INTRAPARTUM ASSESSMENT OF FETAL WELL-BEING

1. INTRODUCTION

During labour, the risk of oxygen compromise in the fetus increases, as uterine contractions produce transient decreases in the flow of blood to the placenta, which may lead to interruptions in gas exchange. Certain degrees of hypoxemia and acidosis are normal in healthy fetuses. In fact, there is a physiological decrease in fetal pH (7.25 in the umbilical artery), without representing an immediate or long-term risk of fetal complications.

Fetal hypoxia is the deficiency of oxygen supply to the fetal organs secondary to the deterioration of gas exchange. When this condition is persistent, it progresses to hypoxic acidity. Hypoxia and, ultimately, acidosis, are the major contributors to the development of cerebral palsy, hypoxic-ischemic encephalopathy, and other morbid situations of the neonate.

The risk of loss of fetal well-being is determined by the presence of clinical signs or symptoms suggestive of being secondary to hypoxic acidosis and that condition clinical behaviour.

Diagnosis of acidosis (values in umbilical artery):

- pH <7.00.
- pCO2 >75 mmHg.
- Base deficit >12 mmHg.
- Lactate >10 mmol/L.

Hypoxic acidosis has two clinical-analytical forms that, in practice, are usually combined in variable degrees:

- **Respiratory acidosis:** decreased pH, increased pCO2 and a normal base deficit. It reflects the alteration in the exchange of gases but in a limited time. It can happen very quickly, but usually resolves after birth, when the newborn eliminates the accumulated CO2 through respiration. Therefore, postnatal complications are rare, and the prognosis is excellent. If there is a more persistent deterioration in the gas exchange, compensatory physiological mechanisms are activated to increase the availability of O2.

- **Metabolic acidosis:** when the oxygen deficit is prolonged, the fetal metabolism changes to anaerobic and this causes a decrease in pH, lactate accumulation and an increase in the base deficit (>12 mmHg). It occurs in 2 % of the deliveries and the majority of these children will be asymptomatic (75 %). Hypoxic-ischemic encephalopathy is the clinical manifestation of metabolic acidosis at the central nervous system (CNS) level.

2. OBJECTIVES OF THE CONTROL OF INTRAPARTUM FETAL WELL-BEING

The objective of intrapartum fetal monitoring is the early detection of the loss of fetal well-being secondary to fetal hypoxia, and the application of effective measures to anticipate the resulting perinatal mortality and/or morbidity.

Fetal heart rate (FHR) is modulated by the fetal brain via the sympathetic and parasympathetic nervous systems. Changes in normal FHR patterns form the basis for intrapartum FHR monitoring.

3. METHODS OF CONTROL OF FETAL WELL-BEING INTRAPARTUM: CARDIOTOCOGRAPHY

Monitoring of the **FHR** will always be carried out through **external monitoring.**

The exceptions for internal monitoring are cases of high obstetric risk, in which the quality of the record is insufficient for its evaluation, or when there is suspicion of fetal arrhythmia. Contraindicated if:

- HIV, HBV, and HCV positive pregnant women.
- Suspected fetal haematological alteration < 34 weeks.

Monitoring of **uterine dynamics** (UD) can be **external or internal**.

- External control of UD: the tracing allows us to objectify the frequency of the contractions and the relationship between contractions and FHR. It is the method of choice.
- Internal control of UD: allows a precise assessment of the recording of uterine tone at rest, and of the intensity, duration, and frequency of contractions. It is indicated in cases of lack of progression of labour in which it is desired to safely monitor the uterine dynamics (up to 200 MU in the dilation phase and 250 MU in the expulsive phase), in inductions with an increased risk of uterine rupture and in obese patients where abdominal transmission is not adequate.

4. INTRAPARTUM ELECTRONIC FETAL MONITORING

Intrapartum fetal monitoring can be **intermittent or continuous.**

4.1. INTERMITTENT FETAL MONITORING

Intermittent monitoring compared to continuous monitoring does not show significant changes in perinatal outcomes (mainly acidemia, cerebral palsy and perinatal mortality) but reduces the rate of caesarean sections that are necessary due to a risk of loss of well-being, as well as the rate of instrumented deliveries and the need for epidural anaesthesia. Therefore, intermittent auscultation is the method of choice in low-risk pregnant women.

Intermittent auscultation should be performed using Doppler (only in very selected cases could it be done using a Pinard horn stethoscope). The latter will be an option when there are sufficient staff available in the delivery room.

CLASSIFICATION

- Normal FHR: FHR 110-160 beats per minute (bpm)
- Abnormal FHR: FHR >160 bpm or <110 bpm or presence of decelerations

FREQUENCY of the recommended auscultation is the following:

In the first period of labour: every 15-30 minutes for 60 seconds in the immediate post contraction period, or windows of 20 minutes will be made every hour.

In the second period of labour: the risk of fetal acidosis is higher and continuous monitoring of FHR will be carried out. However, if it is decided to perform an intermittent auscultation, agreed with the woman, this will be done every 5 minutes for 60 seconds after a contraction.

ASSESSMENT

The FHR assessment will be carried out by the professional delivering the baby and must always be recorded in the clinical history every hour.

ATTITUDE if FHR is abnormal:

1. Place the patient in right or left lateral decubitus.
2. Assess maternal constants: HR, BP, Ta. If hypotension, administer crystalloids IV. If maternal fever (>38 ºC) administer antipyretics (Paracetamol 1 g IV).
3. Perform a vaginal examination to rule out umbilical cord prolapse.
4. Ensure the persistence of an abnormal HR by auscultating the HR again in the next contraction. If abnormal HR persists, continuous HR monitoring will be performed.

4.2. CONTINUOUS FETAL MONITORING

Continuous fetal monitoring is the method of choice in pregnant women with high risk or very high obstetric risk (see Appendix 1).

It will also be the method of choice if:

- Maternal vaginal bleeding during labour.
- Intrauterine infection/chorioamnionitis.
- Induction of labour or stimulation of labour (using oxytocin).

- Uterine hypertonia or tachysystole.
- Significant meconium in amniotic fluid.
- Gestation > 41 weeks.
- Maternal pulse >120 bpm on 2 occasions 30 minutes apart.
- Temperature >38 ºC or >37.5 ºC on 2 occasions separated by 1 hour.
- Referred pain other than contractions.
- Epidural analgesia.
- Pregnant women of low obstetric risk with anomalous FHR during intermittent auscultation.

5. INTERPRETATION OF THE CARDIOTOCOGRAPHIC RECORD (CTG)

When evaluating electronic fetal monitoring, it is of great importance to use a standardised nomenclature in order to minimise differences in its interpretation (Appendix 2, examples of CTG).

5.1. Generalities

- The date and time must be correctly set.
- Correct identification of the patient.
- It is necessary to record in the obstetric clinical history-partogram any intrapartum event that may affect FHR (vaginal touch, amniorrhexis, fetal pH extraction, epidural anaesthesia, etc.).
- Periodic reassessment considering that childbirth is a dynamic process and that the CTG category can change.
- The CTG must always be interpreted according to the clinical context of the patient (gestational age, administration of drugs, etc.).

5.2. Evaluation

The CTG will be evaluated every hour and must be recorded in the partogram. When evaluating a cardiotocographic trace, the following aspects must be taken into account:

1. Uterine contractions
2. Baseline FHR
3. Baseline FHR variability
4. Presence of accelerations
5. Periodic or episodic decelerations
6. Changes in the trend of FHR patterns over time

- **Uterine contractions:**

To quantify the uterine contractions, the number of contractions is evaluated in a 10-minute window, making an average of 30 minutes. The presence or absence of associated FHR decelerations should be assessed.

- Normal: 2 - 5 contractions in 10 minutes, averaged over a 30-minute window.
- Polysystole: >5 contractions in 10 minutes, averaged over a 30-minute window.
- Bradysystole: <2 contractions in 10 minutes, averaged over a 30-minute window.

- Hypertonia: contraction of more than two minutes duration or if uterine relaxation is not achieved after contraction (uterine tone > 12 mmHg if internal recording of uterine dynamics).

Applies to both spontaneous and induced contractions.

- **Basal FHR:**

Baseline FHR is determined by the approximate average FHR per minute, over a 10-minute segment excluding accelerations and decelerations.

- Normal FHR: baseline FHR between 110 and 160 bpm.
- Bradycardia: basal FHR <110 bpm.
- Tachycardia: basal FHR >160 bpm.

In case of prematurity, it should be noted that they tend to have higher basal FHR lines than term and post-term fetuses. Apart from that, β-mimetics and maternal fever are the most frequent causes of increased FHR.

- **FHR variability:**

FHR is defined as the variation in the amplitude of the baseline FHR. It is the measure of the difference between the minimum and maximum FHR of a segment without accelerations and decelerations.

- Normal: amplitude between 5-25 bpm.
- Absent: undetectable amplitude.
- Minimal: amplitude between undetectable and ≤ 5 bpm.
- Marked: amplitude > 25 bpm.

Fetal cardiac variability is episodic and may be intermittently minimal, even in a healthy fetus, due to fetal sleep, drugs (narcotics, sedatives, beta-blockers, magnesium sulphate, betamethasone), prematurity, fetal tachycardia, or congenital anomalies.

- **Accelerations**

Accelerations are defined as a transient, sharp increase in FHR above baseline. A sharp increase is defined as an elevation in FHR that occurs in < 30 seconds from the start of the acceleration to the maximum peak of the acceleration. To be considered an acceleration, the maximum peak must be ≥ 15 bpm above baseline (10 bpm in gestations < 32 weeks) for ≥ 15 seconds from onset to return of baseline and < 2 minutes.

- Prolonged acceleration: ≥ 2 minutes and ≤ 10 minutes.
- An acceleration lasting > 10 minutes is a change in baseline.

The presence of ≥ 2 accelerations in 20 minutes is suggestive of fetal well-being. Absence of accelerations with other parameters within normality is of uncertain significance.

Absence in: fetal sleep (transient), chronic hypoxia, CNS depressants (sedatives and narcotics), cerebral haemorrhage and infection.

- **Decelerations**

They consist of a transient drop in FHR below baseline of > 15 bpm for 15 seconds. The decelerations reflect the fetal response to hypoxia or stress mechanism in labour to protect against cerebral ischaemia or hypoxic myocardial damage.

Decelerations fall into 4 categories; early, late, variable, or prolonged. Combinations of all are possible.

Early deceleration

- Gradual decrease in FHR associated with a uterine contraction: the onset, nadir and recovery of the deceleration coincide with the onset, peak and end of the contraction, respectively.
- Visually they are symmetrical in the contraction (in some cases they present a mirror image).
- They are associated with fetal head compression, are considered benign and inconsequential (generally not associated with fetal acidosis). They account for only 2% of decelerations.

Late deceleration

- Gradual decrease in FHR and return to basal FHR, from onset to minimum FHR of > 30 seconds duration.
- The onset of the deceleration does not coincide with the onset of the contraction, is delayed (onset after 20-30 seconds from the onset of the contraction), and recovery from the deceleration occurs after the end of the contraction.
- Associated with uteroplacental insufficiency and hypoxia.

Variable deceleration

- Abrupt decrease in FHR (interval of < 30 seconds from the onset of deceleration to the onset of the nadir of deceleration).
- FHR decrease ≥ 15 bpm, lasting ≥ 15 seconds and < 2 minutes.
- These are the most common and are a response of the FHR to cord compression.

a) *Typical or uncomplicated:* they consist of an initial acceleration, a rapid deceleration of the FHR at its lowest point, followed by a rapid return to baseline, with a secondary acceleration. They are not associated with poor perinatal outcome.

b) *Atypical or complicated:* if they have any of the following characteristics:

- < 70 bpm > 60 seconds.
- Loss of baseline FHR variability <5 bpm after deceleration.
- Loss of initial transient ascent.
- Biphasic decelerations.
- Prolonged secondary acceleration (>20 bpm or lasting >20 seconds).
- Basal tachycardia or bradycardia.
- Continuation of baseline FHR at a lower level than before deceleration.
- Slow return to previous baseline FHR.

Prolonged deceleration

- Defined as a decrease in FHR from baseline of ≥ 15 bpm, lasting ≥ 2 minutes, but < 10 minutes.

*Decelerations are defined as recurrent if ≥ 50% of uterine contractions occur in a 20-minute window. They are defined as sporadic or occasional if < 50% of uterine contractions.

** Other CTG patterns:

- SINUSOIDAL: a very rare, pathological pattern defined by a regular oscillation of baseline variability reminiscent of a sine wave, minimum 10 minutes in duration, with fixed periods of 3-5 cycles per minute and an amplitude of 5-15 bpm above and below baseline that persists ≥ 20 minutes. There is no variability. It is associated with suspected loss of fetal well-being and fetal death.

6. CLASSIFICATION OF THE CARDIOTOCOGRAPHIC RECORDING

FHR patterns are defined by baseline FHR characteristics, variability, presence of accelerations and decelerations. FHR tracings are dynamic and transient, requiring frequent assessment.

	NORMAL	ATYPICAL	ANNORMAL
Baseline	110-160 bpm	100-110 bpm 160-180 bpm (>30' and <90')	<100 bpm 160-180 bpm >90' >180 bpm >20' Sinusoidal pattern >20'
Variability	5-25 bpm	<5 bpm (>30' and <90') > 25 bpm 15-25'	<5 bpm >90' >25 bpm >25'
Decelerations	No decelerations Variable typical <90' Early	Variable typical >50% contractions >90' Occasional late (<50% contractions >30') Prolonged and only <3' Variable atypical <50% contractions >30' >50% contractions <30'	Recurrent late (>50% contractions >30') Prolonged and only >3' Variable atypical >50% contractions >30'

1. **Normal CTG:** 3 normal criterion.
2. **Suspicious CTG:** one atypical criterion and the rest normal.
3. **Pathological CTG**: ≥ 2 atypical criterion or ≥ 1 abnormal criterion.

7. PERFORMANCE ACCORDING TO THE CTG CLASSIFICATION

FHR tracings must be interpreted in the clinical context and their inclusion in a certain category is only useful for the period under study.
Depending on the CTG classification, additional tests will be necessary to ensure fetal well-being.

It is important not to make decisions based on the CTG alone, but to individualise the clinical approach by considering other factors such as:

- Maternal signs and symptoms, presence of fever.
- Maternal preferences.
- Presence of meconium, bleeding.
- Drugs administered (magnesium sulphate, etc.).
- Obstetric conditions, evolution, and stage of labour - parity, history of previous caesarean section.
- Estimated fetal weight, weight of previous children.
- Previous history/number of calcium pH tests carried out.

7.1. Normal CTG

It is highly predictive of normal fetal acid-base status at the time of observation. It should be re-evaluated every hour and classified by recording it on the partogram.

7.2. Suspicious CTG

Not predictive of fetal acidosis. They require continuous monitoring and reassessment, taking into account the associated clinical circumstances.
Additional tests or general intrauterine resuscitative measures may be necessary to ensure fetal well-being.

General measures

- Evaluate maternal constants: heart rate, blood pressure, temperature.
- Right or left lateral decubitus.
- Determination of glycaemia in diabetic patients or if prolonged fasting (> 8 hours).
- If maternal fever, administer antipyretics.
- If hypotension (systolic blood pressure 20% lower than baseline or SBP < 90 mmHg) or suspected hypovolaemia (absolute diet, serum deficiency, vomiting or sympathetic nerve block), crystalloids IV +/- ephedrine should be administered.
    - Crystalloids: rapid infusion of 500-1000 mL of Ringer's Lactate or saline.
    - Ephedrine: direct IV administration in 5 mg bolus.
- Oxygen therapy should only be considered in patients with hypoxaemia criterion.
- If prolonged fasting or capillary glycaemia < 60 mg, administer 5% glucose saline.
- Assess for hypertonia (1 contraction lasting more than 2 minutes) or polysysystole (> 5 contractions in 10 minutes). In these cases, consider stopping/reducing oxytocin perfusion and/or administering betamimetics (Ritodrine IV 60 ml/h=200 mcg/min).

- Perform a vaginal examination to rule out cord prolapse, determine rapid dilatation of the cervix or descent of the fetal head.

Stimulation of the fetal scalp

If, despite these measures, an atypical CTG persists, fetal scalp stimulation should be performed during the vaginal examination (this may reduce the need for fetal calyceal micro blood sampling by up to 50%). Stimulation should be gentle and excessive digital pressure should be avoided as it may cause vagal bradycardia and should not be performed during a deceleration, as it prevents the establishment of a sympathetic response during stimulation.

The presence of an acceleration > 15 bpm for >15'' almost always ensures the absence of fetal acidaemia (< 32 weeks, a rise of 10 bpm for 10'' is considered sufficient). The presence of an acceleration would correspond to a pH >7.20 in 90% of cases; otherwise < 7.20 in about 50%.

Induced accelerations exclude hypoxic damage in the same way as spontaneous accelerations. Above all, it serves to see the nature of decreased variability (hypoxia *vs.* fetal). In other CTG patterns, its use is more questionable.

7.3. Pathological CTG

These are associated with alterations in fetal acid-base status at the time of observation. They require prompt evaluation and exclusion of acute accidents.

In the case of pathological CTG, the individual should assess the case by taking into account the clinical situation of the woman and the cervical conditions. While intrauterine resuscitation attempts are being made, the delivery should be prepared and the anaesthesiologists and neonatologists should be informed.

8. BIOCHEMICAL CONTROL: ACID-BASE EQUILIBRIUM (FETAL SCALP BLOOD SAMPLING)

Fetal scalp blood sampling is an intrapartum procedure intended to assess the presence and degree of fetal acidemia by analysing fetal capillary blood. An amnioscope with a light source is used to expose the fetal scalp, which is cleansed of blood, mucous, and amniotic fluid.

Indications

- Suspicious CTG in which fetal head stimulation does not produce accelerations.
- Pathological CTG.

Described complications (rare but serious)

- Infection, haemorrhage, cerebrospinal fluid leakage.

Contraindications
- Maternal infections: HIV or hepatitis.
- Fetuses at increased risk of a bleeding diathesis: thrombocytopenia, haemophilia.
- Gestational age < 34 weeks.

If impossible to perform or contraindicated, in case of a pathological CTG, administration of IV betamimetics and immediate fetal extraction will be performed.

There was recent evidence that fetal blood sampling does not improve outcomes for women and babies compared with CTG alone or compared with CTG in combination with fetal scalp stimulation. Intrapartum fetal scalp blood sampling to measure pH (and base excess/deficit) or lactate has not been clearly proven to reduce emergency caesarean deliveries or operative vaginal births or to improve long-term perinatal outcome.

For this reason and many others, including quality control issues, cost, patient discomfort, and sample failure rates up to 10 percent, fetal scalp blood sampling is not entirely recommended

Recent international committees were aware, based on their knowledge and experience, that the time taken to carry out fetal blood sampling can delay appropriate expedition of birth, and that it can be an unpleasant procedure for the woman, especially in the absence of an effective epidural.

9. IV BETAMIMETIC ADMINISTRATION (INTRA-UTERAL RESUSCITATION)

Indications
- Sustained fetal bradycardia (FHR <100 bpm, 5-7 minutes), whether or not related to uterine hypertonia or polysysystole.

Dosage and method of administration
- Ritodrine 10 mg/ml. Total ampoule 50 mg in 5 ml.
- 2 cc of Ritodrine in 100 cc of glucose serum 5%. Rate of 200 mcg/min (60 mL/h).
- Always administer by infusion pump.

Ritodrine infusion will be saturated if: maternal HR > 140 bpm, systolic blood pressure (SBP) < 80, sensation of faecal pain, chest tightness or other symptoms attributable to side effects of betamimetics.

Absolute contraindications of Ritodrine
- Serious cardiovascular pathology.
- Pulmonary pathology (pulmonary hypertension).

Relative contra-indications to Ritodrine

- Hyperthyroidism in treatment.
- Pregestational diabetes (strict glycaemia control will be necessary).
- Repeated migraines.
- Multiple pregnancy.

10. FETAL BLOOD-ACID-BASE BALANCE

It shall be done systematically in all parts as it allows us to carry out adequate care of the newborn, establish the severity and type of fetal acidosis (metabolic or respiratory) and plan further management.
It is important to obtain two samples (arterial and venous) to study the acid-base balance (pH, base, deficit, PCO2, HCO3, pO2, O2 saturation) and values of cord blood parameters (see Appendix 3).

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APPENDIX

APPENDIX 1

High risk

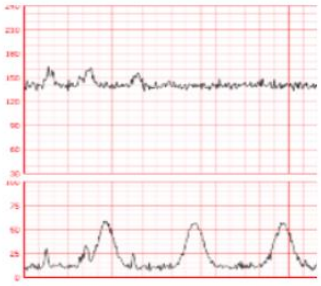
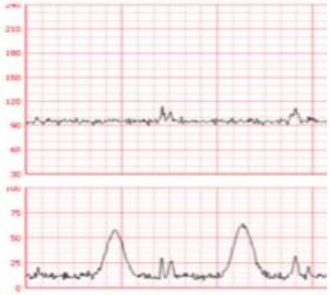
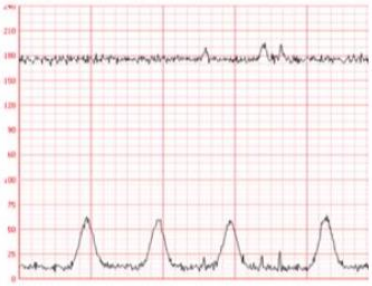
- Morbid obesity
- History of miscarriage
- A history of uterine cervical incompetence
- Previous chromosomal abnormality
- Previous ectopic pregnancy
- Previous gestational trophoblastic disease
- Previous preterm birth
- Endocrinopathies
- Cardiovascular disease WHO III
- Pre-pregnancy hypertension
- Twins
- Gestational diabetes corrected with diet and insulin
- Mild pre-eclampsia
- Maternal infection diagnosed during pregnancy
- Isoimmunization
- Severe current mental pathology
- Severe anaemia
- Suspected fetal malformation

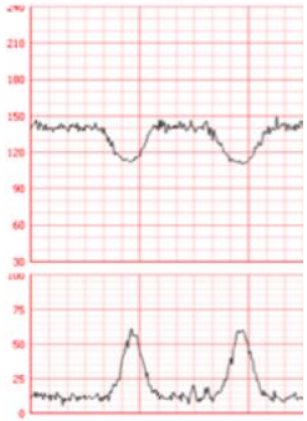
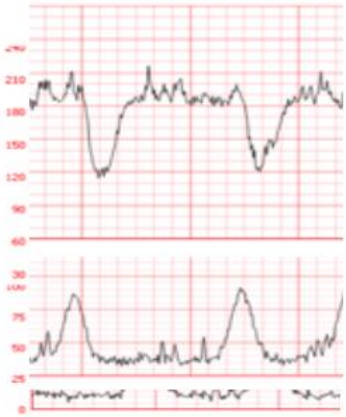
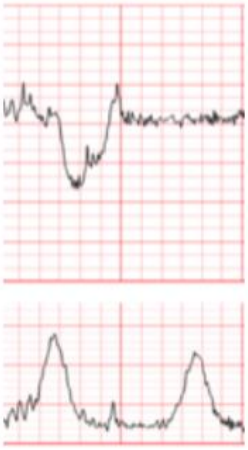
Very high risk

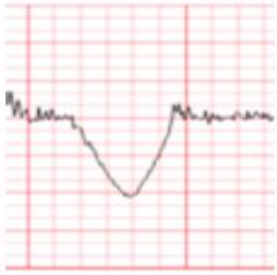
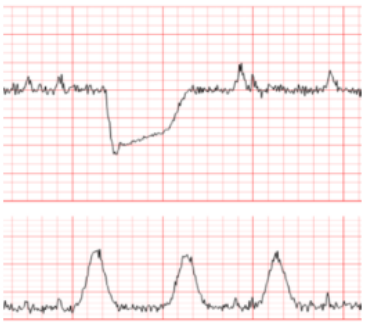
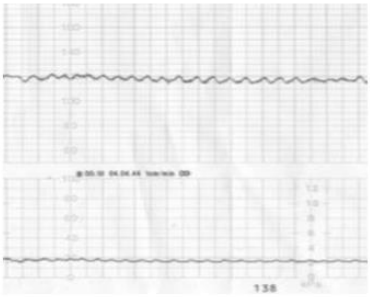
- Severe associated pathology
- Previous perinatal death
- Diabetes 1 or 2
- Cardiovascular risk WHO IV
- Alcohol and/or other drug dependence syndrome
- Diagnosed uterine malformations
- Multiple gestation
- Intrauterine growth retardation
- Confirmed fetal malformation
- Abnormal placenta status
- Severe pre-eclampsia
- Threatened preterm labour
- Preterm rupture of membranes

APPENDIX 2

CTG examples

NORMAL	BRADYCARDIA	TACHYCARDIA
		

EARLY DECELERATION	TYPICAL VARIABLE DECELERATION	ATYPICAL VARIABLE DECELERATION
		

LATE DECELERATION	PROLONGED DECELERATION	SINUSOIDAL PATTERN
		

APPENDIX 3

	ARTERIAL BLOOD	VENOUS BLOOD
pH	7.20-7.34	7.28-7.40
pCO2 (mmHg)	39.0 – 61.4	32.8 – 48.6
HCO3 (mEq/L)	18.4 – 25.6	18.9 – 23.9
Base excess (mEq/L)	-5.5 – 0.1	-4.4 – 0.4