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Obstetric

(Abstracts and Case Report/Series)

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A Novel Approach to Optimize Programmed Intermittent Epidural Bolus (PIEB) Delivery for Labour Analgesia

Authors:

Munro, Allana¹; George, Ronald B.²; Andreou, Pantelis³

¹Department of Women's and Obstetric Anesthesia, IWK Health, Dalhousie University, Halifax, NS, Canada

²Department of Anesthesia and Perioperative Care, UCSF, San Francisco, CA, USA

³Department of Community Health & Epidemiology, Dalhousie University, Halifax, Nova Scotia, Canada

Introduction:

How to effectively deliver Programmed Intermittent Epidural Bolus (PIEB) for the maintenance of labour analgesia is still being investigated. When programming the PIEB pump for labour analgesia, three key parameters need to be programmed: the Next bolus (PIEBn), the bolus interval (PIEBi) and the bolus volume (PIEBv). There is limited evidence available to guide the dose or interval for pump settings trying to optimize the quality of analgesia and patient satisfaction. We hypothesized a mathematical modelling tool, Response Surface Methodology (RSM)¹, could evaluate multiple PIEB pump settings to optimize clinically important patient outcomes simultaneously. The objective of this study was to use RSM to best estimate PIEB settings (PIEBn, PIEBi, PIEBv) to optimize the primary outcome measures: maternal satisfaction score, need for a clinician administered rescue bolus, and the ratio of Patient Controlled Epidural Analgesia (PCEA) boluses (PCEA delivered/PCEA requested).

Methods:

With institutional ethics approval, a double-blind randomized trial was completed in the labour and delivery unit of a tertiary care centre. Nulliparous, English-speaking ASA Status II women aged 18-45 years, at full term, with single gestation in vertex presentation and in spontaneous labour, between 2-6 cm cervical dilatation were eligible for inclusion. Women with significant comorbidities, contraindication to neuraxial analgesia, using chronic analgesic medications, less < 152 cm or BMI > 45 kg/m² were excluded. After informed consent, labour analgesia was initiated via the epidural catheter using 10 ml ropivacaine 0.2% with 10 ug/ml fentanyl solution. PCEA was standardized for all patients. After two unsuccessful PCEA attempts the anesthesiologist could provide a manual rescue bolus or a clinician bolus via the CADD-Solis[®] Epidural Pump. Women were randomly assigned, by computer generation, to predetermined PIEB settings (Figure 1A & 1B). Clinical outcomes were gathered within 24 hours of delivery from the epidural pump, and anesthesia and nursing electronic medical records. RSM identified coordinates for the three pump settings that represented a stationary point optimized for the three outcomes: PCEA ratio (a ratio closest to 0.1), rescue clinician bolus (optimal is zero) and maternal satisfaction (Visual Analog Scale, 0-100, optimal response is ≥ 90).

Results:

Modelling calculations estimated a sample of 70 participants would suffice to estimate the optimal response for each outcome. Of 287 potential participants, 191 did not meet inclusion criteria or declined to participate and 26 were withdrawn, leaving 70 women for study inclusion. Using RSM, the suggested PIEB settings when identifying a stationary point while trying to simultaneously optimize all the primary study outcomes were: PIEBn = 29.4 minutes, PIEBi = 59.8 minutes, and PIEBv = 6.2 mL (Figure 1C). These values were associated with the optimized outcomes of Maternal Satisfaction 94%, PCEA ratio 0.78, and Need for Rescue Bolus 0.28. Only 5 patients met criteria for hypotension and were all randomized to different groups. The dermatome sensory

score was between T10 and T5 in 89% of the women. The median lowest Bromage score was 4.

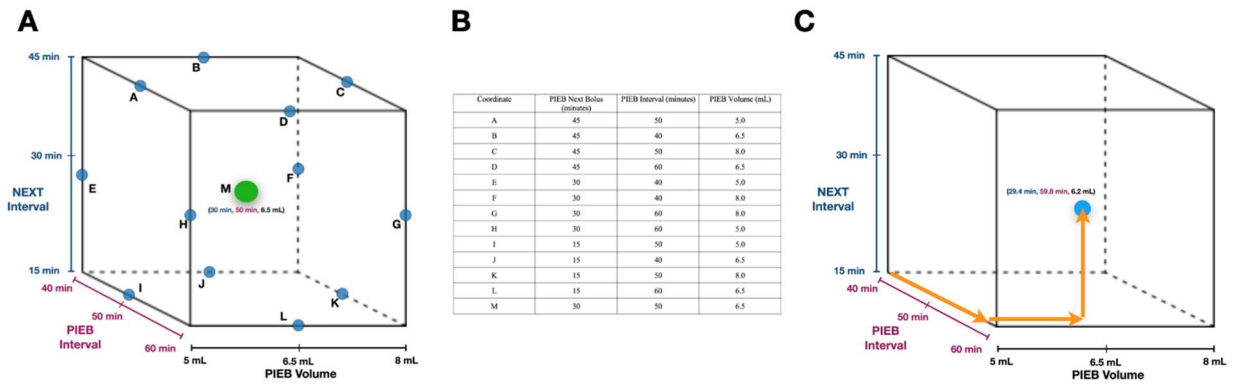
Discussion:

This is the first study to use a mathematical model to optimize three PIEB pump settings simultaneously. Our study recommends a PIEBi of approximately 60 minutes which matches results presented in the literature.^{2,3} The suggested PIEBv in our study is slightly lower than previously reported,² however, this study considered patient satisfaction, an outcome not previously examined in PIEB studies. This study has used all clinical outcomes weighted equally in terms of importance, which may not represent patient priorities for labour analgesia. Future studies could use RSM methodology to target alternate obstetric populations with comorbid disease and multiparity.

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Figure 1



Congenital Central Hypoventilation Syndrome and Pregnancy – A Case Report

Authors:

Yu, Janice¹; Araujo Monteiro da Silva, Rodrigo¹; Adly, Elaheh¹; Cordovani, Daniel¹

¹Department of Anesthesia, McMaster University, Hamilton, Canada

Introduction:

Congenital Central Hypoventilation Syndrome (CCHS) is a rare condition caused by genetic mutations in the paired-like homeobox 2B (PHOX2B) gene.¹ There are only approximately 1000 cases worldwide.² This mutation will affect the autonomic central control of ventilation resulting in alveolar hypoventilation.¹ Additionally, vasovagal syncope and cardiac arrhythmias such as sinus pauses can be present.¹ There is currently no cure for CCHS. However, in recent years, patients with CCHS may undergo diaphragm pacing (DP) by phrenic nerve stimulation to assist with their ventilation.³ This device stimulates the phrenic nerves, causing the diaphragm to contract, thereby assisting with ventilation.³ There is limited data related to the management of CCHS during pregnancy and just as few reports of anesthetic management of phrenic nerve stimulators.^{2,4} Herein we describe a parturient with CCHS and bilateral phrenic nerve stimulators who safely underwent an urgent caesarean section under epidural anesthesia.

Case Presentation:

A 34-year-old G3A1L1 parturient at 39+2 weeks gestational age had labour induced due to maternal and fetal CCHS. She breathes spontaneously during the daytime, and typically activates DP during sleep. During the third trimester of pregnancy, DP caused discomfort due to her enlarged abdomen; therefore, she switched to BiPAP ventilation overnight. Other significant medical history included vasovagal syncope and symptomatic sinus pauses, typically triggered by pain.

Induction of labour happened at the L&D step-down unit. Monitors included 5-lead ECG, radial arterial line, pulse oximetry, and continuous cardiotocography. An epidural catheter was placed at L3/L4 level without complications prior to active labour to avoid painful stimulation. She maintained a sensory block to T8 throughout labour with bupivacaine 0.08% with fentanyl 2mcg/ml infusing between 2 - 12 ml/hr.

Due to atypical fetal heart rate and suspected placental abruption, an urgent caesarean section was indicated. Neonatology team was present for delivery. Her epidural was topped up with 3 incremental boluses of 5ml of Lidocaine 2% with Epinephrine 1:200,000 and Fentanyl 100mcg to achieve a sensory block to T4. She remained in sinus rhythm with mean arterial blood pressure maintained within 20% of baseline with intermittent IV administration of Phenylephrine 100mcg as needed. No respiratory compromise was noted. After delivery, Epidural Morphine at a reduced dose of 2mg was administered. She was transferred back to step-down in stable condition for further telemetry and oximetry monitoring and resumed her BiPAP ventilation that evening without adverse events. She was discharged from hospital three days later.

Conclusion:

The anesthetic care for parturients with CCHS and phrenic nerve pacemakers is challenging. During pregnancy, diaphragm pacing is difficult due to the enlarged abdomen. Early epidural placement could be considered to circumvent active labour pain during labour induction, which may trigger cardiac arrhythmias. Also, either the avoidance or dose reduction of drugs that further depress the respiratory drive should be considered. There is

no literature addressing the effect of electrocautery on phrenic nerve pacemakers. Ultimately, multi-disciplinary planning is crucial in determining the optimal timing, mode of delivery, and anesthetic technique, all while ensuring neonatal care immediately upon delivery.

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General Anesthesia for Cesarean Delivery in Critically Ill Parturients with COVID-19: A Case Series

Authors:

Bradley Kaminski MD¹, William Turner BMBS FRCA¹, Misha Virdee MD¹, Michael Szpejda MD¹, Wendy L. Whittle MD PhD², Mrinalini Balki MBBS, MD^{1, 2, 3, 4}

¹*Department of Anesthesiology and Pain Medicine, Mount Sinai Hospital, University of Toronto, Toronto*

²*Department of Obstetrics and Gynaecology, Mount Sinai Hospital, University of Toronto, Toronto*

³*Department of Physiology, University of Toronto, Toronto*

⁴*Samuel Lunenfeld Research Institute, Toronto*

Introduction:

COVID-19 ARDS has been associated with an increased likelihood of surgical intervention in the peripartum patient, with approximately 72-84% of cases being managed with cesarean delivery (CD).^{1,2} Further, these cases are more likely to be conducted under general anesthesia due to maternal respiratory failure.^{3,4} Despite an increasing global caseload of critically ill parturients with COVID-19, there is a general lack of information regarding the anesthetic management and maternal outcome following CD in such cases. Accordingly, our purpose is to describe the anesthetic technique and postoperative outcomes of three critically ill parturients with COVID-19 and provide the evidence base for our clinical decisions.

Case Presentation:

Three pregnant patients presented to our hospital with severe COVID-19 ARDS in their 3rd trimester of pregnancy. Although currently, COVID-19 is not an indication for delivery prior to 34-weeks gestation due to neonatal morbidity, an SpO₂ cut-off of <93% indicates that mechanical ventilation may be necessary. Severe hypoxemia and precipitous clinical decline served as the basis for managing our cases under general anesthesia. Oxygenation and ventilation did not improve immediately following delivery in any patient, obviating the need for post-operative transfer to ICU. Requirements for hemodynamic support varied between patients. Postpartum hemorrhage was encountered in one case, requiring a combination of oxytocin, ergometrine, hemabate, tranexamic acid, and red blood cell transfusion. Worsening respiratory status observed in this patient was likely multifactorial - a combination of ARDS, ventilator-associated pneumonia, and fluid redistribution. Prone positioning, iNO and ECMO were considered and utilized as temporary measures to improve oxygenation.

Conclusion:

While COVID-19 infection alone is not an indication for delivery, concomitant obstetric indications and clinical deterioration may necessitate emergency intervention. Our case series is novel as it describes the management of emergency CD under general anesthesia in parturients with COVID-19 ARDS, which has not been reported in the literature. The critically ill parturient with COVID-19 presents a unique challenge for optimal anesthetic management, combining considerations for ARDS, multi-system critical illness, and concurrent pregnancy. The potential for respiratory and hemodynamic decline should be anticipated, particularly where post-partum hemorrhage is observed, and post-operative ICU admission is necessary.

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Table 1: Overview of patient characteristics including COVID-19/ ARDS management and intra-operative management.

Demographics	Comorbidities	COVID Species	Medical Management	Anesthetic Management/blood loss	Neonatal outcome	Timeline/ Complications
Patient 1: Age: 26 years G1 P0 Gestational age at delivery: 36 ⁶ weeks	Preeclampsia	SARS-CoV-2 N501Y S Gene Mutation (Beta variant - B.1.351)	Dexamethasone 6 mg IV x10 days; Tocilizumab 8 mg/kg IV x1 dose; Enoxaparin 40 mg sc	GA maintenance: MAC (Sevoflurane)= 0.4, Propofol 100 mcg/kg/min Ventilation: VCV, FiO ₂ 1.0 Hemodynamic Support: Labetolol 10 mg EBL = 800 ml PPH Management: Oxytocin 3U IV, Oxytocin (20U/L) 125ml/h infusion	Weight: 2.61 kg APGAR scores: 1, 6 and 8 at 1, 5, and 10 minutes, respectively	Symptom day at CD: 12 ICU LOS: 5 days (4 days intubated) Total LOS: 8 days
Patient 2: Age: 33 years G1 P0 Gestational age at delivery: 36 ⁴ weeks	Morbid obesity (BMI 62 kg/m ²)	SARS-CoV-2 N501Y S Gene Mutation (Beta variant - B.1.351)	Dexamethasone 6 mg IV x10 days; Tocilizumab 8 mg/kg IV x1 dose, Ceftriaxone 1 g; Enoxaparin 40 mg sc	GA maintenance: MAC (Sevoflurane)= 0.8-1.0 Ventilation: PCV switched to manual ventilation, FiO ₂ 1.0 Hemodynamic Support: Ephedrine 10 mg IV, Epinephrine 20 µg IV, Norepinephrine 6 µg IV x 9 doses EBL = 800 ml PPH Management: Ergometrine 250 µg IM x 2 doses, Oxytocin (20U/L) 125ml/h IV infusion, pRBC x1 unit	Weight: 2.58 kg APGAR scores: 5, 9 and 9 at 1, 5, and 10 minutes, respectively	Symptom day at CD: 4 ICU LOS: 9 days (8 days intubated) Total LOS: 16 days Complications: cardiomyopathy, AKI
Patient 3: Age: 31 years G3 P2 Gestational age at delivery: 28 ⁶ weeks	N/A	SARS-CoV-2 N501Y S Gene Mutation (Beta variant - B.1.351) & E484K Gene Mutation (B1.1.7 variant)	Dexamethasone 6 mg IV x 2 days - switched to Methylprednisolone 40 mg IV x 8 days; Tocilizumab 8 mg/kg IV x1 dose; Ceftriaxone 1 g; Piperacillin-Tazobactam; Meropenem; Caspofungin; Heparin 5000 IU sc	GA maintenance: Propofol 60 µg/kg/min, Fentanyl 250 µg/hr, Midazolam 10 mg/hr Ventilation: VCV, FiO ₂ 1.0 Hemodynamic Support: Norepinephrine (0.02 µg/kg/min) IV infusion, Vasopressin (0.04 IU/min) IV infusion EBL = 1500 ml PPH Management: Oxytocin infusion 5U IV bolus x4, Oxytocin (40U/L) 125 ml/hr infusion, Ergometrine 250 µg IM x2 doses, Hemabate 250 µg IMM, Tranexamic acid 1g IV x 2 doses, pRBC x3 units.	Weight: 1.49 kg born apneic with flaccid tone. APGAR scores: 2, 5 and 8 at 1, 5, and 10 minutes, respectively, with resuscitation	Symptom day at CD: 28 ICU LOS: >30 days Total LOS: >30 days Complications: VAP, AKI

G = gravidity, P = parity, BMI = body mass index, GA = general anesthesia, sc = subcutaneous, EBL = estimated blood loss, PPH = post-partum haemorrhage, VCV = volume control ventilation, PCV = pressure control ventilation, pRBC = packed red blood cells, IMM = intramyometrial, CD = cesarean delivery, LOS = length of stay, VAP = ventilator associated pneumonia, AKI = acute kidney injury

In Vitro Optimization of Oxytocin-Induced Myometrial Contractility by Propranolol - Potential Applications in Induction of Labor and Treatment of Postpartum Hemorrhage

Authors:

Balki, Mrinalini¹; Caliaperumal, Jayalakshmi¹; Miller, Lauren¹; Wang, Stella²; Huszti, Ella²; Kingdom, John³

¹ Department of Anesthesia and Pain Management, Mount Sinai Hospital, University of Toronto, Toronto, Canada

² Biostatistics Research Unit, University Health Network, Toronto, Canada

³ Department of Obstetrics and Gynaecology, Mount Sinai Hospital, University of Toronto, Toronto, Canada

Introduction:

The rates of cesarean deliveries (CD) and postpartum hemorrhage (PPH) due to failed labor continue to rise worldwide. Oxytocin (OT) is used to induce and augment labor; it is also used to reduce the risk of PPH.[1] However, continual exposure to OT during labor can result in OT receptor desensitization and downregulation, further resulting in poor responsiveness in many women.[2] It is proposed that propranolol, when administered with OT, can shorten the period between induction and active labor, with improvements in uterine contractility. However, clinical studies are heterogeneous with conflicting results [3,4]. This study aims to determine: 1) the effect of propranolol on myometrial contractions induced by low-dose (LD) OT by simulating labor induction (LI) and 2) the effect of propranolol on contractions induced by high-dose (HD) OT in OT-desensitized myometrium for prevention of PPH.

Methods:

This *in-vitro* study was conducted utilizing myometrial samples from women undergoing CDs. Each dissected myometrial strip was suspended in organ bath chamber in physiological salt solution under homeostatic conditions. The strips were subjected to any one of the groups (Gr) in LI or PPH models (fig1). The LI model consisted of 3 Grs (A [control], B, C) with each undergoing either no pretreatment (pretx) (Gr A and Gr C) or propranolol (10^{-6} M) pretx (Gr B) for 1h prior to dose response (DR) to LD OT from 10^{-13} M to 10^{-9} M. The PPH model consisted of 4 Grs (D [control], E, F, G), each undergoing a pretx with OT (10^{-5} M) for 2h to induce desensitization with addition of propranolol (10^{-6} M) in Grs E and G. Each Gr underwent DR with HD OT from 10^{-9} M to 10^{-5} M, with addition of propranolol (10^{-6} M) during DR in Gr F and G. Contraction amplitude (g) and frequency (contractions/10 min) were recorded using an isometric force transducer. Primary outcome was motility index (MI; Amp x Freq). A multivariable linear regression model, applying generalized estimated equation to adjust for the type of drug and patient group, was used to analyze the data during DR relative to baseline.

Results:

Sixty-two women were recruited, and 225 successful experiments ($n=32/Gr$) were conducted. In the LI model, Gr B showed a significantly higher MI of contractions than Gr A (estimated mean difference [EMD] 72.98%, $p=0.015$), while MI between Gr C and Gr A was not significantly different (EMD 29.05 %, $p=0.27$). The MI in the PPH model did not show any significant group differences (EMD: Gr D and E, 5.76%, $p=0.803$; Gr D and Gr F, -10.95%, $p=0.605$; Gr D and Gr G, 4.98%, $p=0.844$).

Discussion:

In vitro propranolol pretreatment improved OT induced contractility in the LI model, however, it did not affect contractions in the PPH model. This implies that propranolol administration during early labor can improve OT induced contractility, thus reducing the risk of failed labor and facilitating vaginal delivery. Further studies are required to establish clinical correlation of these findings.

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Figure 1:

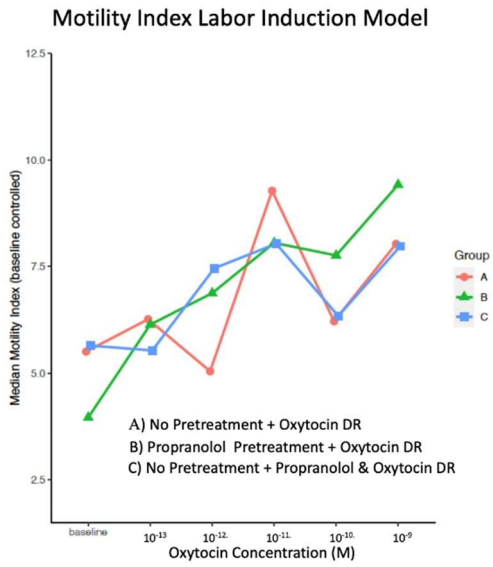


Figure 1 A. Dose-response (DR) curves for motility index (g*contractions/10 min) relative to baseline after oxytocin exposure. Myometrial strips underwent no pretreatment or pretreatment with oxytocin 10⁻⁵M for 1hour.

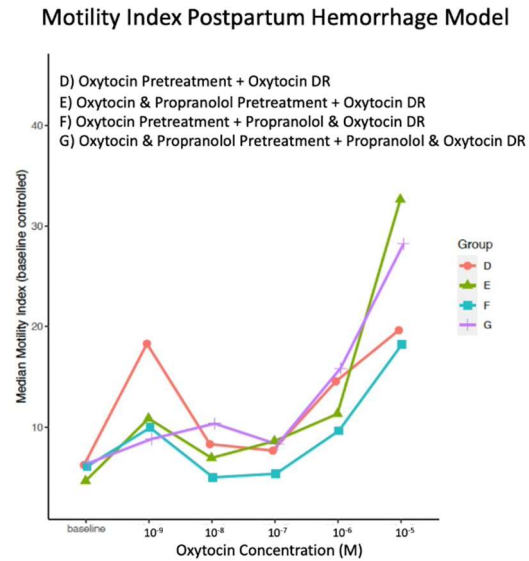


Figure 1 B. Dose-response curves for motility index (g*contractions/10 min) relative to baseline after oxytocin exposure. Myometrial strips were pretreated with oxytocin 10⁻⁵M or oxytocin with propranolol 10⁻⁶M for 2 hours.

Paroxysmal Supraventricular Tachycardia During Pregnancy – Cardiac Ablation During the Second Trimester, a Case Report

Authors:

Araujo Monteiro da Silva, Rodrigo¹; Cordovani, Daniel¹; Gibson, Kara¹

¹ Department of Anesthesia, McMaster University, Hamilton, Canada

Introduction:

It is well known that pregnancy can exacerbate pre-existing arrhythmias or be the period of the first manifestation. The causes are possibly related to the increase of catecholamines during pregnancy, increased adrenergic receptor sensitivity, and increased maternal circulating volume.¹ The risk factors are congenital and structural heart disease. The most common arrhythmia in women of reproductive age is Paroxysmal Supraventricular Tachycardia (SVT). Atrioventricular Nodal Re-entry and Wolf-Parkinson-White are the two main causes of SVT in pregnancy.² There are few studies regarding the management and urgent treatment of SVT in pregnant women. Several recommendations from the American College of Cardiology, the American Heart Association Task Force Guidelines, and the European Society of Cardiology are mostly based on expert consensus.³ Herein we describe a pregnant patient with SVT who safely underwent radiofrequency ablation under sedation.

Case Presentation:

A 25-year-old primipara at 25 weeks gestational age presented to hospital for SVT ablation. She had a pre-pregnancy history of SVT described as sudden onset of palpitations but no chest pain. Symptoms worsened as pregnancy progressed. Medical therapy with trials of sotalol and flecainide had failed. Echocardiogram from 3 months prior confirmed SVT but ruled out structural heart disease. Other co-morbidities included mild thrombocytopenia and anxiety. On clinical examination she was hemodynamically stable with a blood pressure of 111/64 mmHg, but pulse rate was 190 bpm. Immediate pre-procedural fetal nonstress test was reassuring except for mild fetal tachycardia. Continuous oxygen saturation, capnography, electrocardiogram, and invasive blood pressure monitoring were used. The need to access the groin impeded left tilt positioning. Supplemental oxygen mask 6 L/min, midazolam 1mg, fentanyl 30mcg, and lidocaine 50mg were administered. A continuous infusion of propofol was maintained throughout the case between 30 – 50 mcg/Kg/min. Other drugs used in her care included heparin, protamine, and isoproterenol. The patient remained hemodynamically stable during the whole procedure. After successful ablation the patient was transferred in stable condition and in regular sinus rhythm to the recovery area for continued monitoring. Intermittent fetal heart rate by doppler was normal during this period. She was discharged from hospital four hours later with no complications.

Conclusion:

The management of SVT in pregnancy remains a clinical challenge. Echocardiography is essential to exclude structural and functional heart diseases. Adenosine and electric cardioversion are safe during pregnancy. Elective SVT ablation brings its own considerations: ideal timing, radiation, positioning, and exposure to anesthesia and cardiac medications. Teratogenesis has not been associated with the use of any commonly used agents, including opioids, ketamine, and benzodiazepines.⁴ Opioids decrease fetal heart rate variability, but this is not concerning in the absence of maternal hypotension. Good communication and planning between obstetrics, interventional cardiology, anesthesia, nursing, and the patient can lead to a successful outcome.

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Spinal Anesthetic with Bupivacaine for an Urgent Caesarean Section in a Parturient with Brugada Syndrome: A Case Report

Authors:

Yu, Janice¹; Nishi, Cameron¹; Adly, Elaheh¹; Cordovani, Daniel¹

¹Department of Anesthesia, McMaster University, Hamilton, Canada

Introduction:

Brugada syndrome, a hereditary cardiac rhythm dysfunction that can lead to lethal tachyarrhythmias, has been described for only two decades.¹ Pregnancy, physical or mental distress, and many drugs can trigger lethal arrhythmias.² Given the significant potential for sudden cardiac death, particular attention should be paid to labour and perioperative management of parturients. Many drugs used in neuraxial or general anesthesia can theoretically be life-threatening; for example, bupivacaine is considered as a medication to be avoided, but literature has not yet demonstrated that bupivacaine leads to morbidity or mortality in parturients under spinal anesthetic specifically.^{2,3} Herein, we describe a parturient with Brugada syndrome who safely underwent a spinal anesthetic with bupivacaine for an urgent caesarean section. Growing numbers of similar reports indicate that some trigger drugs might actually be safe for short-term use in obstetrical patients with Brugada syndrome.

Case Presentation:

A 23-year-old G2A1 woman at 35+4 days gestational age presented to hospital with decreased fetal movement. An urgent caesarean section was indicated due to atypical fetal heart rate. The patient had Brugada syndrome with an ICD in-situ, along with anxiety and gastroesophageal reflux disease.

Vital signs and blood work were within normal limits. She was already betamethasone-optimized earlier in pregnancy. Preoperative cefazolin was given, but the routine metoclopramide was withheld. Anesthetic monitoring included 5-lead ECG, radial arterial line, non-invasive blood pressure cuff, pulse oximetry, and external defibrillator pads. Two large-bore intravenous cannulas were placed, and a magnet was placed on her ICD. Isoproterenol, noradrenaline, adrenaline, and a crash cart were available in the operating room. After 2 ml of 2% plain lidocaine for skin freezing, her spinal was performed with a 25 G Pencan needle using 1.6 ml 0.75% hyperbaric bupivacaine, 15 mcg fentanyl, and 100 mcg preservative-free morphine. The patient tolerated the procedure well and developed a block to T4. Transient hypotension was treated with either 10 mcg noradrenaline or 10 mg ephedrine boluses to maintain mean arterial pressure within 20% of baseline.

Intraoperatively, bipolar cautery malfunction necessitated use of monopolar cautery in short bursts, to minimize ICD interference. After delivery, she received 100 mcg of carbetocin intravenously. There were no intraoperative adverse events, and she was transferred to the ICU in stable condition for 24 hours of ECG monitoring. Her postoperative course was uncomplicated with no arrhythmias, and she was discharged from hospital 2 days later.

Conclusion:

Brugada syndrome in obstetrics brings many challenges. Commonly used uterotonics, anti-emetics, and anesthetic agents such as propofol, ketamine, and bupivacaine may all pose risks.^{2,3} Avoidance of bupivacaine has been recommended due to prolonged binding to sodium channels.² However, multiple case reports have demonstrated safe use of bupivacaine in spinal anesthesia.^{4,5} Our experience adds to this body of evidence.

A multidisciplinary approach involving anesthesia, obstetrics, cardiology, and nursing is paramount to ensuring a safe delivery. Close follow-up for exacerbations of Brugada during pregnancy, and early discussion between the patient and anesthesiologist ensures the patient understands potential anesthetic concerns around their delivery.

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Spontaneous and Oxytocin-Induced Contractility after Exposure to Intravenous Anesthetic Agents: An In-Vitro Study in Human Myometrium

Authors:

Portela, Natalia C¹; Drew, Thomas¹; Carvalho, Jose C A¹; Engel, Tess¹; Balki, Mrinalini¹

¹Department of Anesthesiology and Pain Medicine, Mount Sinai Hospital, University of Toronto, Canada

Introduction:

General anesthesia is associated with higher blood loss than neuraxial anesthesia after cesarean delivery (CD) [1] and the effects of anesthesia drugs on uterine tone have been pointed as contributors for bleeding [2]. The objective of this study was to investigate the dose-response profiles of anesthesia induction agents (with and without oxytocin) on gravid human myometrium.

Methods:

In this in vitro study, myometrial samples were obtained from patients undergoing elective CD. Individual strips were mounted in organ bath chambers filled with physiological salt solution (PSS) to mimic physiologic conditions. Samples were allocated to one of the 7 groups (Grs): 1) Propofol (P), 2) Etomidate (E), 3) Ketamine (K), 4) Propofol+Oxytocin (PO), 5) Etomidate+Oxytocin (EO), 6) Ketamine+Oxytocin (KO), and 7) Oxytocin (O) (Control Gr). After equilibration, each strip was subjected to dose-response (DR) testing with the study drug in a pattern of 0.5 log molar increase from 10^{-7} M to 10^{-4} M; however, PO, EO and KO Grs received additional oxytocin 20 nM during DR. Amplitude (g) and frequency (contractions (c) per 10 min) were recorded. The primary outcome was motility index (MI; amp x freq; g*c/10 min). All outcomes were expressed as a % change from baseline during equilibration. Generalized linear regression models with log link function were used to compare the groups. Generalized estimating equation was implemented in all models to account for repeated measures from the same patient. All tests were 2-tailed; P < 0.05 was considered statistically significant.

Results:

We analyzed 189 experiments on samples from 35 patients. MI decreased progressively with increasing concentration of study drug in all Grs. MI during spontaneous contractions with each study drug was significantly lower when compared to control Gr O (Fig1). When compared with Gr O, the estimated mean diff (EMD) (95% CI) in Gr P -71.3% (-90.50% to -13.3%) P=0.027, Gr E -86.7% (-3.9% to -71.0%) P<0.0001 and Gr K was -82.6% (-90.2% to -69.0%) P<0.0001. Amongst oxytocin treated groups, PO demonstrated significantly lower MI (-69.8% (-85.2% to -38.4%) P=0.001), while EO and KO were not different from Gr O. Grs EO (308.6% (76.2% to 847.3%) P=0.001) and KO (374.3% (152.9% to 789.8%) P<0.0001) produced higher MI than E and K, respectively, however, Grs PO and P were not significantly different.

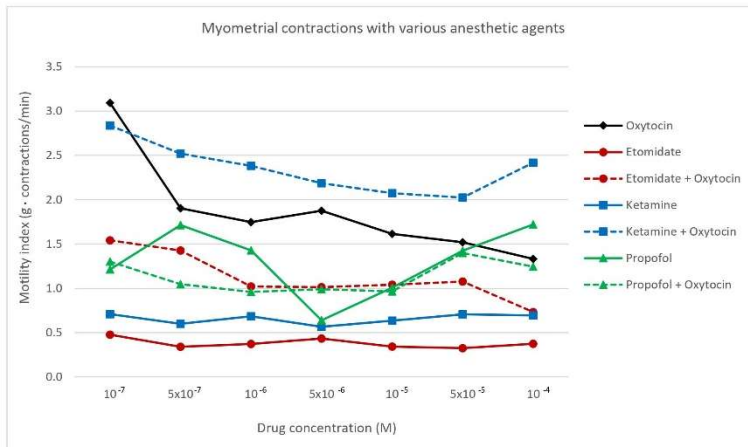
Discussion:

All the studied intravenous induction agents cause dose dependent decrease in uterine contractility. Oxytocin helps augment contractility produced by etomidate and ketamine, but not propofol. We suggest the use of etomidate or ketamine rather than propofol in the postpartum period along with oxytocin to produce superior uterine tone. This finding warrants further clinical studies to explore uterine responsiveness to oxytocin in women with oxytocin-augmented labor requiring general anesthesia for CD.

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Figure 1:



The Incidence and Predictors of Failed Spinal Anaesthesia in Caesarean Delivery

Authors:

Munro, Allana, BSc Pharm, MD, FRCPC,¹ Jin, Sherry, MD, FRCPC,² McKeen, Dolores, MD, FRCPC, MSc, CCPE,³ Aidemouni, Milia, MSc,¹ Uppal, Vishal MBBS, FRCA¹

¹ IWK Health, Dalhousie University, Halifax, Canada

² Humber River Hospital, Toronto, Canada

³ Memorial University of Newfoundland, St. John's, Canada

Introduction:

The incidence of failed spinal anaesthesia for caesarean deliveries according to previous studies ranges from 2.7 to 9.1%.^{1,2} Although many risk factors are implicated to be associated with spinal failure, their relative predictive value is unknown. Our primary aim was to identify predictors of failed spinals in the obstetric population and to quantify their relative importance in a predictive model for failure. The secondary aim of this study was to determine the incidence of failed spinal anaesthesia for caesarean deliveries at a tertiary care obstetric hospital. The primary study outcome was the failure of spinal anaesthesia, defined as the need to provide an alternative anaesthetic, such as a repeat spinal, a new epidural, or convert to GA, within one hour of the initial spinal. The secondary outcome was the need to provide supplemental analgesia or sedative medication within one hour of the initial spinal.

Methods:

With institutional Research Ethics Board approval and a waiver for the need for written informed consent, we conducted a retrospective review of the electronic anaesthetic records (Innovian Anaesthesia®) for all caesarean deliveries performed at a tertiary care hospital from September 28, 2010 to September 30, 2019. Of approximately 10,000 anaesthetic records, 5799 cases were performed under either spinal anaesthesia or combined spinal-epidural (CSE). We excluded caesarean deliveries performed under an epidural or general anaesthesia (GA). The anaesthetic records of all patients with failed spinals were manually reviewed to confirm inclusion criteria was satisfied. Anaesthetic, medical, surgical, and demographic information were extracted from the Innovian database. Variables that were not collected by Innovian but were potentially important to the success of spinal anaesthesia (e.g., weight) were captured using data linkage with a provincial perinatal database. Binary logistic regression with performed with the spinal failure as an outcome variable. Multivariable analysis assessed the association of predictors, chosen a priori, with failed spinals. The effect size was measured using odds ratios. A p value of <0.05 was considered statistically significant. Data analysis was performed using SPSS 24. Dominance analysis (RStudio) assessed the importance of each predictor on the primary outcome.

Results:

The incidence of failed spinal anaesthesia requiring an alternative anaesthetic was 2.0%, with conversion to GA occurring in 0.7% of surgeries. Supplemental analgesia or sedation was provided to an additional 3.2% of women. Predictors for a failed spinal, identified using multivariate regression, is presented in Table 1. The most important predictors for a failed spinal were previous caesarean delivery (OR, 11.39; 95% CI, 7.15-18.26; P < 0.001), tubal ligation (OR, 7.51; 95% CI, 2.87-17.27; P < 0.001), lower BMI (OR, 0.94; 95% CI, 0.90-0.97; P = .001), and surgery duration (OR, 1.02; 95% CI, 1.00-1.03; P = .022). Dominance analysis identified the most important predictors for failed spinal anaesthesia requiring an alternate anaesthetic were previous caesarean

delivery, tubal ligation, and BMI, which accounted for 9.6%, 1.4%, and 1.2% of the variance in outcome, respectively.

Discussion:

Spinal anaesthesia alone failed to provide a pain-free surgery in 5% of our caesarean deliveries with a 2% failure rate and 0.69% requiring conversion to GA within one hour of the spinal. The most important predictors were a history of previous caesarean delivery, tubal ligation, lower BMI, and surgery duration. Future studies may investigate the reasons for these associations and techniques to prevent failures.

References:

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Table 1 Multivariate logistic regression on predictors of failed spinal anaesthesia.

Predictors	Alternate anaesthetic vs. no failure			Alternate anaesthetic, supplemental analgesia/sedation vs. no failure		
	OR	CI	p value	OR	CI	p value
(Intercept)	0.01	0.00 – 45.06	0.289	0.67	0.00 – 114.86	0.883
Gestational age	0.92	0.85 – 1.00	0.048	0.88	0.84 – 0.93	<0.001
Parity (Para 0:0; Para 1,2,3:1)	2.17	1.27 – 3.88	0.006	1.39	1.01 – 1.94	0.051
Previous caesarean delivery	11.39	7.15 – 18.26	<0.001	3.94	2.83 – 5.45	<0.001
Body Mass Index	0.94	0.90 – 0.97	0.001	0.99	0.97 – 1.01	0.203
Psychiatric illness	1.51	0.73 – 2.84	0.232	2.08	1.43 – 2.98	<0.001
Tubal ligation	7.51	2.87 – 17.27	<0.001	3.05	1.49 – 5.77	0.001
Surgery duration	1.02	1.00 – 1.03	0.022	1.03	1.02 – 1.03	<0.001
Type of Incision	0.92	0.13 – 3.78	0.918	1.30	0.52 – 2.98	0.554
Urgent or emergent surgery	1.70	1.00 – 2.82	0.046	1.19	0.85 – 1.66	0.306
Vertebral level (L2/3 and L3/4:0; L4/5:1)	2.23	1.14 – 4.07	0.013	1.31	0.81 – 2.04	0.246
Intrathecal bupivacaine dose	1.40	0.71 – 2.80	0.363	1.06	0.71 – 1.61	0.781

OR: odds ratio, CI: confidence interval