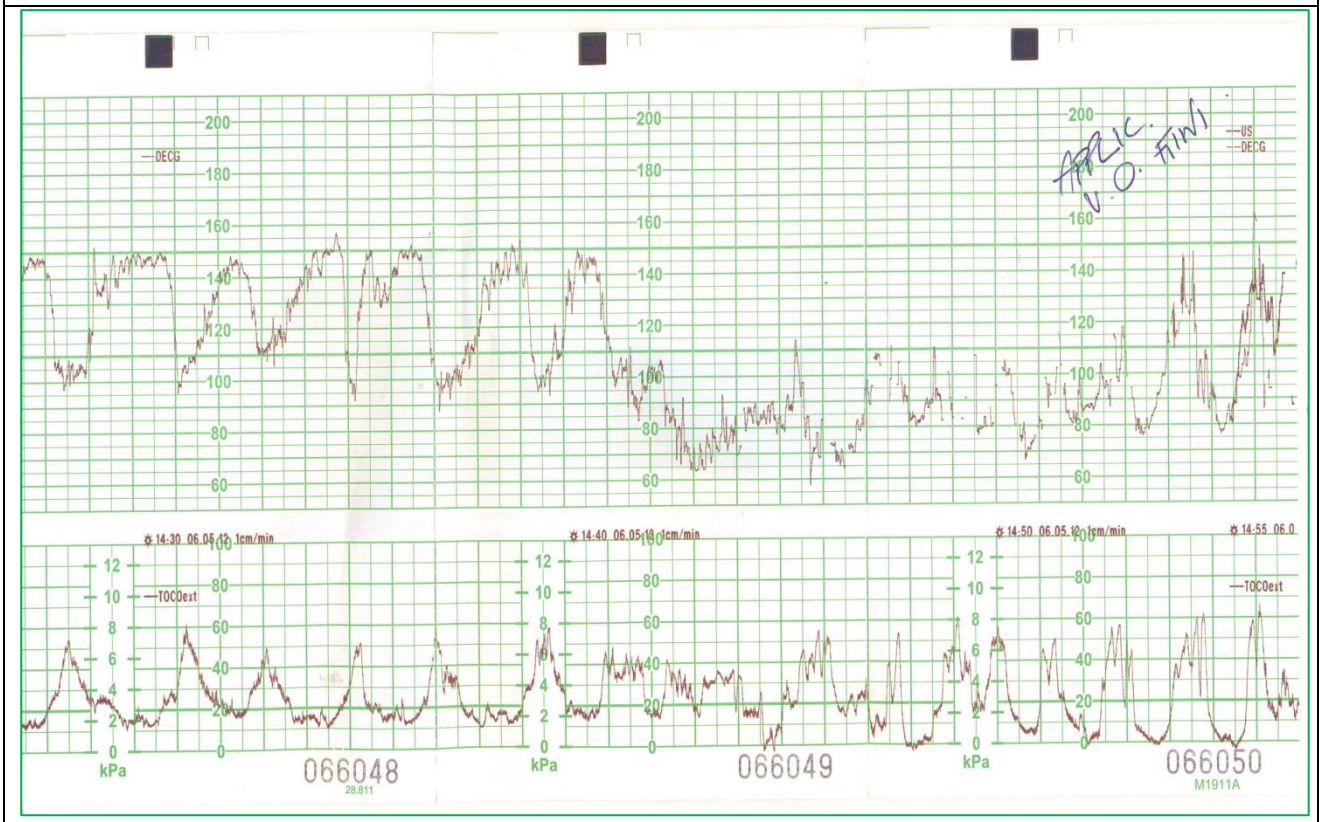

Piquard 1-0



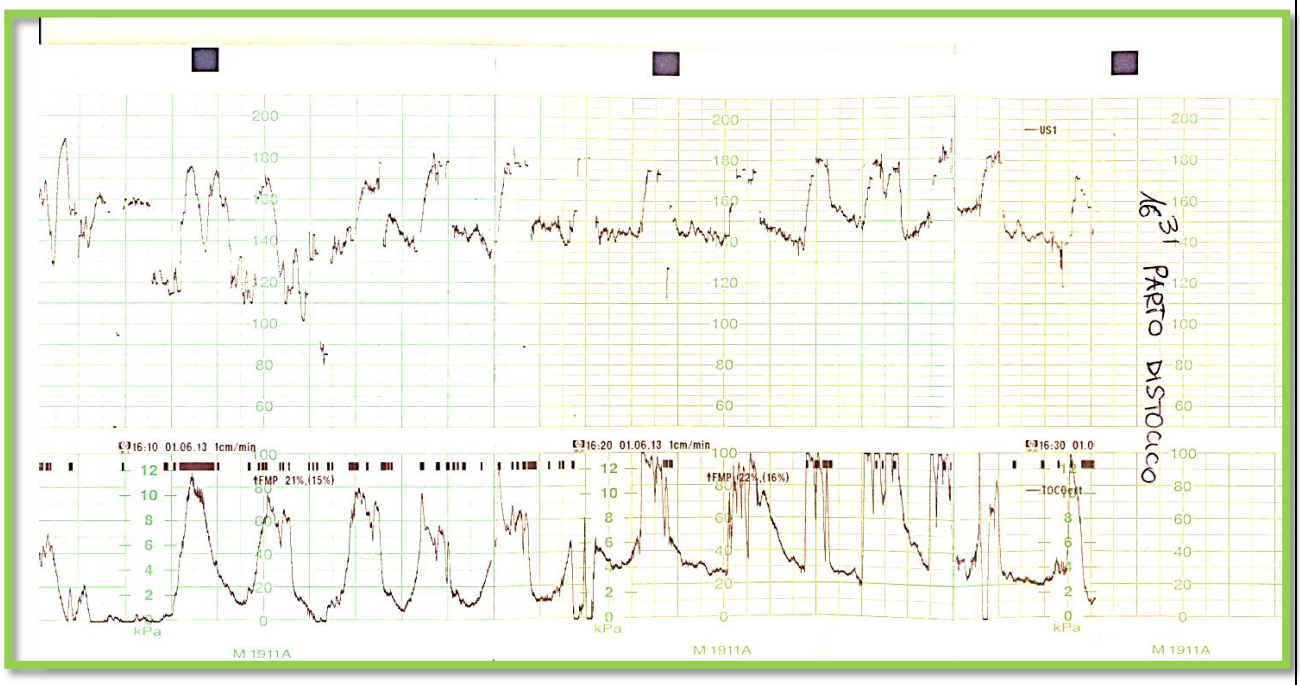
Piquard 1-2A




Piquard 1-2A



Piquard 1-0




Piquard 1-4

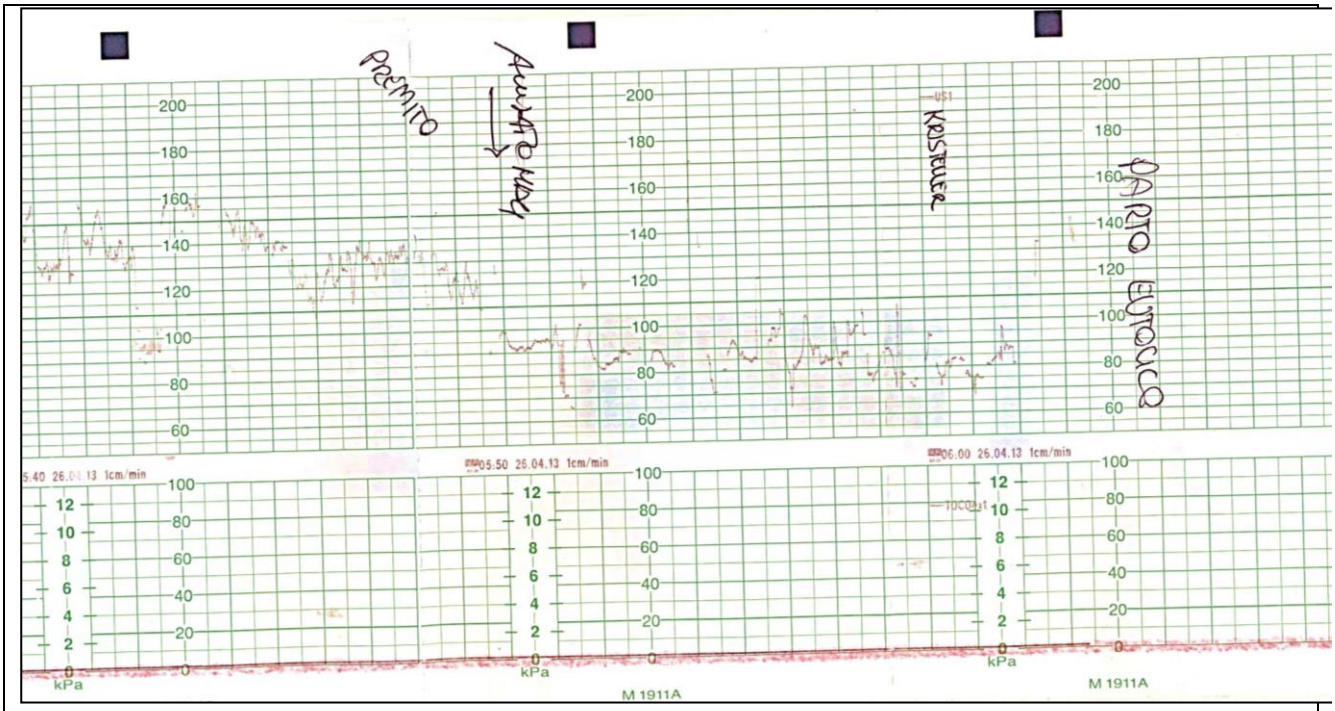
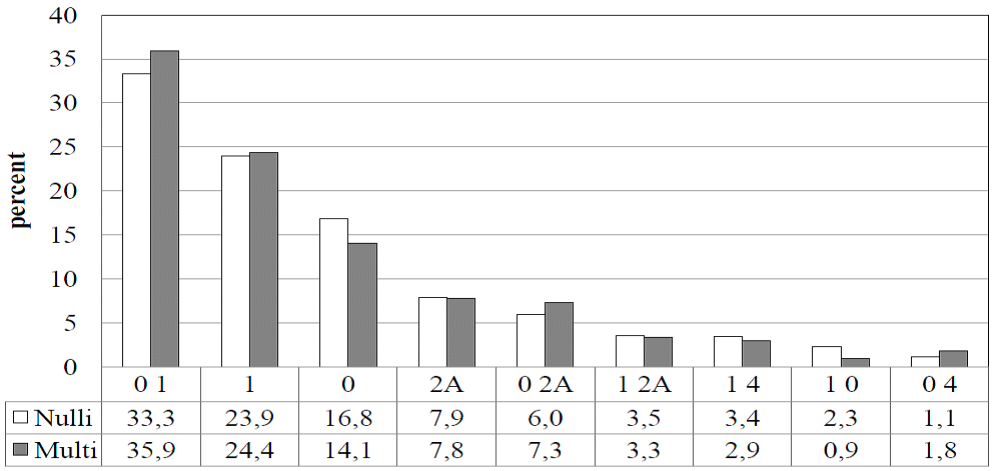


Figure 3 shows the 9 types that accounted for 98.3% and 98.4% of tracings in nulliparous and multiparous, respectively. The most frequent CTG pattern in the II stage of labor both in nulliparous and multiparous was a combination of type 0 and 1, regardless of the CTG type present in the last 60 minutes of the first stage.

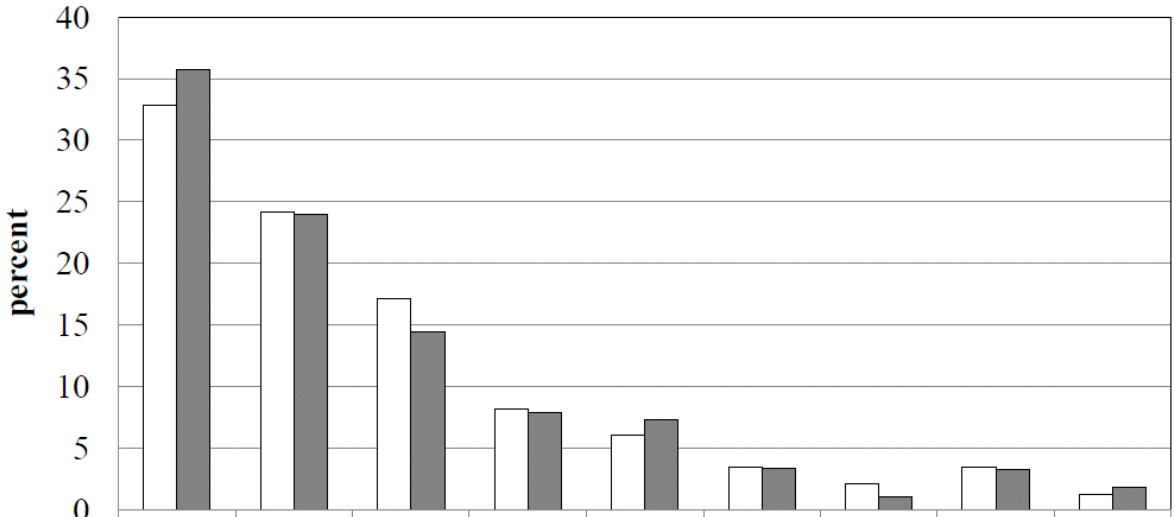



| | 0 1 | 1 | 0 | 2A | 0 2A | 1 2A | 1 4 | 1 0 | 0 4 |
|---|---|---|---|---|---|---|---|---|---|
| Nulli | 33,3 | 23,9 | 16,8 | 7,9 | 6,0 | 3,5 | 3,4 | 2,3 | 1,1 |
| Multi | 35,9 | 24,4 | 14,1 | 7,8 | 7,3 | 3,3 | 2,9 | 0,9 | 1,8 |

**Figure 3**. The 9 most frequent combinations of CTG patterns in the II stage of labor

When we analyzed the CTG patterns found in the II stage of labor in relation to that found in the I stage we found that 32.8% of nulliparous and 35.7% of multiparous with a type I CTG in the first stage exhibited this combination in the second stage (type 01). These data are shown in Figure 4.

**Figure 4**. Percentage of type I CTG in the I stage of labor compared to the different patterns found in the II stage.




| | 0 1 | 1 | 0 | 2A | 0 2A | 1 2A | 1 4 | 1 0 | 0 4 |
|---|---|---|---|---|---|---|---|---|---|
| Nulli | 32,8 | 24,2 | 17,1 | 8,2 | 6,0 | 3,4 | 2,1 | 3,4 | 1,2 |
| Multi | 35,7 | 24,0 | 14,4 | 7,9 | 7,3 | 3,3 | 1,0 | 3,2 | 1,8 |

Table 4 shows that 54/144 nulliparous (37.5%) and 27/70 (38.6%) multiparous with a type II CTG in the first stage had also this combination in the second stage.

**Table 4**.

| | CTG type I stage | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | I | IIA | IIB | IIC | I | IIA | IIB | IIC |
| CTG Type II stage | Nulliparous n=1307 | | | | Multiparous n= 989 | | | |
| 0 1 | 381 (32.8) | 12 (46.2) | 29 (33.7) | 13 (40.6) | 328 (35.7) | 5 (35.7) | 15 (35.7) | 7 (50) |
| 1 | 282 (24.2) | 5 (19.2) | 19 (22.1) | 7 (21.9) | 221 (24) | 3 (21.4) | 14 (33.3) | 3 (21.4) |
| 0 | 199 (17.1) | 4 (15.4) | 13 (15.1) | 4 (12.5) | 132 (14.4) | 2 (14.3) | 5 (11.9) | |
| 2A | 95 (8.2) | 3 (11.5) | 3 (3.5) | 2 (6.3) | 73 (7.9) | | 4 (9.5) | |
| 0 2A | 70 (6) | | 6 (7) | 2 (6.3) | 67 (7.3) | 2 (14.3) | 1 (2.4) | 2 (14.3) |
| 1 2A | 39 (3.4) | | 6 (7) | 1 (3.1) | 30 (3.3) | 1 | 1 (2.4) | 1 (7.1) |
| 1 0 | 25 (2.1) | | 3 (3.5) | 2 (6.3) | 9 (1) | | | |
| 1 4 | 39 (3.4) | | 5 (5.8) | 1 (3.1) | 29 (3.2) | | | |
| 0 4 | 14 (1.2) | | 1 (1.2) | | 17 (1.8) | | 1 (2.4) | |
| Others | 19 (1.6) | 2 (7.7) | 1 (1.2) | | 13 (1.4) | 1 (7.1) | 1 (2.4) | 1 (7.1) |
| Total | 1163 | 26 | 86 | 32 | 919 | 14 | 42 | 14 |

CTG patterns and neonatal acidemia

48/2297 (2.1%) neonates had umbilical arterial cord blood pH values ≤7.10.

**Table 5** shows the characteristics of acidemic and non acidemic pregnancies.

| | Acidemic N= 48 | Non Acidemic N= 2248 | p |
|---|---|---|---|
| Maternal age, years | 32.1 ± 6.4 | 31,1 ± 5.8 | 0.24 |
| Maternal height, cm | 162 ± 4.8 | 163 ± 6.2 | 0.35 |
| Maternal pre pregnancy weight, kg | 65.8 ± 12.2 | 61.6 ± 11.8 | 0.016 |
| Maternal pre pregnancy body mass index, $kg/m^2$ | 24.9 ± 4.3 | 23.1 ± 4.2 | 0.016 |
| $<18.5\ kg/m^2$ | 1 (2) | 173 (7.6) | 0.254 |
| $\geq 30\ kg/m^2$ | 7 (14.5) | 156 (6.9) | 0.0452 |
| Weight increase, kg | 13.2 ± 3.8 | 12.2 ± 4.7 | 0.148 |
| Nulliparous (%) | 34 (70.8) | 1273 (56.6) | 0.0554 |
| Gestational age, weeks | 39.5 ± 1.9 | 39.4 ± 1.3 | 0.8 |
| ≤34 weeks | 1 (2) | 3 (0.13) | 0.08 |
| Duration of II stage, minutes | 55.2 ± 44 | 38.1 ± 30 | 0.0001 |
| Neonatal weight, grams | 3341 ± 524 | 3305 ± 433 | 0.09 |
| < 2500 grams | 2 (4.1) | 71 (3.1) | 0.66 |
| > 4000 grams | 4 (8.3) | 140 (6.2) | 0.545 |
| Male fetus (%) | 27(56.2) | 1193(53) | 0.77 |
| Apgar score at 5’ | 9.2 ± 0.9 | 9.9 ± 0.3 | 0.001 |
| Apgar score ≤7 at 5’ (%) | 3 (6.2) | 7 (0.31) | 0.001 |
| Umbilical artery pH | 7.056 ± 0.05 | 7.268 ± 0.07 | 0.001 |
| Umbilical artery BD, mmol | -9.9 ± 2.4 | -4.4 ± 2.5 | 0.001 |
| BD ≥-12 (%) | 10 20.8) | 38 (1.7) | 0.001 |
| Complicated pregnancy (%) | 4 (8.3) | 197 (8.7) | 1 |
| Smoker (%) | 5 (10.4) | 210 (9.33) | 0.8 |
| Vacuum extraction (%) | 13 (27) | 226 (10) | 0.0008 |
| Induction of labor (%) | 19 (39.5) | 569 (25.3) | 0.03 |
| NICU | 31 (65) | 7 (0.3) | 0.001 |

This table shows that maternal BMI is significantly higher in mothers of acidemic fetuses and also the rate of obesity is significantly increased in these women, due to a significantly higher pre pregnancy weight. Induction of labor was more common in acidemic pregnancies but the rate of complicated pregnancy was similar in the two groups. Also, the length of the second stage was increased by more than 50% in acidemic pregnancies. More acidemic fetuses were delivered by vacuum. Not surprisingly, the mean Apgar score at 5' and the rate of neonates with Apgar scores ≤7 was higher in acidemic fetuses as was the rate of NICU admission. On the contrary, we could not find differences in maternal age, smoking habit, gestational age and neonatal sex and weight. The rate of nulliparity, was higher in acidemic pregnancies but it did not reach statistical significance.

When we analyzed the relationship between acidemia and type of CTG recorded in the I stage of labor we found that the presence of variable decelerations (type II B) and a reduced long term variability (type II C) were strongly associated with acidemia at birth whereas tachycardia (type II A) occurred too infrequently to found a difference (**6 Table)**

| | Acidemic N= 48 | Non Acidemic N= 2248 | p |
|---|---|---|---|
| Type I CTG in the I stage | 35 (72.9) | 2082 (92.5) | 0.0001 |
| Type II A CTG in the I stage | 0 (0) | 40 (1.7) | 1 |
| Type II B CTG in the I stage | 9 (18.7) | 128 (5.6) | 0.0017 |
| Type II C CTG in the I stage | 4 (8.3) | 46 (2) | 0.0191 |

To assess independent factors associated with acidemia at birth, we then constructed a multiple logistic regression model that included all the relevant variables that were associated with acidemia in the univariate analysis (maternal pre pregnancy weight and BMI, obesity, length of the second stage, and induction of labor).

**Table 6** shows that the presence of a type II B (repetitive decelerations) or II C (minimal FHR variability) tracing in the I stage was independently associated with the presence of acidemia at delivery; similarly, maternal BMI, induction of labor and a II stage lasting >120 minutes were also independently associated with acidemia whereas gestational age played a protective role. No significant association was found with parity.

**Table 6**

| **Characteristics** | **aOR** | **LCI** | **UCI** |
|---|---|---|---|
| Type I CTG, I stage | 1,862 | 0,022 | 1.523 |
| Type II A CTG, I stage | - | - | - |
| **Type II B CTG, I stage** | **4,405** | **1,950** | **9,95** |
| **Type II C CTG, I stage** | **5,956** | **1,942** | **18.264** |
| **BMI < 18.5** | **3.6** | **1.1** | **11.9** |
| **MBI > 25** | **8.3** | **2.06** | **33.2** |
| Male sex | 1.26 | 0.69 | 2.3 |
| Pregnancy complications | 0,181 | 0,024 | 1,381 |
| Gestational age, weeks | 0,048 | 0,006 | 0,376 |
| **Induction of labor** | **1,765** | **1,045** | **3,615** |
| Delivery by Vacuum | 1.749 | 0,817 | 3,746 |
| Parity | 0,863 | 0,424 | 1,755 |
| < 3 CENTILE | - | - | - |
| 3-10 CENTILE | - | - | - |
| > 90 CENTILE | 1,030 | 0,299 | 3,553 |
| > 97 CENTILE | 1,388 | 0,383 | 5,031 |
| Length of II stage 31-60 minutes | 1,44 | 0.688 | 3,0331 |
| Length of II stage 61-120 minutes | 1,946 | 0.816 | 4,462 |
| **Length of II stage >120 minutes** | **6,225** | **1.877** | **20.646** |

We then analyzed the relationship between the CTG types recorded in the II stage and neonatal acidemia. The results are presented in table 7, showing that only 2 acidemic fetuses (4%) presented a CTG without anomalies in the second stage (type 0) when compared to 347 (15.4%) non acidemic fetuses ($p<0.03$). On the contrary, the rate of tracings with anomalies was not different in the two groups. Only the combination of type 1 and 4 was doubled in acidemic fetuses but this didn't reach statistical significance.

**Table 7**

| | Acidemic N= 48 | Non Acidemic N= 2248 | p |
|---|---|---|---|
| 0 | 2 (4) | 347 (15.4) | 0.0254 |
| 1 | 15 (31) | 562 (25) | 0.3159 |
| 0 1 | 19 (39.5) | 780 (34.6) | 0.54 |
| 2A | 2 (4) | 181 (8) | 0.58 |
| 0 2A | 3 (6.2) | 150 (6.6) | 1 |
| 1 2A | 2 (4) | 84 (3.7) | 0.70 |
| 1 0 | 1 (2) | 42 (1.8) | 0.226 |
| 1 4 | 4 (8.3) | 78 (3.5) | 0.0895 |
| 0 4 | 0 | 32 (1.4) | 1 |
| Others | 0 | 40 (1.7) | 1 |

When we repeated the multiple logistic regression analysis to assess independent factors associated with acidemia we found that type 0 tracing was protective against acidemia whereas type 1-4 tracing was significantly associated with acidemia and type 1 was associated with acidemia with border like significance. Induction of labor and a II stage lasting >120 minutes were also significantly associated with acidemia whereas gestational age played a protective role.

**Table 8**

| | OR | LCI | UCI |
|---|---|---|---|
| **Piquard 0** | **0,048** | **0,004** | **0,577** |
| **Piquard 1** | **4,397** | **0,988** | **19,564** |
| Piquard 0-1 | 3,932 | 0,898 | 17,207 |
| Piquard 1-0 | 3,820 | 0,281 | 46,019 |
| Piquard 2A | 2,043 | 0,551 | 14,832 |
| Piquard 0-2A | 3,741 | 0,552 | 20,713 |
| Piquard 1-2A | 3,678 | 0,492 | 28,427 |
| **Piquard 1-4** | **9,054** | **1,590** | **51,546** |
| Piquard Other | - | - | - |
| Length of II stage 31-60 min | 1,697 | 0,813 | 3,545 |
| Length of II stage 61-120 min | 1,880 | 0,794 | 4,452 |
| **Length of II stage >120 min** | **4,312** | **1,291** | **14,396** |
| Pregnancy complications | 0,224 | 0,031 | 1,652 |
| **Gestational age, weeks** | **0,066** | **0,009** | **0,500** |
| **Induction of labor** | **1,886** | **1,017** | **3,496** |
| **Delivery by Vacuum** | **1,923** | **0,910** | **4,065** |
| **BMI < 18.5** | **3.77** | **1.15** | **12.3** |
| **BMI > 25** | **8.1** | **2.04** | **32.7** |
| Male sex | 1.86 | 0.64 | 2.18 |
| Parity | 0,838 | 0,410 | 1,714 |

In the acidemic group, type 1 alone o combined was present in 41/48 (85%) fetuses: 32/41 neonates (78%) had ominous characteristics criteria that we called malignant type I when decelerations were characterized by one or more of the following characteristics :

1. repetitive decelerations (≥ 3 in 10 minutes);
2. deep: ≥70 bpm;
3. duration: more than 2 minutes;
4. interval < 2 minutes between decelerations;
5. lack of return to the base line;
6. baseline characterized by tachycardia (> 160 bpm);
7. reduced variability (<6 bpm).

**Table 9**

| | acidemic | Non acidemics | p |
|---|---|---|---|
| repetitive decelerations (≥ 3 in 10 minutes) | 7 | 12 | 0.0002 |
| deep: ≥70 bpm | 6 | 9 | 0.0002 |
| duration: more than 2 minutes; | 1 | 1 | 0.043 |
| interval < 2 minutes between decelerations | 3 | 3 | 0.0002 |
| lack of return to the base line | 4 | 15 | 0.091 |
| baseline characterized by tachycardia (> 160 bpm) | 1 | 5 | 0.12 |
| reduced variability (<6 bpm) | 2 | 30 | 0.08 |
| Repeated decelerations and duration> 2 minutes | 3 | 21 | 0.012 |
| Repeated decelerations and tachycardia | 2 | 11 | 0.028 |
| Repeated decelerations and deep | 2 | 2 | 0.0025 |
| Repeated deep decelerations and tachicardia | 1 | 5 | 0.0003 |

As shown in table 9, when the first four criteria were present alone (repetitive decelerations, deep decelerations, duration more than 2 minutes and interval < 2 minutes between decelerations) or combined, the risk for acidemia was higher. Less "malignant" criteria were: lack of return to the baseline between decelerations, baseline characterized by tachycardia and reduced variability.

Interestingly, all acidemic neonates admitted in the NICU had type 1 CTG in the second stage with at least one malignant criteria.

**Table 10** presents the mean ± SD of the umbilical arterial lactate, $pCO_2$, $HCO_3$ in acidemic and non acidemic fetuses at the time of delivery, showing that lactate concentration and $pCO_2$ were significantly higher and $HCO_3$ significantly lower in acidemic when compared to non acidemic neonates.

Table 10:

| | Acidemic N=48 | Non acidemic N=2248 | p |
|---|---|---|---|
| Lactate, mmol/L | 9.4 ± 2.3 | 5 ± 2.6 | 0.001 |
| $pCO_2$, mmHg | 69.7 ± 10.8 | 49.8 ± 11.6 | 0.001 |
| $HCO_3$ | 13.7 ± 1.6 | 18.7 ± 2 | 0.001 |

**Discussion**

The main objective of our study was to reanalyze the classification of Piquard of the CTGs in the II stage of labor.

In fact, in our opinion, this study has two main limitations: the first is that it includes only 234 women (145 nulliparous and 89 multiparous) and the second is that the tracings were recorded in a very short time period, approximately 12 minutes, defined as the "final stage of labor" i.e. the period between the first voluntary bearing-down effort and the moment of delivery. In recent years, many Authors have challenged the guidelines on the length of the first and second stages of labor based on Friedman's studies [(14)] . Although human biology likely has not changed, the duration and definition of delay in the second stage of labor nowadays have an upper limit of three hours in nulliparous and two hours in multiparous [(15)] . As a matter of fact in the present study the main length of the active II stage was 48 ± 34 minutes in nulliparous and 26 ± 20.4 minutes in multiparous.

Despite this, there is no doubt that the classification of Piquard, is the only one and comprehensive for the description of the tracings in this stage of labor.

As a matter of fact, the most widely used classifications of the CTG patterns in labor do not specifically consider the second stage of labor other than for the time intervals in recording.

The American College of Obstetrician and Gynecologists in 2009 in the evaluation of critical decelerations takes into consideration the prolonged decelerations defined as the decrease of the FHR of ≥15 bpm below the base line that lasts for ≥2 minutes but less than 10 minutes of without distinguishing between I and II stage [(17).]

Similarly, the British National Institute of Health and Clinical Excellence guideline considers abnormal a single prolonged decelerations lasting 3 minutes or more but do not differentiate between I and II stage [(18).]

The Royal College of Obstetricians and Gynaecologists in its 2001 guidelines gave undifferentiated interpretations of recommendations between the 2 stages [(19).]

The Society of Obstetricians and Gynaecologists of Canada in its 2007 guidelines considered abnormal the presence of repetitive (≥3 times) decelerations to ≤ 70 bpm for ≥ 60 seconds, baseline

tachycardia or bradycardia and single prolonged deceleration lasting more than 3 minutes but less than 10 minutes, but again they did not differentiate between I and II stage( 20).

The International Federation of Gyneacology and Obstetrics only underlines that the late first stage and II stage are the moments of increased fetal stress and recommends an adequate and careful choice of mode and time of surveillance (21).

Thus, the interpretation of CTGs in the second stage of labor remains neglected whether culturally and clinically even though it represents a critical time for the fetus that is often subject to reduced oxygenation. In this stage the uterine contractions become stronger and the expulsive forces are greater and more frequent when the woman begins to bear-down; the fetal head starts its descent through the birth canal which increases the intracranial pressure and reduces cerebral blood flow. Baroreceptors are then activated which produces decelerations of the fetal heart rate usually synchronous with the uterine contraction.

When we started analyzing the CTG tracings we indeed found not only that less than 50% of the tracings could be classified according to the original six types proposed by Piquard, but also that 3 types (2B, 3 and 4) were very rare, accounting for only 0.5% of the tracings. Instead, many tracings were actually a combination of different types, type 01 being the most representative. A possible explanation might be that the observation time in the present study (i.e. the duration of the active II stage) is twice as long in multiparous and approximately fourfold longer in nulliparous than the observation time in Piquard's study.

The main workshops in the literature and the various trials aimed to ameliorate interpretation of FHR during labor did not differentiate between the I and the II stage although what is quietly known that fetal stress is different between these 2 phases because more exposed to oxygen reduction. The main difference is regarding uterine contractions which have more duration, frequency and intensity reaching up to 50-100 mmHg causing increased intracranial pressure which reduced cerebral blood flow. The second factor is the start of bearing down efforts which cause additional more intrauterine pressure. These factors reduce whether umbilical than placental blood perfusion because the augmentation of both factors can lead to intrauterine increase rise up to 150-250 mm Hg with resulting impaired fetal oxygenation and increase of carbon dioxide level.

So the interpretation of CTGs in the I stage cannot be applied to the II one because they might have different meanings.

We consider this work as a pilot study done with the aim to better evaluate CTGs in the II stage of labor and correlate them with acidemia and to suggest to clinicians to have a different interpretation between the first and second stage of labor.

We think that our results represent a preliminary interesting data that have to be confirmed including a major number of acidemic fetuses.

BIBLIOGRAPHY:

1-Goodlin RC. History of fetal monitoring. *Am J Obstet Gynecol* 1979; 133: 323–52

2- Brady JP, James LS, Backer MA. Heart rate changes in the fetus and newborn infant during labor. *Am J Obstet Gynecol* 1962; 84: 1–11.

3- Boehm FH. Prolonged end stage fetal heart rate decelerations. *Obstet Gynecol* 1975; 45: 579–82.

4-( Krebs HB, Petres RE, Dunn LJ. Intrapartum fetal heart rate monitoring. V. Fetal heart rate patterns in the second stage of labor. *Am J Obstet Gynecol* 1981; 140: 435–9.

5- CG, Graca CM, Clode N. A study on second stage cardiotocographic patterns an
d umbilical acid-base balance in cases with first stage normal fetal heart rates. *J Maternal Fetal Invest* 1995; 5: 144-7.

6-Gilstrap LC, Hauth JC, Toussaint S. Second stage fetal heart rate abnormalities and neonatal acidosis. *Obstet Gynecol* 1984; 63: 209–13.

7-Sheiner E, Hadar A, Hallak M, Katz M, Mazor M, Shoham-Yardi I. Clinical significance of fetal heart rate tracings during the second stage of labor. *Obstet Gynecol* 2001; 97: 747–52.

8-Gilstrap LC, Hauth JC, Hankins GD, Beck AW. Second stage fetal heart abnormalities and type of neonatal acidaemia. *Obstet Gynaecol* 1987; 70: 191-5.

9-Melchior J, Bernard N. Incidence of and pattern of fetal heart rate alterations during labour. In: Kunzel W, ed. Fetal heart rate monitoring. Heidelberg: Springer-Verlag, 1985:73-81

10-Piquard F, Hsiung R, Mettauer M, Schaefer A, Haberey P, Dellenbach P. The validity of fetal heart rate monitoring during the second stage of labor. *Obstet Gynecol* 1988; 72: 741–50. ).

11- E Bertino, S.Milani, C Fabris AND m De Curtis : Neonatal anthropometric charts : what ther are, what they are not ( Arch Dis Child . Fetal Neonatal Ed. 2007; 92; 7-10

12-the 2008 national institute of child health and human development workshop report on electronic fetal monitoring ; update on definitions, interpretation and research guidelines. George A. Macones, MD, Gary D.V.Hankins MD

13-Cahill AG, Roehl KA, Odibo AO, et al. Association and prediction of neonatal acidemia. Am J Obstet Gynecol 2012;207:206.e1-8.

14-Harper. M, Caughey B, Roehl A, Odibo O, Cahil G. Defining an abnormal first stage of labour based on maternal and neonatal outcomes. Annual meeting for maternal fetal medicine. San Francisco. Feb. 11-16, 2013

15- ACOG 2008. Dystocia and augmentation of labor .Clinical management guidelines for obstetricians-gynecologists. N.49.

16-NICE. Clinical guidelines. Intrapartum care: care of healthy women and their babies during childbirth. 2007, Sep

17- ACOG Practice Bulletin. Intrapartum fetal heart reate monitoring: nomenclature, interpretation, and general management principles.Number 106, July 2009

18-National institute for Health and Clinical Excellence.intrapartum care. September 2007

19-Royal College of Obstetricians and Gynaecologists. The use of electronic fetal monitoring . evidence-based clinical guidelines, 2001, Number 8

20- Liston R, Sawchuck D, Young D; Society of obstetrics and gynaecologists of Canada ;British Columbia Perinatale Health Program . Fetal health surveillance : antepartum and intrapartum consensus guidline. J Obstet Gynaecol Can 2007;29:S3-56

21- International Federation of Gynaecology and Obstetrics. Guidelines for the use of fetal monitoring . Int J Gynaecol Obstet 1987;25:159-167

22- Yvonne M. O'Brien, Deirde J. Murphy.European Journal of Obstetrics and Gynaecology and reproductive Biology 167 (2013) 142-145