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ST-analysis

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Abstract. Analysis of the ST-interval of the fetal electrocardiogram is an adjunct to cardiotocography. Fetal heart rate monitoring should be considered a screening tool, STAN allows more definitive diagnosis of fetal distress when abnormalities on the CTG are detected. Basic pathophysiologic research has demonstrated the rationale behind ST-segment and T/QRS changes, in two randomised controlled trials a lower number of operative deliveries for fetal distress has been demonstrated in CTG+ST versus CTG alone and also a reduction in the number of neonates with hypoxic encephalopathy has been shown.

Keywords: Fetal monitoring; Asphyxia; Fetal distress; STAN; Fetal electrocardiography

1. Introduction

Registration and interpretation of electrocardiographic signals from the fetal heart has been attempted even before the introduction of cardiotocography. The earliest recorded fetal electrocardiogram (FECG) dates back from the 1920s. In the midst of the 20th century Belgian and Dutch researchers made major advances in this field. At first Smyth (1953) and Sureau (1956) used intrauterine electrodes with intact membranes, in 1958 fetal scalp electrodes were introduced by Kaplan and only in 1960 Hon used the technology to produce the first cardiotocographic traces. The original aim of FECG was to have proof of fetal life, shortly thereafter followed by attempts to diagnose fetal distress based on morphological changes in the trace. Already in 1957 Southern recognised S–T segment negativation and changes in the T-wave to be important markers of fetal distress. The basic technology of autocorrelation of the FECG to improve the quality of the weak signals was described by Van Bemmel in 1968 and a Ph.D. thesis on the subject written at Nijmegen

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University by L.A.M. Peeters [1]. Automatic computer-aided analysis became available only during the 1980s, resulting in the development of the STAN®S-21 device (Neoventa Medical, Gothenburg, Sweden).

2. Basic pathophysiology [2]

The aim of fetal monitoring during labour is to identify the fetus at risk of an adverse event (mainly asphyxia) and to allow timely intervention. Therefore we need to understand the causes of fetal distress and the mechanisms by which the fetus reacts to stress. Experimental animal research in the 1970s (mainly by the Swedish Göthenburg group of K.G. Rosen et al. and in the UK at Oxford in Dawes' group) further explored repolarisation changes, reflected in the ST-interval in fetal hypoxia. It was shown that the increase in T-wave amplitude during hypoxia was correlated with myocardial glycogenolysis and that plasma potassium concentration was associated with lactic acid production. The same pattern of FECG changes as seen in hypoxia can be induced by beta-adrenoceptor activation. Further experiments on chronically instrumented fetal lambs made it clear that ST-waveform elevation (expressed as a T/QRS ratio) was strongly correlated to a rise of lactate and could be used to identify anaerobic myocardial metabolism. As there is a strong correlation between the T/QRS ratio and the level of circulating adrenaline-mediated, it is suggested that the rise in T/QRS ratio in case of an anaerobic myocardial metabolism is mediated by adrenaline beta-adrenergic stimulation. Simultaneous registration of somatosensory evoked electro-encephalography and FECG indicate that the mature lamb fetus ST-waveform changes are associated with intact fetal cortical activity. Fetal maturity was shown to have an influence on FECG changes. In mature lamb fetuses catecholamine concentrations rise more significantly in case of hypoxia as compared with immature fetuses; the lesser the rise in catecholamines, the lesser the T/QRS ratio. Not only gestational age but also the growth status of the fetus influences FECG changes in relation to hypoxia. When comparing normal-sized and growth-retarded guinea-pig fetuses, growth-retarded fetuses presented a different reaction pattern with a biphasic or negative ST-segment instead of a rise in T/QRS ratio. In case of a negative ST-waveform plasma catecholamine levels are lower and it is supposed the myocardial glycogen reserves are depleted and/or the sympathoadrenal response is blunted. The mechanism underlying biphasic/negative ST-segment changes are not yet fully elucidated. Negative ST-segments appear in significant cardiac dysfunction with hypotension and are noted in preterm fetuses and/or in case of fetal infection, probably related to lipopolysaccharide endotoxins.

3. Clinical human studies

Instead of using the usual precordial leads a more longitudinal unipolar lead is used by using an electrode under the skin of the presenting part and a second electrode on the maternal thigh, this allows good sensitivity for the Y-vector changes in the fetal ECG and is not affected by rotation during the passage through the birth canal. Single spiral electrodes are most suitable but sometimes present a low-amplitude QRS complex. This is possibly caused by changes in the molecular structure of the needle electrode. Work is in