Diagnostics of the state of the fetus during pregnancy and childbirth

The goal of in utero fetal monitoring is to prevent intrauterine death, the development of asphyxia and other complications that lead to neonatal morbidity.

Intrauterine hypoxia of the fetus arises as a result of impaired oxygenation of maternal blood (maternal cardiac insufficiency, hypotension, hypertension, hypovolemia, preeclampsia), impaired placental function (placental abruption), umbilical cord occlusion (prolapse, knot, compression) and/or fetal cardiovascular insufficiency (anemia, cardiac insufficiency). Impaired fetal oxygenation leads to lactate accumulation, lactic acidosis and organ disorders.

Non-invasive diagnostic methods of fetal hypoxia are based on the evaluation of secondary and non-specific manifestations of changes in the internal environment. A direct diagnosis of fetal hypoxia can only be established by invasive methods based on examination of fetal blood (during pregnancy - cordocentesis, during childbirth - blood sampling from the fetal scalp - a relatively poor predictor of hypoxia).[1].

Non-invasive methods of fetal monitoring

- fetal movements (clinically significant is a significant reduction or disappearance of fetal movements);
- biophysical profile, modified biophysical profile;
- cardiotocography - non-stress test and contraction stress test.

The main controversy of these methods is their low specificity (high rate of false positive findings).

Indications for fetal condition monitoring: maternal diabetes mellitus, hypertension, systemic lupus erythematosus, antiphospholipid syndrome, sickle cell anemia, cyanotic heart disease, hyperthyroidism, multiple pregnancy, IUGR, restriction of fetal movement, isoimmunization, oligo- or polyhydramnios, premature outflow of amniotic fluid, non-immune hydrops etc.[1]

Pathophysiology

Compensatory mechanisms of placental function disorder

Chronic lack of oxygen and nutrition leads to fetal growth restriction. The fetus mobilizes compensatory mechanisms and energy-saving physiological changes appear (restriction of fetal movements, reduction of fetal heart rate variability). With longer duration, redistribution of blood circulation occurs (reduction of peripheral resistance of cerebral vessels, increase of peripheral vascular resistance at the level of the umbilical arteries and aorta of the fetus). Fetal polycythemia develops, which increases the oxygen carrying capacity.[1]

Cardiotocography (CTG)

CTG evaluates the characteristics of fetal echoes in relation to the contraction activity of the uterus.

Short-term phenomena

- beat-to-beat oscillation and variability of fetal sounds during one minute;
- rating: 0-5/min. = silent, 5-10/min. = narrowed undulatory, 10-25/min. = undulating, > 25/min. = saltatory; sinusoidal = periodically repeating wave characteristics, 3-5/min.;

Medieval phenomena

- acceleration (increase in heart rate by at least 15/minute for at least 15 seconds; a sign of good fetal condition),
- deceleration
  - early - have the lowest point at the peak of uterine contractions and are caused by compression of the fetal head;
  - late - the lowest point is delayed beyond the peak of the contraction and they arise as a result of the redistribution of fetal blood circulation during the development of hypoxia;
  - variable decelerations, i.e. decelerations with a variable asymmetric shape are caused by compression of the umbilical cord;

Long-term phenomena

- basal frequency - normocardia: 120-160/min.[1]

An antepartum CTG recording lasting 20 minutes, which shows 2 or more accelerations, especially in connection with fetal movement activity (so-called reactive), indicates a good condition of the fetus. Fetal hypoxia is primarily evidenced by loss of oscillation variability and repeated late decelerations. Sinusoid is a typical finding for fetal anemia or hypoxemia of other origin. When evaluating the antepartum CTG recording, it is necessary to take into account the gestational age and possibly influence of heart action by illness or pharmacotherapy of the mother and pathologies of the fetus (FGR, anemia, dysrhythmia).

The conduction system of the heart develops between the 3rd and 6th week of pregnancy. Cardiac action is regulated by the autonomic nervous system depending on blood pressure and the partial pressure of oxygen and carbon dioxide. Parasympathetic fibers innervate the sinoatrial and atrioventricular nodes via the vagus nerve. The parasympathetic slows the heart action (chronotropic effect) and is responsible for the oscillatory effect (variability of R-R intervals). Sympathetic stimulates the release of noradrenaline, acceleration of heart action and increase in the force of muscle contraction (inotropic effect). It is responsible for periodic accelerations. The development of the parasympathetic leads to a gradual decrease in the heart rate of the fetus with gestational age and an increase in the variability of the heart action after the 24th week of pregnancy. After the 24th week of pregnancy, 50% of healthy fetuses show acceleration, at 30th week 95% of fetuses. Until the 30th on the first day of pregnancy, the accelerations last < 10 minutes and the heart action increases by 10/minute, after 30 weeks they last 15 seconds and the action increases by 15/minute.[1]

Evaluation of antepartum and intrapartum CTG according to FIGO[1]			
	Physiological	**Suspicious**	**Pathological**
Basal frequency	110-150/min.	150-170/min. 100-110/min.	> 170/min. < 100/min.
Amplitude of variability	10-25/min.	5-10/min. for ≥ 40 min. > 25/min.	< 5/min. for ≥ 40 min. sinusoid for ≥ 20 min.
Deceleration	**Antepartum:** absent **Intrapartum:** early decelerations in the late phase I of labor that do not have an amplitude > 50 beats	**Antepartally:** sporadic occurrence outside of severe forms of deceleration **Intrapartally:** variable deceleration - decrease of less than 60/min. lasting < 60s; transient short-term bradycardia (< 100 beats/min. lasting 3 minutes, < 80 beats/min. lasting 2 minutes)	**Antepartum:** periodically occurring, severe forms of decelerations **Intrapartum:** severe recurrent early decelerations (with amplitude > 50 beats); severe variable decelerations; late deceleration
Acceleration	≥ 2 within 20 min.	absent for ≥ 40 min.	

🔎 *For more information see Cardiotocography.*

Fetal Biophysical Profile (BPP)

BPP is an examination method based on the evaluation of a non-stress test and ultrasound parameters of the state of the fetus. Less time-consuming is the modified BPP, which combines a non-stress test and ultrasonographic determination of amniotic fluid volume. BPP includes assessment of CTG, amount of amniotic fluid (determining the diameter of the deepest "pocket" of amniotic fluid; the norm is ≥ 2 cm and < 8 cm), respiratory movements, fetal movements and tone.[1]

Doppler methods

- Doppler examination of fetal vessels: arteria cerebri media (dg. fetal anemia), arteria umbilicalis (dg. uteroplacental insufficiency, FGR), ductus venosus (dg. FGR), vena cava inferior.[1]

Computer analysis of fetal electrocardiogram (ECG) - ST analysis

Special software analyzes the fetal ECG.

🔎 *For more information see Fetal ECG.*

Sampling of blood from the fetal scalp (Sahling's method)

It is not currently used in the Czech Republic.

Fetal pulse oximetry

Examination of the partial pressure of fetal blood oxygen using a probe placed on the face of the fetus during delivery. Normal values of pO2 in the I. period of labor: 59 ± 10%, in the II. period: 53 ± 10%.[1]

🔎 *For more information see Fetal Pulse Oximetry.*

References

Related Articles

- Intrapartum fetal monitoring: Cardiotocography • Fetal pulse oximetry • Fetal ECG
- Neonatal hypoxia • Hypoxic-ischemic encephalopathy • Neonatal cardiopulmonary resuscitation

References

1. STRAŇÁK, Z. *Neonatology.* 2. edition. 2015. pp. 17-27. ISBN 978-80-204-3861-4.