The Physiology of Fetal Surveillance

The Green Book of Neoventa Part I

K G Rosén
Annika Mårtendal

External stimuli

The fetus has the ability to sense and react to changes in its external and internal environment.

During a contraction, the fetus is pushed down the birth canal and an episodic increase in head pressure is seen. The CTG now displays early decelerations where the drop in heart rate matches the uterine activity curve. Another example is the arousal reaction caused by the squeezing and squashing of labour causing tachycardia.

With the mother on her back, there is a risk that the uterus will compress the abdominal veins. This reduces maternal placental blood flow and may cause fetal hypoxia. This results in a prolonged deceleration. The remedy is to turn the mother on her side to improve the maternal uterine blood flow. During the last phase of labour, a marked increase in eye bulb pressure is not uncommon causing a marked vagally induced bradycardia.

Increases in temperature

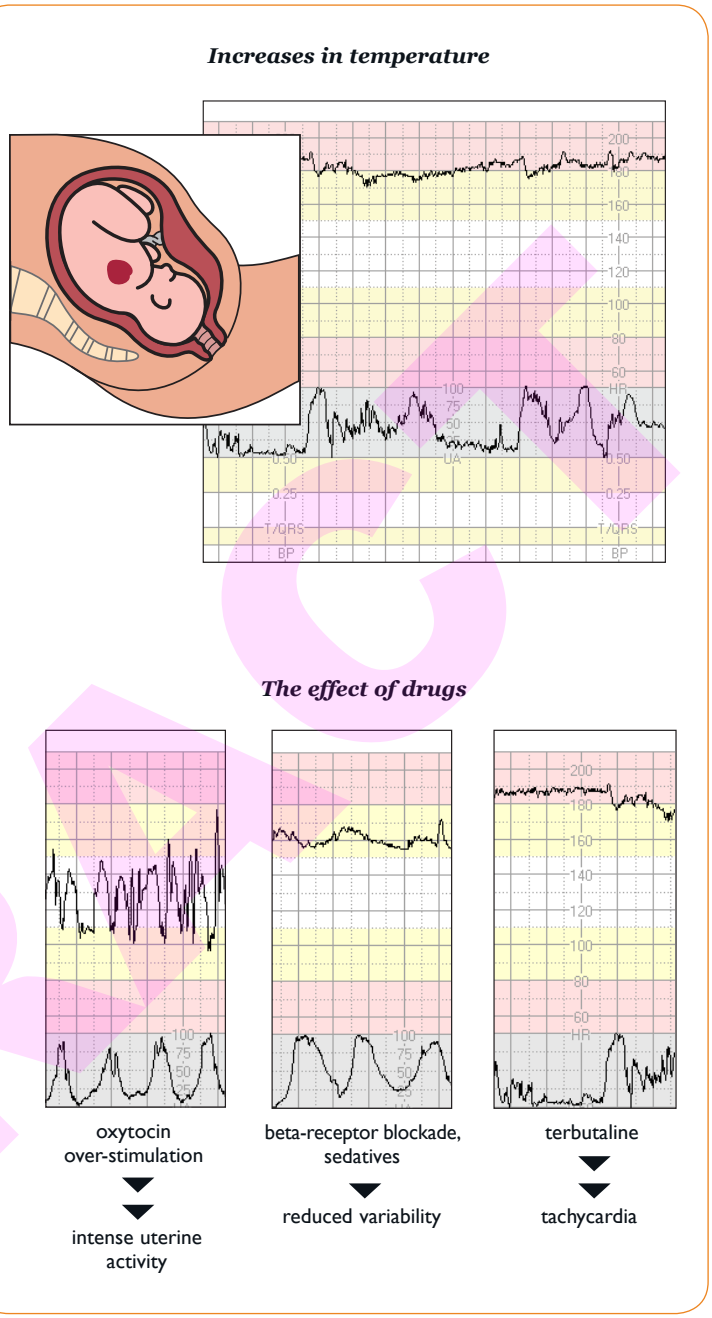
Labour is a physical stress for the mother. As with all physical exercise, water may be lost, which leads to a fluid deficit. This causes the mother to decrease her peripheral circulation because the blood volume is reduced. As a result, she reduces her ability to relieve herself of the extra heat generated by the exercise and fever may occur. The rise in temperature causes an increase in fetal metabolic rate and an increase in oxygen consumption and blood flow through the tissues. This may result in fetal tachycardia. The margins are reduced and the fetal ability to handle oxygen deficiency decreases. Appropriate treatment of maternal fever by increasing fluid intake and paracetamol treatment should make the tachycardia disappear. In the event of an ongoing infection, the ability of the fetus to handle asphyxia is markedly reduced. Furthermore, the combination of an infection + hypoxia may not cause substantial CTG changes otherwise noted with hypoxia per se.

The effect of drugs

As previously discussed, different drugs may not only affect the ability of the fetus to handle hypoxia but may also make CTG interpretation more difficult. There are numerous ways by which drugs could affect the heart rate and the ability of the fetus to handle oxygen deficiency.

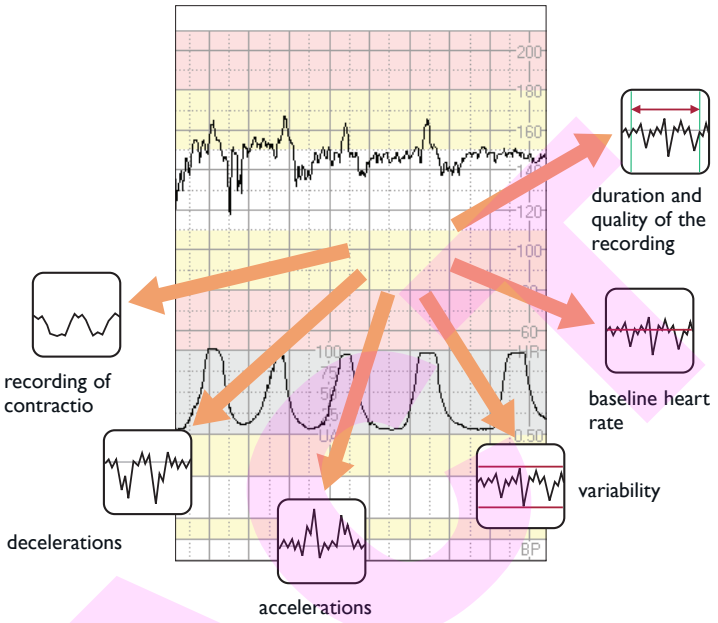
Overstimulation with oxytocin for example may cause hypoxia due to intense uterine activity. Betareceptor blockade and sedatives may cause a curtailed fetal response and reduced variability. Betareceptor activating drugs such as terbutaline may cause tachycardia. Local anaesthetics may be transferred to the fetus and cause fetal bradycardia as a sign of a direct effect on the myocardium.

Epidural anaesthesia may cause a lowering of the maternal blood pressure with reduced maternal blood flow and fetal hypoxia. If the mother is given any sedation, these drugs will be transferred to the fetus and will reduce its activity and CTG reactivity. Furthermore, drugs may be accumulated in the fetus and the potential effect of any drug would have to be considered when administered in conjunction with labour.



CTG Interpretation

To enable an accurate CTG analysis, the terminology has to be known and used appropriately.



DURATION AND QUALITY OF THE RECORDING

A minimum duration of 20 minutes is required for a CTG recording to be properly interpreted because of changes in sleep state and uterine activity. The speed of the recording is usually 1 cm a minute and there are 10 minutes between the printouts of the scaling. The fetal heart rate can be plotted between 50 and 210 bpm. Uterine activity is depicted in a range of 0 to 100 relative units when using a tocometer and 0 to 100 mm of mercury when applying an intrauterine pressure sensor. This is done according to the CTG standard applicable to Europe.

Good signal quality is absolutely essential to enable accurate interpretation. If there is poor signal quality, it is better to spend time improving the signal by replacing the scalp electrode or the toco sensor rather than trying to interpret erroneous data.

BASELINE HEART RATE

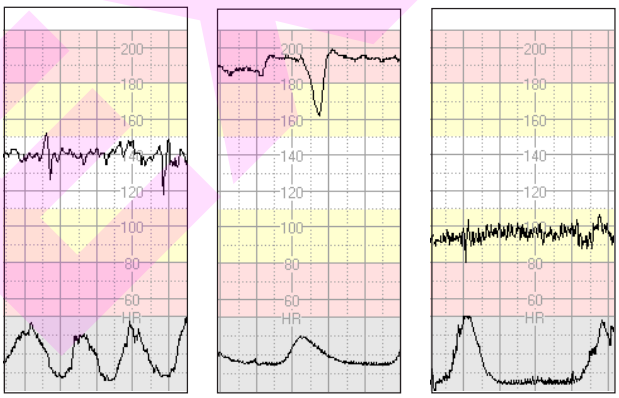
The baseline fetal heart rate is defined as a fetal heart rate recorded between contractions over a period of at least 10 minutes. This is most important in the presence of decelerations. The baseline heart rate reflects what is called the balance of the autonomic nervous system. As the fetus matures, the parasympathetic nervous system dominates, due to an increase in blood pressure, and there is a decrease in baseline heart rate.

A normal fetal baseline heart rate for a term fetus is defined as 110 to 150 bpm. Tachycardia is defined as a baseline heart rate greater than 150 bpm and bradycardia is defined as a baseline heart rate of less than 110 bpm.

VARIABILITY

The fetal heart rate normally displays beat-to-beat variations, which are not accelerations or decelerations. The so called bandwidth of these beat-to-beat variations could be used as a measurement of heart rate variability. This aspect of the CTG recording provides information about the ability of the central nervous system to monitor and adjust the cardiovascular system. This so called short-term variability may vary with time, depending on variations in sleep and activity. The same type of pattern, with a loss of heart rate variability, is one of the most important features when hypoxia is emerging. Reduced variability reflects an increase in sympathetic tone, but, when there is a complete loss of beat-to-beat variation, this may also depend on the inability of the myocardium to respond.

Baseline heart rate

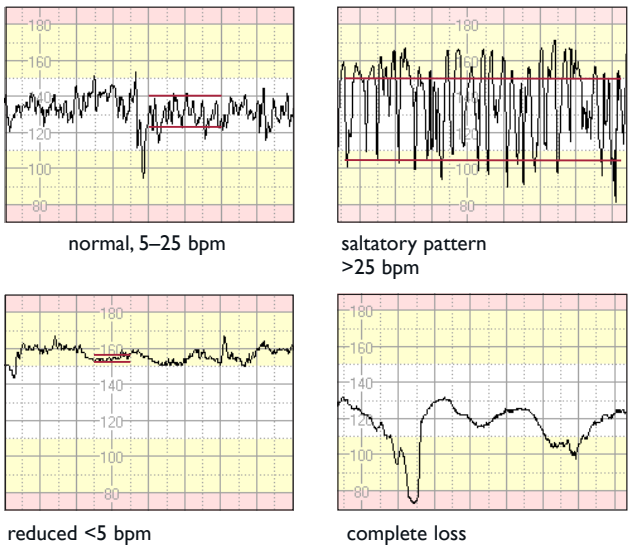


normal
110–150 bpm

tachycardia
>150 bpm

bradycardia
<110 bpm

Variability



normal, 5–25 bpm

saltatory pattern
>25 bpm

reduced <5 bpm

complete loss