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Pathophysiological framing of cardiotocographic tracing and Uterine Tachysystole

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ABSTRACT

Continuous electronic fetal monitoring (EFM) was first introduced commercially over 50 years ago with the hope of improving perinatal outcomes during labor. However, despite the increased use of EFM, definitive improvements in perinatal outcomes have not been demonstrated. Variance in tracing interpretation and intervention has led to increased rates of cesarean and operative vaginal deliveries and perhaps increased maternal and neonatal morbidity. Since its inception, several strategies have been developed in hopes of optimizing EFM and improving these outcomes.

Tachysystole is defined as more than five contractions in 10 minutes, averaged over 30 minutes. The presence or absence of associated FHR abnormalities is the key issue in management. For women with spontaneous labor, tachysystole coupled with recurrent FHR decelerations requires evaluation and treatment. Tachysystole occurring with less frequent FHR abnormalities may or may not require treatment, depending on the specific clinical situation and associated FHR characteristics such as variability and accelerations. In laboring women receiving oxytocin, management of tachysystole generally involves efforts to reduce uterine activity to minimize risk of evolving fetal hypoxemia or acidemia. In labor induction or augmentation or both, a decrease in the oxytocin dose should be considered if tachysystole occurs in the presence of a Category I tracing. If there is a Category II or III tracing, oxytocin should be reduced or stopped in addition to intrauterine resuscitation. In addition, simultaneous initiation of multiple resuscitative measures may improve fetal condition more rapidly than the use of individual therapies (Table 2). If tachysystole induced FHR abnormalities do not resolve with these initial maneuvers, then tocolytic medications (eg, terbutaline) may be warranted.

Keywords: Cardiotocography; Electronic fetal monitoring, Uterine Contraction, Uterine Tachysystole.

Physiology of uterine contractions

Contractile activity is an intrinsic property of the uterus and is carried out through a complex system of muscle fibers and contraction wave propagation under pacemaker guidance. The myometrium consists of three layers of muscle fibers that together are arranged forming two spirals that each start from the region of the tubal ostia, in direct continuation with the musculature of the salpinges, traverse the various planes without interruption, and lead contralaterally and downward to the lower uterine segment, and then exhaust at the level of the cervix [1].

The physiology of uterine contractions was extensively studied in the 1960s by Caldeyro-Barcia and Poisero, creating the "Montevideo unit" for determining uterine performance [2]. A Montevideo unit corresponds to the product of contraction intensity (i.e., the increase in intrauterine pressure above basal tone during contraction, expressed in mmHg) times the frequency of contractions in 10 minutes. For example, 2 contractions in 10 minutes of an average intensity of 60 mmHg corresponds to 120 Montevideo units.

Through the use of multiple intrauterine catheters, these investigators have shown that the impulse for contraction can originate from two distinct uterine areas that act as pacemakers and are each located at the level of each uterine horn. Usually, the right pacemaker prevails over the left and triggers the nerve impulse that within 15 seconds depolarizes the entire myometrium from top to bottom. The intensity of the contraction is greatest at the fundus and decreases progressively going toward the lower uterine segment, and then discharges the peristaltic wave at the level of the cervix allowing it to mature (shortening and dilation).

Before 30 weeks of gestation, the uterus remains physiologically released; from the following weeks it begins to develop sporadic contractions of low intensity referred to as Braxton Hicks contractions, which become progressively larger in amplitude and frequency.

Also, the Montevideo group showed that uterine contractions begin to be perceived as painful by women when they exceed an intensity of 15 mmHg, which is also the minimum threshold needed to cause changes in the cervix. According to their studies, labor in childbirth begins when about 120 Montevideo units are reached, or roughly 3 contractions in 10 minutes of 40 mmHg intensity. During the first stage of labor, the frequency reaches up to 5 contractions in 10 minutes, while the intensity gradually reaches 50-60 mmHg. In the expulsive period, partly due to the enhancement induced by maternal thrusts, the intrauterine pressure developed by contractions reaches 80-100 mmHg. Basal uterine tone is maintained around 8-12 mmHg throughout labor.

Although contractions change in amplitude and frequency as labor progresses, the duration of a single contraction remains almost constant, around 60-100 seconds [3]. During contraction, the intrauterine pressure easily exceeds the pressure within the spiral arterioles, which corresponds to about 30 mmHg. Therefore, the blood flow within them stops and fails to supply the placenta, and thus the fetus, with new oxygenated blood [4,5]. This event is certainly a stressful factor for the fetus, but if the fetus is in a healthy state and has adequate placental reserve, it is able to tolerate the temporary reduction in oxygenation and recover quickly after the contraction ends. This fetal resistance to hypox-

ic stress during contraction is enabled mainly by the reserve of retroplacental oxygenated blood that is "squeezed" into the intervillous space by the contraction itself and that supports the fetus when the spiral arterioles are occluded.

In the uterine relaxation time between contractions, fresh oxygenated blood flows back into the retroplacental space due to the supply from the uterine arteries, allowing the fetus to recover the reserves lost in the previous contraction in order to respond adequately again to the next contraction. It has been shown that fetal blood saturation decreases during contraction reaching a minimum value about 92 seconds after the peak, and the fetus is able to recover basal blood oxygenation after another 90 seconds [6]. Therefore, adequate uterine relaxation time between contractions is crucial for proper restoration of fetal oxygenation.

Obviously, any alteration on either the fetal or maternal side can compromise this delicate balance resulting in a fetal inability to respond to contraction-induced stress, with development of hypoxia and cardiotocographic changes. For example, a fetus with intrauterine growth restriction and placental insufficiency does not have a good retroplacental blood supply and therefore can easily develop hypoxia during the contractions of a physiological labor.

Conversely, a fully well fetus may fail to respond adequately to the hypoxic stress induced by contractions if they are of greatly increased amplitude and especially duration or very high frequency, not allowing physiological recovery between one contraction and the next [7-9].

Measurement of uterine pressure using an intrauterine catheter has been widely used for research purposes to be able to quantify as accurately as possible the intensity, duration and frequency of contractions, but in clinical practice it finds little application because of the invasiveness and discomfort it can cause in the patient. Cases, albeit rare, of fetal injury, placental hemorrhage, uterine rupture, and infection following the use of intrauterine pressure-sensing catheters have also been described [10].

Normally in clinical practice, an external tocodynamometer, which is placed in the upper/bottom portion of the uterus, is used to monitor uterine contractile activity in labor. This instrument detects abdominal wall tension generated by the underlying uterine contraction, so it represents an indirect measurement of intrauterine pressure and therefore is not a reliable method for quantifying the actual force or duration of contraction, let alone basal uterine tone.

But in daily clinical practice it represents a valid method as it provides indications about the frequency of contractions and their timing with respect to any changes in fetal heart rate [11,12]. In obese women, external tocography becomes even less reliable as it often fails to adequately record uterine contractions due to the interposition of abdominal fat. Therefore, in cases of labor in obese women in whom slowing of progression is suspected or otherwise poor contractile activity is documented, it may be indicated to use intrauterine pressure detection [12].

A 2013 Cochrane systematic review compared intrauterine with external measurement of uterine contractile activity in oxytocin-induced or oxytocin-enhanced labor and found no significant differ-

ences between the two methods regarding maternal and fetal outcomes [13]. Given these non-directive results and in any case based on a few studies with a small number of participants and of modest quality, it is suggested that the less invasive and easier device be used in clinical practice, reserving the intrauterine one for difficult and doubtful cases.

Tachysystole and its clinical implications

There has been much confusion in the literature in recent decades about the use of the appropriate terminology to refer to excessive uterine contractility, indifferently using terms such as tachysystole, hypercontractility, hyperstimulation, and uterine hypertone. For this reason, in 2008, a workshop between the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), the American College of Obstetricians and Gynecologists (ACOG), and the Society for Maternal-Fetal Medicine [14], organized to define the state of the art on cardiotocographic monitoring, indicated tachysystole as the most appropriate term to use, defined as a frequency of uterine contractions > 5 in 10 minutes, over a period of at least 30 minutes. Although this definition only describes the frequency of contractions, the same panel experts point out how important it is to also consider the intensity and duration of contractions, as well as the time of uterine relaxation between contractions.

Although the general indication is to abandon the use of terms other than tachysystole, it may be useful for clinical practice purposes to make some particular distinctions. Hyperstimulation properly refers to tachysystole caused by iatrogenic oxytocin administration to induce or enhance labor in labor; whereas the term uterine hypertone refers to increased basal tone of the uterus between contractions with cardiotographic changes [12].

As already mentioned, excessive uterine contractility can lead to fetal decompensation due to reduced placental oxygenation during contractions and insufficient recovery time between contractions, thus leading to progressive hypoxia, alterations in fetal heart rate (FCF), acidosis, and, if the hypoxic noxa persists, severe neurological damage such as hypoxic-ischemic encephalopathy [6-9,15].

Excessive uterine contractile activity is the dominant cause of fetal hypoxia and acidosis in labor, so it must be promptly detected based on cardiotocographic features (presence of tachysystole associated with suspicious or frankly pathological FCF pattern) and treated as soon as possible to prevent irreversible fetal damage [16].

Fetal heart rate may undergo different alterations during tachysystole, depending on the underlying mechanism causing the hypoxia and the general fetal condition prior to the tachysystole itself. A study conducted by Heuser et al. in 2013 [17] that retrospectively analyzed the presence of tachysystolic events in more than 50,000 deliveries, spontaneous and induced, showed that tachysystole was found in 11 percent of these overall.

Approximately in two-thirds of the tachysystolic events there were no changes in FCF and therefore no need for corrective maneuvers, while in the remaining third of cases most of the FCF changes found fell within category II of the NICHD/ACOG classification [14]. In particular, variable decelerations (87%), decreased variability (11.5%), late decelerations (7%), tachycardia (6%), bradycardia or prolonged decelerations (4%) predominated. Depending on the rate of onset of tachysystole-

induced hypoxia, pre-existing fetal well-being at this hypoxic event, and the cardiocirculatory responses that the fetus is able to enact, there are cardiotocographic patterns typical of both slow-evolving hypoxia (variable or early decelerations followed by tachycardia and loss of variability) and subacute hypoxia (deep and prolonged variable decelerations with reduced recovery period to basal heart rate), both of which can evolve toward preterminal bradycardia if not adequately and promptly corrected [7].

Uterine tachysystole can be spontaneous or more frequently provoked by agents that increase uterine contractile activity to induce or enhance labor in childbirth, such as oxytocin and prostaglandins. Among the latter, the agent most likely to induce tachysystole is misoprostol, especially when administered vaginally [18].

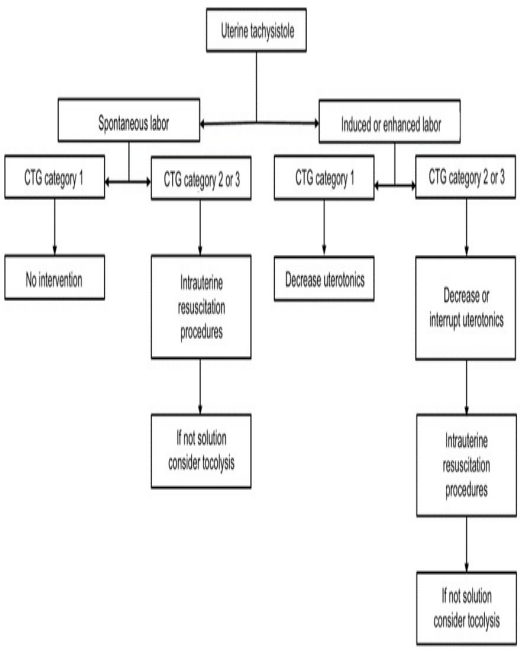
Also in the study by Heuser and coworkers [17], the major risk factors for the onset of tachysystole were found to be precisely oxytocin and misoprostol. In particular, the use of oxytocin increased the risk of FCF alterations by twofold and the need for obstetric intervention, such as discontinuation of oxytocin administration or use of tocolysis, by threefold. In addition, a correlation was shown between oxytocin dose and tachysystole risk: for every 5mUI/min increase in oxytocin infusion, there was a slight but statistically significant increase in tachysystole risk (RR 1.12, 95% CI 1.06-1.13). Finally, the presence of tachysystole increased the use of vaginal operative delivery and the incidence of multiple adverse neonatal outcomes by 30%.

Given the important increase in the risk of tachysystole following oxytocin or prostaglandin administration, all major global guidelines, including those of the International Federation of Gynaecology and Obstetrics (FIGO), those of the National Institute for Health and Clinical Excellence (NICE), and the ACOG, recommend continuous cardiotocographic monitoring in induced or accelerated labor [16,19,20].

In the case of tachysystole with alterations in FCF, and thus probable fetal hypoxia ingravescence, it is recommended to reduce or discontinue oxytocin infusion or remove if possible, the prostaglandins administered, and if this is not sufficient, tocolysis with beta-adrenergic agonists (ritodrine, terbutaline, salbutamol), atosiban or nitroglycerin is recommended, along with intrauterine resuscitation maneuvers [16].

The ACOG has devised a useful algorithm to guide operators in cases of both spontaneous and iatrogenic tachysystole based on the characteristics of FCF (Table 1) [21].

Table No.1 - Algorithm for the management of uterine tachysystole



(Table adapted from ACOG Practice Bulletin No. 116, Management of Intrapartum Fetal Heart Rate Tracings).

Oxytocin and tachysystole

Oxytocin was the first polypeptide hormone to be synthesized in 1955 and is widely used worldwide as the main agent that can induce or enhance labor, as well as in the prophylaxis and therapy of postpartum hemorrhage.

To induce or potentiate labor, it is administered by slow infusion, diluting 10 or 20 units of oxytocin in 1000 ml (or 5-10 IU in 500 ml) of crystalloids such as saline or ringer lactate to reach a concentration of 10 or 20 mU/ml of solution, respectively. Once the infusion is started, the effects on uterine contractile activity can occur as early as 3 to 5 minutes, while at least 40 minutes are required to reach a stable concentration within the plasma [22,23].

Over the decades since its introduction, different therapeutic regimens of oxytocin administration have been used and compared with each other. Two broad categories can be distinguished: low-dose and high-dose protocols [22]. Historically, the earliest to have been used are the low-dose regimens, i.e., those that involve starting with doses of 0.5-2 mU/min to be progressively increased every 15-40 minutes to allow labor to progress. From the 1970s-80s, high-dose regimens (4-6mU/min) began to be adopted on the back of promising results of the "active management" of labor introduced by the Dublin group, which basically involved bringing all labor under 12 hours through the early and routine use of amniorexi and high doses of oxytocin, with the aim of reducing the rate of cesarean sections for dystocia without worsening maternal and neonatal outcomes [24-25].

Several subsequent studies have attempted to compare benefits and risks of these two treatment regimens in relation to fetal and maternal outcomes.

A systematic review conducted by Cochrane in 2013 compared the use of high (≥4mU/min) and low (<4 mU/min) dosages of oxytocin to increase labor progression in women with dystocia. Although preliminary analysis showed that high-dose regimens were more successful in reducing the duration of labor, the rate of cesarean sections, and increasing the likelihood of vaginal delivery than low-dose regimens, no significant difference was confirmed, correcting for the various biases present. In addition, there was no difference between the two groups for the incidence of tachysystole, risk of operative delivery or pathological pH on the umbilical artery, while cardiotocographic changes were not studied [26].

A recent randomized controlled trial of about 1,200 women whose labor was augmented with high or low dosages of oxytocin showed no difference in the cesarean section rate between the two groups, but a significant increased risk of tachysystole in the high-dose regimen group (43.2% versus 33.5%). Furthermore, although the risk of operative vaginal delivery was similar between the two groups, in the high-dose protocol the reason for instrumental delivery was related more to fetal distress (43.8% vs. 22.7%) rather than failure to progress, with a statistically significant difference [27].

In contrast, another Cochrane systematic review compared the two treatment regimens in relation specifically to induction of labor [28]. Again, this

showed a decrease in labor duration in the high-dose regimens, but at the expense of an increased risk of tachysystole (but without specifying whether or not FCF alterations were associated).

In conclusion, both administration regimens are currently used, and in any case of induction or augmentation of labor with oxytocin, careful cardiotocographic monitoring is necessary to rule out the development of tachysystole and possible fetal distress, especially if high-dose regimens are used.

During oxytocin infusion, consideration should be given not only to its initial dose but also to the time intervals of dose increase during labor. Generally, with continuous infusion of oxytocin, the dose is gradually increased at intervals ranging from 15 to 40 minutes depending on the protocols. Again, the risk of tachysystole increases the faster the oxytocin dose is increased [22,29].

At the uterine level, oxytocin binds to its specific receptors, the expression of which on the myometrium increases at term, making the uterus more sensitive to this endogenous or exogenous hormone. In labor treated with oxytocin, if the continuous infusion is prolonged for a long time, the oxytocin receptors may become desensitized to the hormone, which then can no longer achieve the same stimulating effect on the myocells except at increasing dosages. This effect can last for several hours or even days, having very important clinical implications. Indeed, not only is the progression of labor slowed because the uterus responds less to oxytocin, but also the risk of uterine atony in the immediate postpartum with subsequent hemorrhage unresponsive to additional doses of oxytocin is increased [30]. In physiologic labor, endogenous oxytocin is produced pulsatile rather than continuously as is the case with exogenous infusion, and this mechanism likely reduces receptor desensitization. Confirming this, it has been shown that if exogenous oxytocin is also administered pulsatile and not continuously much lower dosages are required to achieve the same effects on uterine contractile activity [31].

In addition to tachysystole, other undesirable effects associated with oxytocin include hypotension and hyponatremia. Hypotension develops due to oxytocin-induced peripheral vasodilatation especially following its rapid bolus administration, such as occurs in the postpartum period for the prevention or treatment of hemorrhage (in fact, an oxytocin bolus infusion time of at least 1 minute is recommended) [32].

Hyponatremia, on the other hand, can be determined following high oxytocin dosages (usually above 20 mU/min) and maintained for a long time. In fact, oxytocin in high plasma concentrations can mimic the action of vasopressin (also produced by the neurohypophysis) and thus result in increased renal free water recall causing hyponatremia, and the subsequent development of cerebral edema, seizures, coma, and death [22].

Oxytocin and intrapartum clinical conduct

As has been shown, oxytocin used in labor does not directly result in changes in FCF, but rather indirectly by going to increase the frequency, intensity, and duration of uterine contractions. In most cases, these modifications are necessary and sufficient to allow proper progression of dystocic labor to delivery, without harmful consequences toward the fetus. Occasionally, however, changes in contractile activity may be such that fetal oxygenation is compromised, leading to worsening hy-

poxemia and alterations in FCF. Oxytocin infusion can result in these fetal alterations not only with tachysystole properly defined (>5 contractions/10 minutes), but also with the appearance of uterine hypertone (increased basal uterine pressure), or with the important increase in the amplitude or duration of individual contractions despite their normal frequency [12,33].

Before starting oxytocin administration to induce or enhance labor, it is always recommended to perform cardiotocographic monitoring of at least 20 to 30 minutes to assess the present uterine contractile activity and fetal heart rate pattern, which should be classified as normal (according to FIGO and NICE classification) or belonging to category I according to NICHD/ACOG classification (baseline rate in the normal range, adequate variability, presence or absence of acceleration, and absence of late or variable deceleration) [14,16,19]. Oxytocin infusion should never be initiated if the cardiotocographic tracing has a suspicious/unreassuring or pathological/abnormal (or category II or III, respectively) pattern. Although this may seem like an obvious statement, sometimes unfortunately in clinical practice this simple recommendation is not followed, often with important neonatal consequences and medico-legal implications.

As discussed above, when initiating oxytocin infusion to induce or enhance labor in labor, continuous cardiotocographic monitoring is recommended.

In case of documented slowing of labor progression, amniorex should be performed before proposing oxytocin administration to the patient, i in case the amniochorionic membranes are still intact, and wait (even up to two hours in stage I labor) to ascertain whether this maneuver alone was helpful in allowing cervical dilation to progress [22,34].

An important factor to consider during stage I labor when considering using oxytocin is cervical maturation and its ability to dilate following uterine contractions. If the cervix appears soft, contractions of low intensity will be sufficient to allow it to dilate; consequently, it is obvious that there is no point in potentiating contractions that appear to be of low amplitude on tocography if cervical dilation is continuing physiologically. Therefore, oxytocin should be initiated only if slowed cervical dilation is documented with low-amplitude uterine contractions.

Conversely, if the cervix is very rigid and not very distensible, the myometrium will produce more intense contractions to try to overcome its resistance. Administering oxytocin in this situation with the intent of promoting cervical dilation would only further increase the intensity of the contractions, risking a greater reduction in blood supply to the fetus and thus leading to fetal hypoxia [12].

In addition to carefully assessing the cardiotocographic pattern and the stage and progression of ongoing labor, other intrapartum and antepartum parameters (IUGR or post-term fetus, oligohydramnios, pre-eclampsia, meconium-stained fluid, hemorrhage hyperpyrexia) to understand whether indeed oxytocin may be a valid and safe method of enhancing labor and what level of surveillance should be implemented based on the presence of other risk factors that may compromise fetal well-being more quickly in conjunction with oxytocin administration.

As we have already discussed, tachysystole can result in several alterations in fetal heart rate, dependent mainly on the degree of feto-placental hy-

poperfusion and basal fetal well-being. Simpson and James in 2008 [35] showed that in women undergoing labor induction with oxytocin, those who had one or more episodes of tachysystole for at least 30 minutes manifested greater incidence of cardiotocographic alterations such as reduced or absent variability, reduced accelerations, and the appearance of late and recurrent decelerations, compared with the group without tachysystole; also minimal differences in the frequency of contractions (≥ 6/10 minutes compared with contractions ≥ 5 but < 6/10 minutes) significantly increased the incidence of recurrent decelerations. But the major finding was the finding that, compared with controls, significant reductions in fetal oxygen saturation (FpO2) were already occurring after 5 minutes of tachysystole, which progressed over the course of the tachysystole, while cardiotocographic changes occurred on average after 20 minutes, when FpO2 dropped around 40%.

In clinical practice, therefore, it is recommended to implement corrective maneuvers as soon as tachysystole is documented, even without waiting for it to cause abnormal FCF patterns to appear, as these take over when often significant fetal hypoxemia has already occurred [33].

The main corrective maneuver is to reduce, usually by half, the ongoing dose of oxytocin or discontinue it, until uterine contractions have reduced and FCF changes have normalized. Along with this, it is always useful to combine other corrective interventions, such as positioning the parturient on her left side and administering crystalloid infusions such as ringer lactate, as the combination of these maneuvers has been shown to be more helpful than oxytocin reduction or discontinuation alone [35]. In case these remedies are not sufficient to bring back cardiotocographic changes, especially frankly pathological or abnormal FCF changes, it is indicated to proceed with tocolysis by administration of beta2-agonists (such as ritodrine or terbutaline), or atosiban [16,34].

In most cases it is not necessary to arrive at the tocolysis to solve the uterine hyperstimulation by oxytocin as its reduction or suspension together with the other corrective operations previously exposed are sufficient. However, it should also be borne in mind that the cardiotocographic changes induced by oxytocin may take some time to regress after its suspension. In fact, although the plasma half-life of oxytocin is only 3-4 min, this remains linked for a long time to its receptors in the myometrium, therefore it may take even more than 40 minutes to obtain a reduction of its action on the uterus, once the administration is suspended [22,33]. In addition, sensitivity to oxytocin by the myometrium increases progressively during labor, especially during the passage between stage I and II, thanks to the Ferguson reflex. This consists in the reflex stimulation of the contractile activity induced by the pressure that the presented part exerts on the cervix and/or on the upper part of the vagina. This reflex should therefore also be considered, both during the administration of oxytocin and when it is suspended and expected to reduce its effect on the uterus [7].

Another critical point in clinical conduct is when you can resume oxytocin infusion after stopping it. It is generally considered that administration may resume when tachysistoly and the abnormal pattern of FCF have disappeared, with a dosage corresponding to half the dose at the time of withdrawal if less than 30 minutes have elapsed from it, or with a dosage of 1 mu/min if more than 30 minutes

have elapsed [21]. But some authors recommend that you wait twice as long as needed for normalization of the cardiotocographic pathway, as, even if the FCF is back to normal, changes in pH, pCO2 and fetal po2 may still persist.

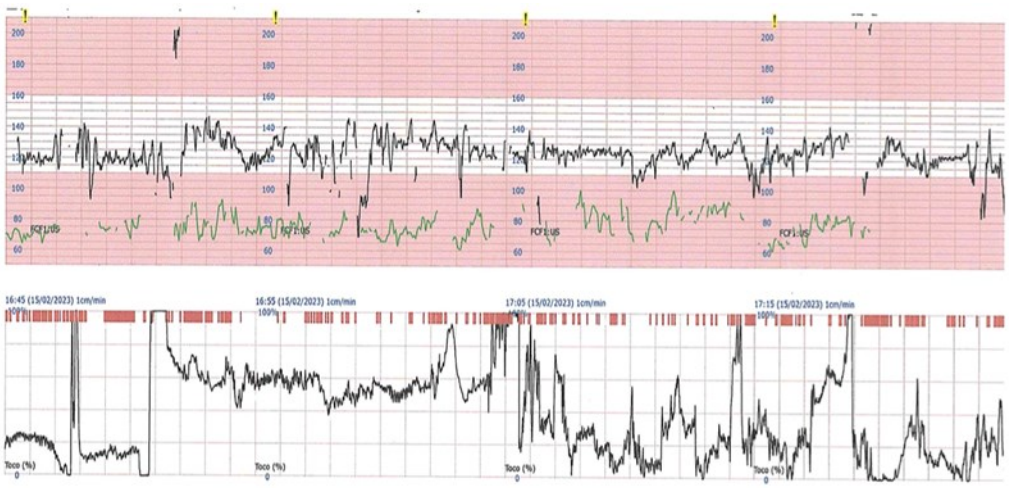
In the event that a labor continues to be slowed down and oxytocin causes pathological changes in CTG every time you try to reintroduce it, it may be useful to implement other maneuvers of intrauterine resuscitation (for example amnioinfusion if compression of the cord is suspected which is further worsened by oxytocin) and withdrawal from the fetal scalp, useful for evaluating the acid-base balance after oxytocin suspension. Obviously, if labor cannot progress without pathological changes in CTG, the only solution is to perform a caesarean section [33].

The management of oxytocin in labor is far from easy and is frequently implicated in medical-legal disputes involving obstetric personnel. The problems related to its inappropriate use in clinical practice depend mainly on the beginning of administration in conjunction with important pre-existing risk factors (for example meconium-dyed liquid, suspicion of corioamnionite or other parameters indicating the presence of an already partially compromised fetus) or suspected or frankly pathological cardiotocographic patterns. In addition, there may be a lack of recognition of tachysistoly and alterations of the FCF caused by oxytocin, as well as a cessation of continuous monitoring despite continuing with infusion, or failure to use tocolytics when CTG abnormalities are not resolved [7,12].

In conclusion, therefore, it is essential to adequately monitor the induced or enhanced labor with oxytocin, to be able to recognize promptly the alterations from this determined and know how to set the appropriate interventions to solve them.

Let's now analyze a cardiotocographic path.

Patient of 35 aa PARA 0010 to 40+5sg comes to the observation of obstetric first aid for discharge of amniotic fluid and contractions. After evaluation is hospitalized for prom in early labor with a cervical dilation of 4cm and drainage of LA tinto2. From the medical record describes a physiological pregnancy. Regular fetal growth at 50 β p. negative serology, negative rectal vagine tampon.



At the shift change of the obstetrical staff the expansion is at 8 cm a part presented to the SS is arranged infusion of 5 IU Syntocinon in 500 elect III low dose scheme.

Figure 2 baseline 125/130, variability retained >5bpm, CU present, NO decelerations

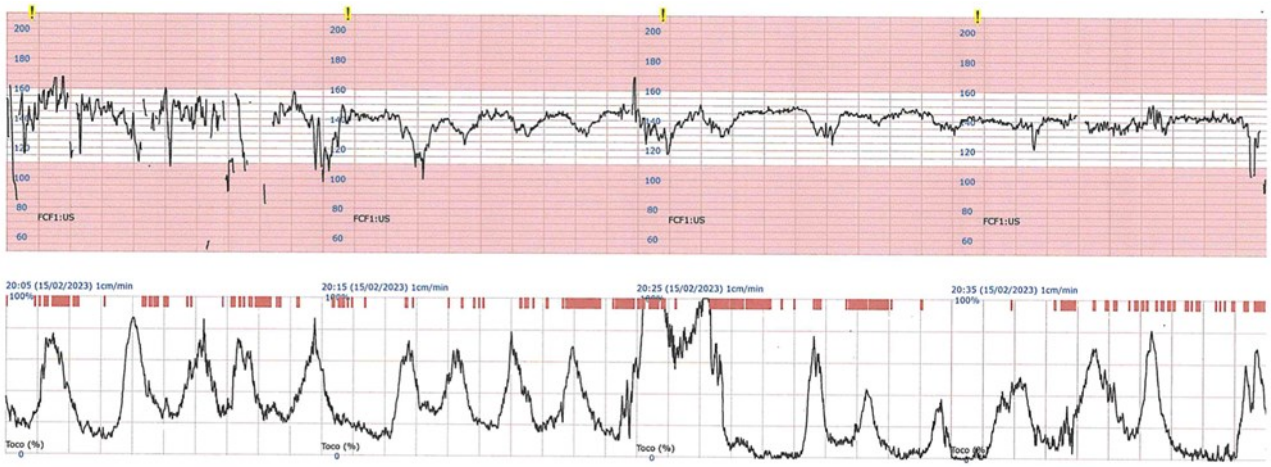


Figure 3 Start oxytocin infusion at low doses, LB increase at 145/150, variability preserved, decelerations present, CU 5 in 10 min

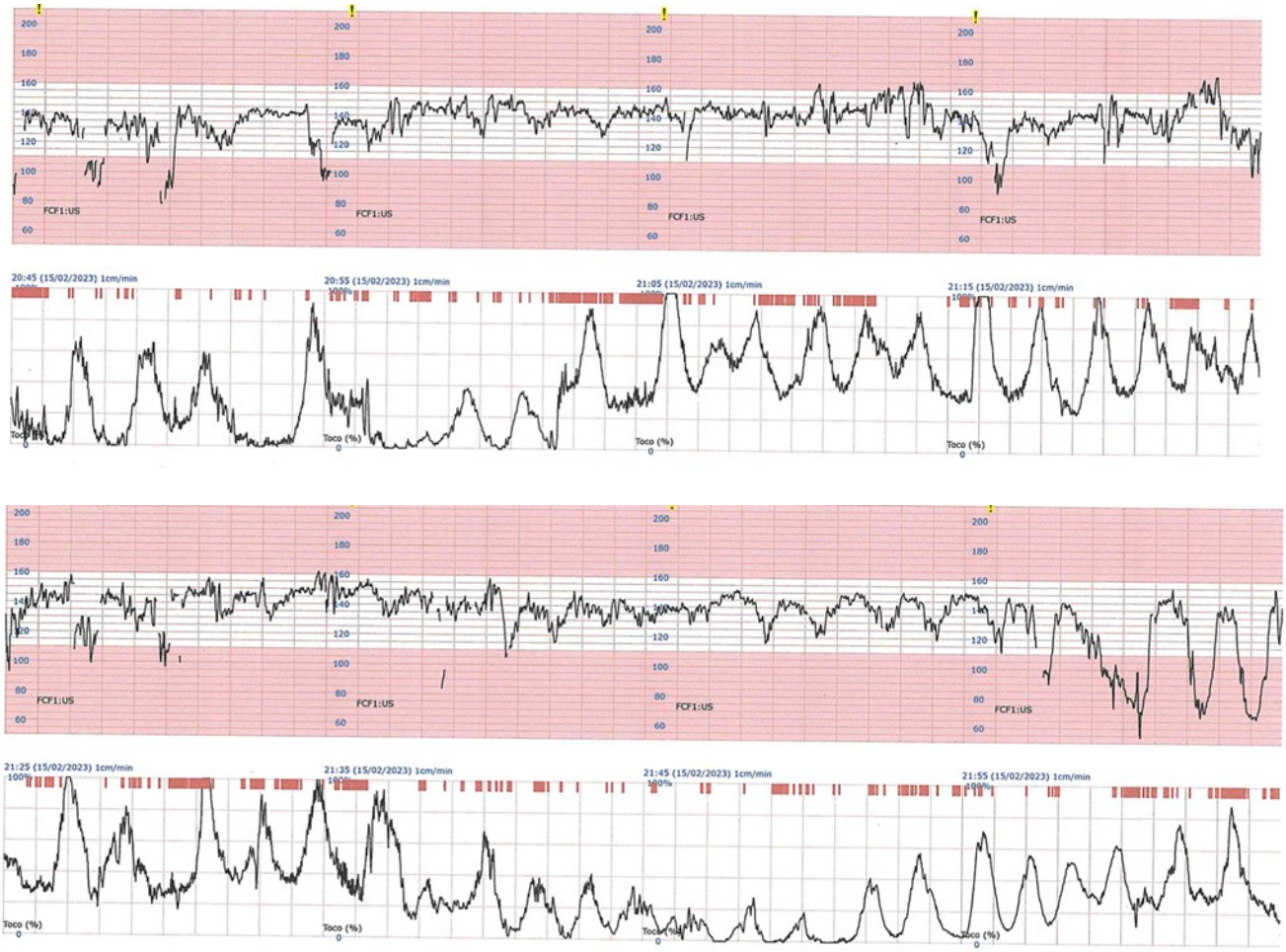


Figure 4/4 baseline increase to 150/160, retained variability, uterine contractions> 5 in 10 min

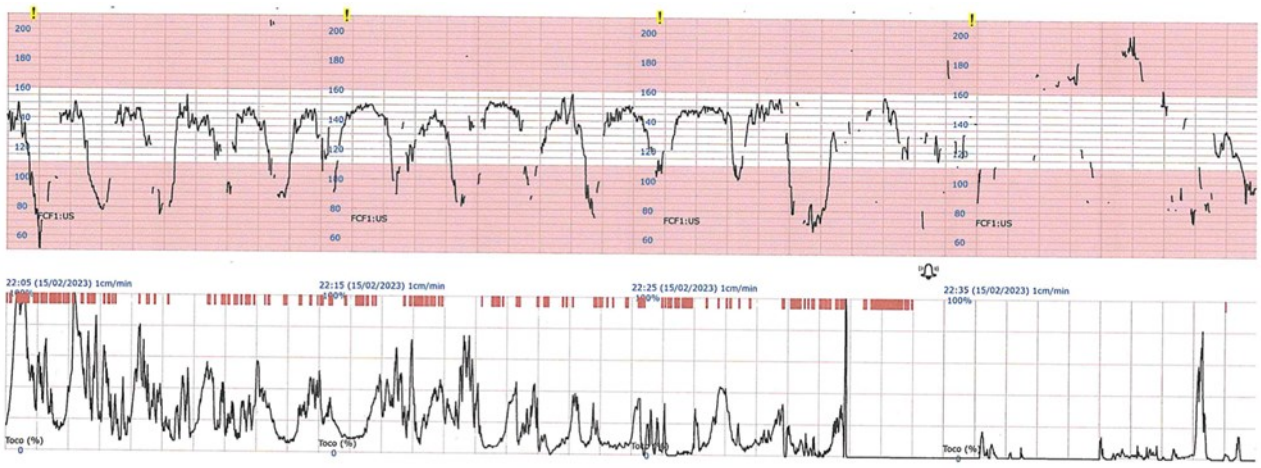


Figure 5 expulsion period, baseline 140/145, repetitive DECELERATIONS with no variability, TACHYSISTOLIA

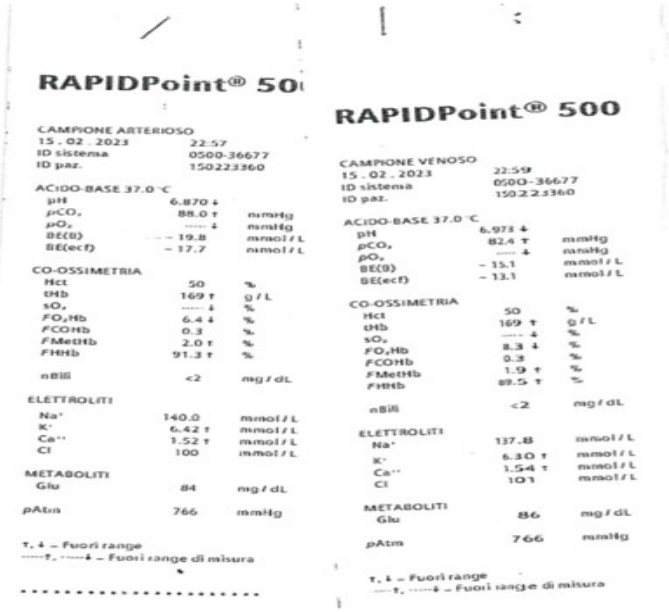


Figure 6: 3 attempts of failed kiwi sucker, emerging TC

This case clearly and clearly specifies what has been described above:

- Interpretation of the CTG track is subject to high inter- and intra-observer variability (B)
- Increased uterine activity is significantly associated with an increased incidence of an umbilical artery pH of 7.11 or lower.
- the intermittent and close contractile activity interrupts the blood flow to the intervillous space causing fetal hypoxemia which, if it persists, can manifest itself with alterations of the CTG of type II or III and finally in fetal acidosis.

There were no decisive interventions of tachysistoly that led to a neonatal hypoxia picture with a PH 6.87 and Abec -19.8 resulting in hospitalization in TIN and greater assists such as mechanical ventilation intubation and hypothermia.

Below we will analyze instead the resolution of a hypertone and subsequent outcome.

Patient of 38aa PARA 1001 to 41+2sg arrives at the hospital for the presence of uterine contractions. She is subsequently hospitalized for prodromi of labor in pregnancy characterized by polyhydramnios at term. After a normoinsorta pregnancy and normodecorsa is reported at 40sg of idiopathic polyhydramnios. Based on the visit that turns out to be 4cm. Intact sac no leaks in place is decided by induction, based on bishop's score, with amniorexi and oxytocin.

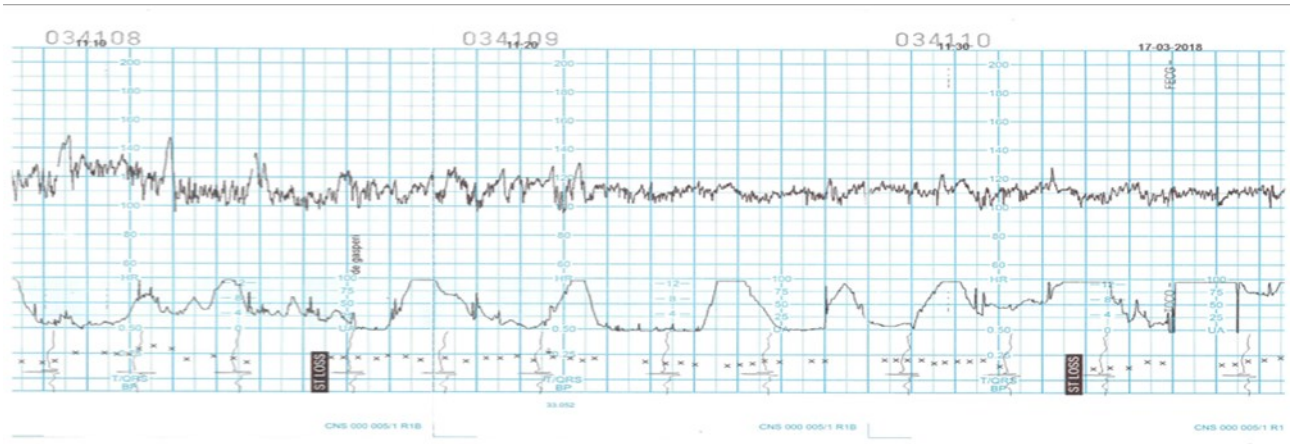


Figure 7: Baseline 115/120, retained variability >5, uterine contractions present, NO decelerations. Low dose oxytocin scheme ongoing

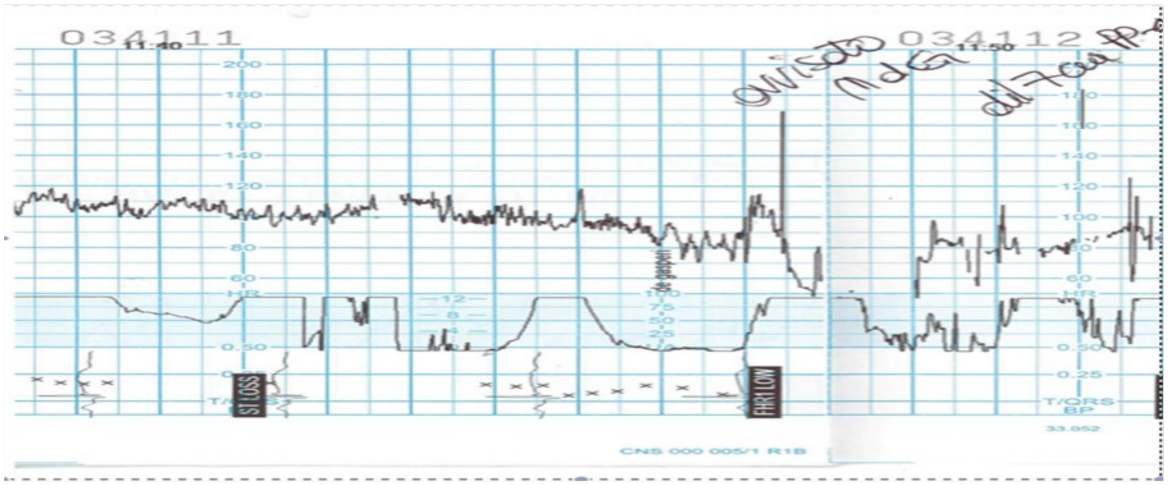


Figure 8 presence of hypertone, fetal bradycardia, conservative maneuvers: stop oxytocin, patient side sx, 1:10 intravenous mylene

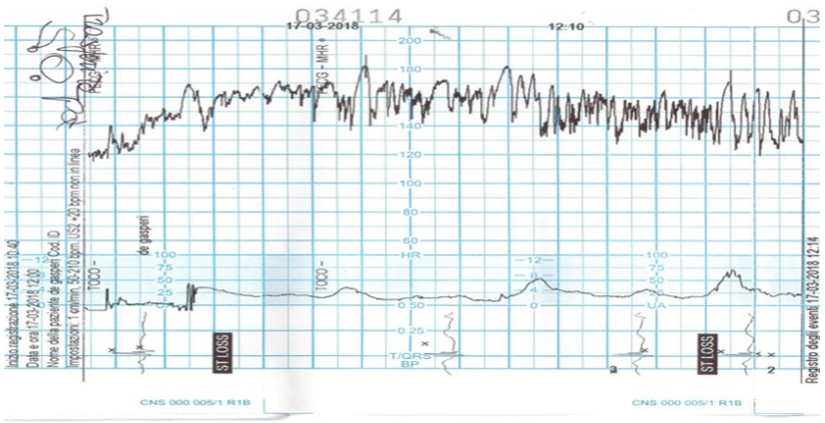


Figure 9: hypertone resolution and fetal fc with compensatory tachycardia fcm>100, fcf >150

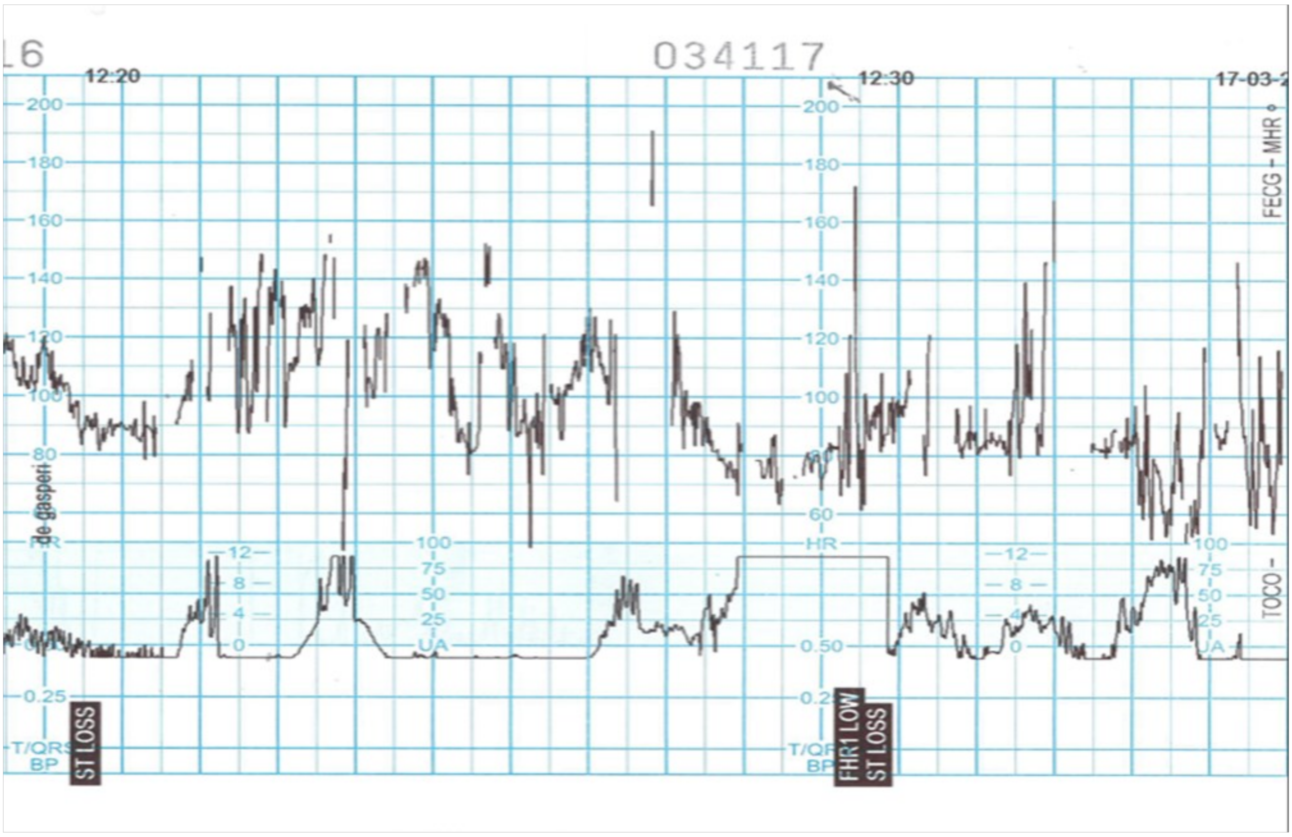


Figure 10: Expulsion period, complete expansion

SERIE ABL90 RADIOMETER

ABL90 Rianimazione 1 I393-C9DRC091N0002 12:36 17/03/2018
REFERTO PAZIENTE Siringa - S 65uL Campione 72866

Identificazioni

ID paziente	
Operatore	3339
Cognome paziente	caterina
Nome paziente	
Tipo di campione	Arterioso
T	37,0 °C
$FO_2(I)$	21,0 %

Stato acido-base			
↓ pH	7,256		[7,350 - 7,450]
↓ pO_2	27,7	mmHg	[83,0 - 108]
pCO_2	41,2	mmHg	[32,0 - 48,0]
↓ sO_2	54,9	%	[95,0 - 99,0]
↓ ABE,c	-6,5	mmol/L	[-2,0 - 3,0]
↓ $cHCO_3^-(P,st)$,c	16,9	mmol/L	[21,8 - 25,9]
↓ $cHCO_3^-(P)$,c	16,3	mmol/L	[21,0 - 29,0]
↑ ctHb	17,1	g/dL	[12,0 - 16,0]
Hct,c	52,3	%	[36,0 - 53,0]
Valori elettroliti e metaboliti			
↑ cK^+	4,8	mmol/L	[3,4 - 4,5]
↓ cNa^+	135	mmol/L	[136 - 146]
↑ cCa^{2+}	1,43	mmol/L	[1,15 - 1,29]
↑ cCl^-	109	mmol/L	[98 - 106]
↑ cGlu	126	mg/dL	[70 - 105]
↑ cLac	6,0	mmol/L	[0,5 - 1,6]
↑ ctBil	2,4	mg/dL	[0,1 - 1,0]
Anion Gap,c	7,2	mmol/L	[7,0 - 16,0]
Valori ossimetrici			
↓ FO_2Hb	52,5	%	[94,0 - 98,0]
↑ FCOHb	1,8	%	[0,5 - 1,5]
↑ FHHb	43,1	%	[0,0 - 5,0]
↑ FMetHb	2,6	%	[0,4 - 1,5]
Valori corretti con la temperatura			
$pO_2(a/A,T)$,e	27,8	%	
pH(T)	7,256		
$pO_2(T)$	27,7	mmHg	
$pCO_2(T)$	41,2	mmHg	
↓ ctO_2,c	12,5	Vol%	[15,8 - 22,3]
p50,c	25,69	mmHg	[24,00 - 28,00]

Note

↑	Valore/i sopra l'intervallo di riferimento
↓	Valore/i sotto l'intervallo di riferimento
,c	Valori calcolati
,e	Valori stimati
	0293: Ossi compensata per HbF

Figure 11: kiwi operative delivery, Apgar 9/10/10, ph 7.25 Abec -6.5

In this second proposed case, the outcome is clearly different in that maneuvers have been undertaken to resolve hypertone, among other things induced by the oxytocin introduced. Also in this case we talk about:

- Interpretation of the CTG track is subject to high inter- and intra-observer variability (B)
- the intermittent and close contractile activity interrupts the blood flow to the intervillous space causing fetal hypoxemia which, if it persists, can manifest itself with alterations of the CTG of type II or III and finally in fetal acidosis.

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