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ELECTROCARDIOGRAPHIC CHANGES DURING CAESAREAN SECTION UNDER REGIONAL ANAESTHESIA

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Introduction

Electrocardiographic (ECG) changes which are characteristic of myocardial ischaemia have been shown to occur during caesarean section under regional anaesthesia. Palmer et al showed that over 37% of their patients developed such changes and suggested that the aetiology may be an imbalance of myocardial oxygen supply and demand at delivery. The aims of this study were to determine the incidence of the ECG changes and to establish whether they were associated with echocardiographic evidence of myocardial ischaemia.

Methods

Twenty five healthy mothers undergoing elective caesarean section under regional anaesthesia were studied. Prior to the institution of regional anaesthesia each patient was fitted with an Oxford Medilog 111 two channel ECG recorder. Spinal or epidural anaesthesia was then instituted and a block induced to a level of at least T6 bilaterally. Precordial two-dimensional echocardiography was carried out on 13 patients using a midpapillary short-axis view with repeated basal and apical sweeps. Imaging was carried out prior to surgery, restarted immediately after delivery and continued until the patient was haemodynamically stable and symptom free and there was normalisation of any ECG abnormality. Significant ST segment depression was deemed to be \( \geq 0.1 \) mv at 0.08 s after the J point. Echocardiograms were reported by a cardiologist who was unaware of the ECG details. Normal systolic wall motion was deemed to have occurred if a radius from the centre of the left ventricle shortened by 30% with uniform wall thickening.

Results

Sixteen out of the 25 patients had ST segment depression which was highly suggestive of myocardial ischaemia. Of the 13 patients who underwent echocardiography 8 had significant ST segment depression but in every case wall motion remained entirely normal during the ECG changes. There were no demographic differences between those with and without ST changes. Heart rates were higher in the group with ST depression with the difference becoming statistically significant at delivery (p<0.05). There was a marked temporal correlation between the sudden increase in heart rate at delivery and acute ST change.

Conclusion

We conclude that ST depression is a common feature of the ECG during caesarean section under regional anaesthesia and occurs without underlying myocardial ischaemia. The aetiology of the ST depression is unclear but it may, at least in part, be a rate-related phenomenon.

Reference

CONTINUOUS AMBULATORY ECG MONITORING FOR DETECTION OF PERIOPERATIVE ISCHAEMIA DURING CAESAREAN SECTION

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A high incidence of electrocardiographic changes occurring during caesarean delivery under regional anaesthesia has been reported recently. This study found that 35 of 93 patients had ECG changes characteristic of myocardial ischaemia. Fifteen patients complained of symptoms of chest pain, pressure or dyspnoea, associated in every case with electrocardiographic changes.

However this study did not include preoperative ECG assessment, monitoring of peroperative ECG changes was intermittent and no control group of patients receiving general anaesthesia was included in the study. We therefore decided to investigate the incidence of ST segment abnormalities occurring during caesarean delivery under regional and general anaesthesia, using a computerised ambulatory ECG surveillance system (Compas, Cardiac Care Units Inc.).

Methods

Twenty-seven patients scheduled for elective caesarean section under epidural, spinal and general anaesthesia were investigated.

Continuous ambulatory ECG monitoring was used to record ECG abnormalities during the perioperative period. ST segment changes of 1 mm or greater for a minimum of 1 min were considered significant.

Results and conclusion

Evidence of myocardial ischaemia was not detected in any patients undergoing caesarean section under regional or general anaesthesia.

Reference


MONITORING THE FETUS WITH A PULSE OXIMETER

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Pulse oximetry has changed the way anaesthetists and neonatologists monitor their patients. It provides a continuous non-invasive method of measuring oxygen saturation. We have refined the technology and used it to monitor the fetus in labour. In a prospective observational study we correlated fetal oximetry readings with outcome.

Methods

We studied 96 women in normal labour. They had no known risk factors and at the onset of labour the cardiotocograph was normal. We excluded women who were less than 37 weeks gestation, women with a low-lying placenta or known genital infection. We used a newly designed fetal oximetry sensor and a pulse oximeter specifically adapted for fetal use. We used a specially designed software programme to calculate the average saturation in each case. The pulse oximetry data from each case were examined retrospectively.

Results

Ninety-one of the 96 babies had normal CTGs and were born in good condition. The average pulse oximeter oxygen saturation was 65% (SD 10%) during labour in these cases. Of the 96 ‘normal’ labours, 5 babies were delivered because of an abnormal CTG. Two of these were delivered in good condition (Apgars of 9 at 1 min and 10 at 5 min in both cases). The average oxygen saturations, as recorded by the pulse oximeter, were 65% and 63% respectively. Two babies were born in poor condition with Apgars of 6 and 9 and 5 and 10 at 1 min and 5 min. The average oxygen saturations were 46% and 43% respectively. The fifth baby was born in poor condition but monitoring with the pulse oximeter was discontinued before any abnormality became apparent.

Discussion

In this series, the oxygen saturation recorded by the pulse oximeter only dropped significantly in babies born in poor condition. This suggests that pulse oximetry is more specific and sensitive than cardiotocography. Pulse oximetry could be used in association with the CTG to increase specificity and to distinguish the truly hypoxic trace from other ‘abnormal’ traces. It also has the potential to become an alternative to fetal scalp sampling. This could lead to a decrease in both caesarean section rates and hypoxia related fetal injuries.
CAN IN VITRO TECHNIQUES ELUCIDATE THE MECHANISM OF LABOUR?

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The factors controlling human labour remain elusive, due in part to our ignorance of the physiological mechanisms of uterine contraction. The underlying mechanism is important since its determination would allow a rational approach to the pharmacological manipulation of uterine activity. Within the myometrial cell, intracellular calcium ([Ca\(^{2+}\)]\(_i\)) is of fundamental importance in the initiation of contraction. In addition to controlling actin and myosin interaction it is the final mediator of numerous intracellular second messengers.

Invasive physiological studies are inappropriate during human labour. We have developed an approach using isolated myometrial cells. These can retain the characteristics of the parent tissue. Cell culture has been combined with fluorescent indicator techniques in order to determine [Ca\(^{2+}\)]\(_i\) in single human myometrial cells.

Myometrial biopsies were obtained, with informed consent, following hysterectomy or at caesarean section. Cells were separated by enzymatic digestion and cultured in 5% CO\(_2\) and 95% air. Once confluent, cells were plated onto glass coverslips and studied after a further 3–4 days culture. Calcium was measured by loading cells with a calcium-sensitive fluorescent indicator dye (Fura-2) and the change in fluorescence ratio determined in single cells by microspectrofluorimetry. The extracellular environment was manipulated as required.

Application of oxytocin (OT) or prostaglandin E\(_2\) (PGE\(_2\)) caused a transient increase in [Ca\(^{2+}\)]\(_i\). In 4 of the 200 cells studied, spontaneous transient increases in [Ca\(^{2+}\)]\(_i\) occurred. This may represent spontaneous activity which occurs in the pregnant or non-pregnant uterus. Removal of extracellular calcium ([Ca\(^{2+}\)]\(_o\)) prevents calcium influx. In calcium-free medium, OT caused a small increase in [Ca\(^{2+}\)]\(_i\) but the response to PGE\(_2\) was completely prevented. This suggests that these agents have a different mechanism of action and intracellular stores of calcium may be released by OT but not PGE\(_2\). Spontaneous transient increases in [Ca\(^{2+}\)]\(_i\) are prevented by removal of [Ca\(^{2+}\)]\(_o\) or application of 2mM Ni\(^{2+}\) (a calcium channel blocker). The OT antagonist (CAP 476) reduced the frequency of the transients.

We conclude that physiological techniques can be used to determine the mechanism of action of agents which influence myometrial contractility. OT and PGE\(_2\) increase [Ca\(^{2+}\)]\(_i\), but only OT releases intracellular stored calcium. Spontaneous increases in [Ca\(^{2+}\)]\(_i\) occur in some cells. These are caused by the influx of extracellular calcium and may be modified by the OT antagonist CAP 476.

Acknowledgement

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Reference