1. <Title name>
   1. Analysis of physiological aspects of fetal ECG

Heart defects are among the most common birth defects and the leading cause of birth defect-related deaths. Every year, about one of 125 babies are born with some form of congenital heart defects. The defect may be so slight that the baby appears healthy for many years after birth, or so severe that its life is in immediate danger. Congenital heart defects originate in early stages of pregnancy when the heart is forming and they can affect any of the parts or functions of the heart. Cardiac anomalies may occur due to a genetic syndrome, inherited disorder, or environmental factors such as infections or drug misuse [1].

There are at least two ways of fECG assessment. The key features in are FHR rhythm-related, and FECG morphology related. The second one includes changes in ST and QT segments. It is known that QT interval reacts to situations of stress and exercise. It has been shown that a significant shortening of the QT interval was associated with intrapartum hypoxia irrespectively of changes in FHR [2], whereas in normal labor these changes do not occur.

However, for fECG recordings, it is unclear how robust these measures are, particularly when accompanied by:

* noise/artefacts;
* fetal movements;
* different electrode configurations;
* undesired distortions caused by extraction algorithms.

Moreover, even when using modern monitoring equipment like STAN [3], it is not possible to assess how well the morphology of the fetal signal is preserved. This because the reference (invasive FECG) is based on a different lead, which represents another projection of the cardiac electrical activity.

* + 1. Fetal QT interval feature

A small number of researches conducted on the association between the fetal QT interval and newborn outcome. Although, many studies note QT interval abnormalities during the fetal and newborn period with serious events, including sudden death [4].

A prolonged QT interval, either genetic or acquired, predisposes to ventricular tachycardia and sudden death. Changes in the QT interval have also been shown during exercise, stress, infection and heart failure [5]. A QT shortening was noticed in conjunction with an increase in T-wave amplitude. It seemed logical to assume that the QT shortening would depend on the ability of the fetal myocardium to enhance its performance in response to a catecholamine surge and on h-receptor activation known to elicit the rise in T-wave amplitude.

* + 1. Fetal ST interval feature

The ST interval comprises the ST segment and the T wave, and both relate to the repolarization of myocardial cells in preparation for the next contraction, an energy-intensive process. An increase in T-wave height (fig. 1), quantified by the T/QRS ratio, occurs when cellular energy production within myocardial cells begins to decline, that is, when the oxygen supply is inadequate to maintain metabolic activity so that cells are forced to generate energy by ß-adrenoceptor-mediated anaerobic breakdown of glycogen reserves.

ST interval depression indicates an imbalance between the endocardium and epicardium because of the difference between the lower blood perfusion pressure of the endocardium and the higher mechanical strain, which delays myocardial repolarization.

All the factors that modify the performance characteristics of the myocardial wall, including hypoxia, prematurity, infections, maternal fever, myocardial dystrophy, maternal diabetes and cardiac malformations may depress the ST interval.



Figure 1. Changes in fetal ST segment