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Review

Understanding fetal physiology and second line monitoring during labor

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ABSTRACT

Cardiotocography (CTG) is a technique used to monitor intrapartum fetal condition and is one of the most common obstetric procedures. Second line methods of fetal monitoring have been developed in an attempt to reduce unnecessary interventions due to continuous cardiotocography and to better identify fetuses at risk of intrapartum asphyxia. The acid-base balance of the fetus is evaluated by fetal blood scalp samples, the modification of the myocardial oxygenation by the fetal ECG ST-segment analysis (STAN) and the autonomic nervous system by the power spectral analysis of the fetal heart variability. To correctly interpret the features observed on CTG traces or second line methods, it seems important to understand normal physiology during labor and the compensatory mechanisms of the fetus in case of hypoxemia. Therefore, the aim of this review is first to describe fetal physiology during labor and then to explain the modification of the second line monitoring during labor.

Introduction

Cardiotocography (CTG) is a technique used to monitor intrapartum fetal condition and is one of the most common obstetric procedures [1]. It provides a continuous simultaneous record of the fetal heart rate (FHR) and uterine activity. Classifications have been proposed for the FHR pattern and to predict neonatal outcome [2,3]. In the case of non-reassuring FHR, "second line" methods can be used. In clinical practice, fetal scalp blood samples (FBS) or fetal ECG ST-segment analysis (STAN®, Neoventa Medical, Molndal, Sweden) are commonly performed to improve the prediction of neonatal outcome [4–7]. Recent studies have evaluated the power spectral analysis of heart rate variability, which reflects the autonomic nervous system activity [8–11], and other techniques have been proposed, such as pulse oximetry and near-infrared spectroscopy [12–14].

The correct interpretation of the features observed on CTG traces or second line methods requires an understanding of the normal physiology during labor and the compensatory mechanisms of the fetus in cases of hypoxemia. Therefore, the aim of this review is first to describe fetal physiology during labor and then to explain the second line monitoring modifications during labor.

Methods

A literature search was conducted in March 2016 using the Medline and Cochrane Library computer databases. This research focused on articles published on the subject, and many articles were experimental studies conducted on fetal sheep, which is an animal model that closely resembles the human fetus. The following keywords (MeSH) were selected and combined: hypoxia; physiology; fetal; sheep; chemoreceptor; baroreceptor; hemodynamic; cardiotocograph; ST analysis; and power spectrum. Only publications written in English or French were selected. The computer search was supplemented with a manual search of the references of selected articles.

Comment

The autonomic nervous system, chemoreceptors, and baroreflex

The fetal heart rate depends on its innervation by the autonomic nervous system and involves chemoreceptors and

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baroreceptors: the sympathetic system increases heart rate and the parasympathetic system slows it [15–17].

Peripheral chemoreceptors, located at the aortic arch and at the carotid, are sensitive to modifications in the partial pressure of oxygen (O_2), while central chemoreceptors, located in the brainstem, are sensitive to modifications in the partial pressure of carbon dioxide (PCO_2) and of blood pH [18]. These chemoreceptors act on the peripheral vessels: a decrease in PO_2 causes vasoconstriction, whereas an increase in PO_2 increases induces a vasodilatation [18,19].

Baroreceptors are located in the sinus carotid and the aortic arch and respond to variations in arterial pressure and the pulsatility of the flow in the sinus. Efferent fibers stimulate the vasomotor area through the glossopharyngeal nerve (nerve IX), which then provokes a parasympathetic response through the vagus nerve (nerve X), inducing bradycardia and arterial vasodilatation [15].

Fetal physiology during labor

Knowledge of the mechanisms of regulation has largely been derived from studies on experimental animal models, in particular the fetal sheep [20–24].

Response to hypoxemia

The aim of the adaptation of cardiovascular mechanisms during oxygen deficit is to maintain the correct blood flow and optimal oxygenation, and thus an adapted cell metabolism in vital organs, especially the central nervous system and the heart.

Chemoreflex, catecholamines, and metabolic acidosis. The occurrence of hypoxia stimulates chemoreceptors and induces peripheral vasoconstriction and increased blood pressure, which in turn stimulate baroreflex and result in a slower fetal frequency heart due to parasympathetic responses [20,22,25,26] (Fig. 1). The peripheral vasoconstriction is enhanced by a direct action of hypoxemia on smooth muscle cells in the vascular walls and by the stimulation of the adrenal medulla due to sympathetic stimulation [18,27,28]. This results in the production of catecholamines, which then enhances the vasoconstrictor effect [17,26,29]. These effects counterbalance the negative chronotropic effect induced by parasympathetic stimulation of baroreceptors; thus, they also impact the balance of the autonomic nervous system [30].

The persistence of an O_2 deficit activates an anaerobic cellular metabolism in peripheral tissues, which maintains ATP production and leads to the production of lactic acid, which in turn results in metabolic acidosis [31]. This acidosis then stimulates chemoreceptors that activate the cerebral vasomotor area via the sinocarotid nerve.

Acid-base balance of the fetus. Before labor, the normal arterial pH of the fetus is close to 7.35, and the normal fetal PaO_2 is between 20 and 30 mmHg [32]. During labor, the physiological pH decreases, and two types of acidosis can occur [33]:

- respiratory acidosis arises due to the accumulation of CO_2. It is responsible for the production of H^+ ions due to a movement to the right of the equation: $CO_2 + H_2O \leftrightarrow H^+ + HCO^-_3$. Its diagnosis is based on an elevation of PCO_2 in the umbilical artery blood ($\geq$ 75 mmHg);
- metabolic acidosis is related to a shift to anaerobic pathways during prolonged hypoxia. Anaerobic glycolysis converts glucose to pyruvate, and then to lactate and H^+ ions, thereby decreasing the pH. Its diagnosis is based on the elevation in

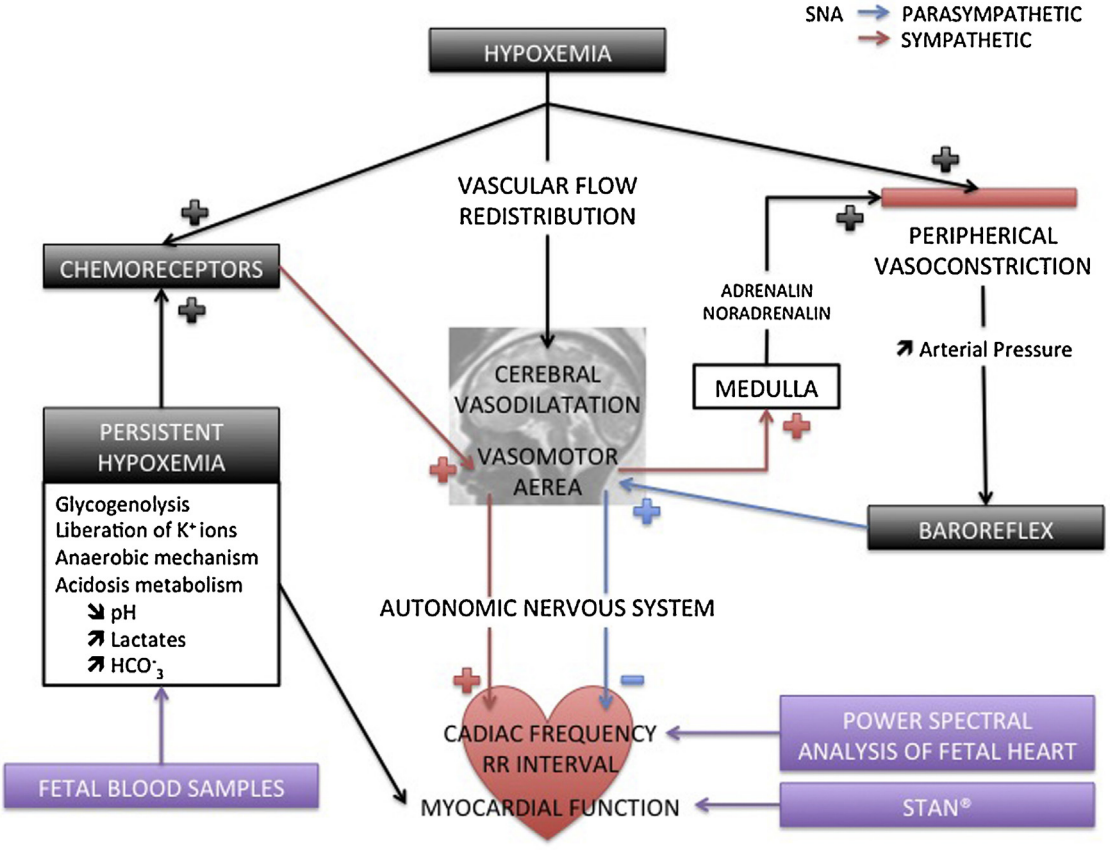

Fig. 1. Fetal response to hypoxemia.

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