Bupivacaine-fentanyl epidural analgesia for a parturient in status asthmaticus

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Regional anaesthesia is a suitable technique for the management of the asthmatic parturient. We report the case of an asthmatic gravida in labour in whom prompt institution of bupivacaine-fentanyl epidural analgesia was associated with enhancement of the effectiveness of concurrent medical therapy for bronchospasm. Prior to the initiation of epidural blockade, inhaled atropine was employed in an effort to reduce parasympathetic tone in the bronchial smooth muscle. Sustained clinical improvement did not occur until after delivery of the fetus and placenta.

The reported incidence of asthma in pregnancy is approximately 1.0 per cent; these same reports reveal an incidence of status asthmaticus approaching 0.2 per cent.^{1,2} The majority of asthmatics appear to tolerate pregnancy and parturition with minimal exacerbation of their disease. However, it should be recognized that those patients who have a history of status asthmaticus when not pregnant, as well as those asthmatics whose symptoms have worsened during earlier pregnancies, represent a category of patients with a remarkably high incidence of maternal and fetal morbidity and mortality.¹⁻⁴ Indeed, termination of pregnancy by emergency Caesarean section has been performed as a life-saving measure in moribund asthmatic mothers.^{5,6}

Regional anaesthesia has been described as the technique of choice for the management of the asthmatic parturient.^{7,8} We report the case of an asthmatic gravida in labour in whom prompt institution of bupivacaine-

fentanyl epidural analgesia enhanced the effectiveness of concurrent medical therapy for bronchospasm. Before the initiation of epidural blockade, inhalation therapy with aerosolized atropine was employed in an effort to reduce parasympathetic tone in the bronchial smooth muscle. Sustained clinical improvement did not occur until after delivery of the fetus and placenta.

Case report

A 34-year-old gravida 3, para 2, in her 35th week of pregnancy was admitted to the labour suite following the onset of regular uterine contractions and the spontaneous rupture of the membranes. She was diaphoretic, had laboured respirations with audible wheezes and a cough productive of green, tenacious sputum. She was very agitated and would not lie supine.

Her past medical history revealed episodes of coughing and wheezing which regularly occurred during her menstrual periods and which abated without therapy. During her first two pregnancies she had been free of respiratory complaints. Early in the current pregnancy she had developed a persistent cough, wheezing and dyspnoea which had necessitated seven admissions for medical management because her symptoms could not be controlled on outpatient therapy. Her chronic medications were oral aminophylline, oral terbutaline and inhaled metoprolol. During her eighth week of pregnancy, she had required a rapidly tapered course of oral corticosteroid therapy. Her theophylline levels (random) were consistently within the therapeutic range. During symptom-free periods, her forced vital capacity, one-minute forced expiratory volume and mid-expiratory flow rates were normal. She had no history of atopy.

Her vital signs at the time of admission were: axillary temperature 36.9°C, heart rate 130 beats·min⁻¹, respiratory rate 30 breaths·min⁻¹ and laboured, and blood pressure 150/74 mmHg. Auscultation of the chest revealed inspiratory and expiratory wheezes to be present diffusely over both lung fields. The remainder of the physical examination was unremarkable. Chest radiography revealed a right middle lobe infiltrate. Arterial blood gas analysis (FiO₂ 0.21) showed: PO₂ 77 mmHg, PCO₂

Key words

ANAESTHESIA: obstetric; ANAESTHETIC TECHNIQUES, REGIONAL: epidural, epidural narcotics; LUNG: asthma; ANALGESICS: fentanyl.

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32 mmHg, pH 7.41 and haemoglobin saturation 95 per cent. The serum electrolytes and haematocrit were normal for this stage of pregnancy. A serum theophylline determination was reported as $9.5 \text{ mg}\cdot\text{dl}^{-1}$. The white blood cell count revealed $14,800 \text{ cells}\cdot\text{mm}^{-3}$ and no eosinophils were noted on peripheral blood smear. The fetal heart rate was $146 \text{ beats}\cdot\text{min}^{-1}$.

At this time, $6 \text{ L}\cdot\text{min}^{-1}$ of humidified supplemental O_2 were administered by non-rebreathing face mask. Analysis of arterial blood gases at this time revealed: PO_2 243 mmHg, PCO_2 35 mmHg, pH 7.41 and haemoglobin saturation 99 per cent. A Gram's stain of sputum revealed Gram-positive intracellular diplococci. The following drugs were then administered: terbutaline 0.25 mg SC, aminophylline 250 mg slowly IV, metoprolol 0.3 ml in 3.0 ml sterile saline by jet nebulizer and piperacillin 4.0 gm slowly IV. At this time, an internal fetal monitor, an ECG and a left radial arterial line were placed. The fetal heart rate was $170 \text{ beats}\cdot\text{min}^{-1}$.

Ninety minutes later, the patient complained that she was too tired to breathe. The fetal heart rate decreased to $100 \text{ beats}\cdot\text{min}^{-1}$. Analysis of arterial blood gas, with $10 \text{ L}\cdot\text{min}^{-1}$ humidified O_2 delivered by a non-rebreathing mask showed: PO_2 78 mmHg, PCO_2 36 mmHg, pH 7.34 and haemoglobin saturation 95 per cent. Metoprolol 0.3 ml in 3.0 ml sterile saline was administered by jet nebulizer and hydrocortisone hemisuccinate 1.0 g was given slowly IV. Preparations for emergency intubation and delivery of the fetus by Caesarean section were made. However, the patient reported a slight improvement in her shortness of breath. A second arterial blood gas analysis showed: PO_2 140 mmHg, PCO_2 30 mmHg, pH 7.41 and haemoglobin saturation 99 per cent. The fetal heart rate had increased to $140 \text{ beats}\cdot\text{min}^{-1}$.

Three hours later, the patient became diaphoretic, developed audible wheezes and complained of severe shortness of breath. Atropine 2.0 mg in 3 ml sterile saline was administered by jet nebulizer. Analysis of arterial blood gases following atropine administration and with the patient breathing 50 per cent humidified O_2 delivered by a Venturi mask revealed: PO_2 104 mmHg, PCO_2 30 mmHg, pH 7.42 and haemoglobin saturation 99 per cent. Following administration of the atropine, the maternal heart rate did not increase and the fetal heart rate ranged between 140 – $150 \text{ beats}\cdot\text{min}^{-1}$. The patient was noted to be sleeping comfortably on her left side.

Two hours later, the decision was made to induce labour with pitocin. At this time, the patient was diaphoretic and complained of mild shortness of breath. Arterial blood gas analysis (50 per cent humidified O_2 delivered by Venturi mask) revealed: PO_2 91 mmHg, PCO_2 27 mmHg, pH 7.49 and haemoglobin saturation 96 per cent. It was thought to be beneficial for the patient to

institute epidural analgesia for labour, prior to initiation of pitocin augmentation. Therefore, following a "preload" of 1000 ml of crystalloid solution, analgesia to the tenth thoracic dermatome was obtained utilizing bupivacaine 0.5 per cent, 3.0 ml with $15 \mu\text{g}$ epinephrine as a test dose followed by a solution of bupivacaine 0.125 per cent 10.0 ml with fentanyl $5 \mu\text{g}\cdot\text{ml}^{-1}$ injected in divided doses through the epidural catheter. Maternal vital signs following institution of epidural blockade were: blood pressure 110/50 mmHg, heart rate $100 \text{ beats}\cdot\text{min}^{-1}$ and respiratory rate $20 \text{ breaths}\cdot\text{min}^{-1}$ and unlaboured. Fetal heart rate ranged between 130 – $140 \text{ beats}\cdot\text{min}^{-1}$.

The patient slept quietly throughout an uneventful three-hour labour, requiring no further injections of the bupivacaine-fentanyl mixture for analgesia. At the end of labour, she delivered spontaneously a male infant with Apgar scores of nine at one and five minutes after birth. One hour after delivery, the patient had no wheezes on examination of the chest and analysis of arterial blood gases (with supplemental humidified O_2 $3 \text{ L}\cdot\text{min}^{-1}$ delivered by nasal cannulae) revealed: PO_2 179 mmHg, PCO_2 22 mmHg, pH 7.47 and haemoglobin saturation 99 per cent.

Discussion

Several reviews outline the pathophysiology and pharmacotherapy of acute and chronic asthma.^{7,9–11} From these studies, guidelines emerge which aid the obstetric anaesthetist in managing the parturient in status asthmaticus.

The maternal arterial carbon dioxide tension during the third trimester of pregnancy ranges between 28 mmHg–31 mmHg.^{12,13} The normal maternal alkalaemia of pregnancy and a painful labour easily may be worsened by the hyperventilation present in the early stages of an acute asthmatic attack. If the arterial oxygen tension of a pregnant asthmatic is reduced in this alkalotic milieu, it is logical to assume that fetal oxygenation will be rapidly and severely compromised.^{14,15} Therefore, it is essential to avoid maternal arterial hypoxaemia through the use of supplemental humidified oxygen therapy.^{16–19} Adequate hydration, treatment of any concurrent maternal pulmonary infection and chest physiotherapy for mobilization of secretions are necessary secondary measures designed to maintain or enhance oxygen exchange.

A decreasing maternal arterial PO_2 in the presence of a maternal arterial $\text{PCO}_2 > 35 \text{ mmHg}$ and a pH < 7.35 indicates respiratory failure in the pregnant asthmatic.² At this point, intubation and assisted mechanical ventilation should be considered, while every effort is made to optimize medical therapy. Because the insertion of an endotracheal tube into an unanaesthetized trachea is a potent stimulus for bronchospasm, intubation itself should

be considered a temporizing measure until the status asthmaticus is controlled through other means. Had the patient described above not responded to the medical regimen developed for her, immediate orotracheal intubation and expeditious delivery of the fetus and placenta were planned.

The addition of inhaled atropine to a regimen of metoprolol, corticosteroids and aminophylline was suggested by evidence which indicates its efficacy in those nonpregnant asthmatics in whom bronchial hyperactivity to cholinergic stimuli elicits bronchospasm.²⁰⁻²⁴ Unfortunately, there are limited published data concerning the effects of inhaled anticholinergics on the course of labour or on the fetoplacental unit. Hernandez *et al.*⁵ describe a case in which inhaled atropine was successfully used to alleviate the symptoms of a pregnant asthmatic; no specific mention is made of adverse side effects. Its use in our patient, who was under continuous monitoring with an electrocardiogram, intra-arterial line and external tocodynamometer, appeared to result in subjective symptomatic improvement without associated maternal or fetal tachycardia or signs of fetal distress.

In our patient, the judicious institution of epidural analgesia prior to pitocin augmentation of labour appeared to provide an additional beneficial effect on the resolution of her status asthmaticus. Effective pain relief will allay maternal anxiety, which of itself may provoke an attack of asthma.³ Such relief may also reduce the tremendous involuntary increase in maternal minute ventilation which occurs during a painful labour.¹¹ Uncontrolled hyperventilation will worsen the respiratory alkalosis usually present in the early stages of status asthmaticus with resultant detrimental effects on fetal oxygenation. In the patient presented above, epidural analgesia was carefully instituted before labour was induced with pitocin. Fentanyl was added to the local anaesthetic mixture in an effort to increase the maternal analgesia and to minimize the total dose of bupivacaine.²⁵⁻²⁷

In a patient such as this, it is essential to avoid a respiratory arrest resulting from a high epidural blockade or from a total spinal block following unintentional dural puncture. Therefore, close attention should be given to the technique of induction of epidural analgesia, raising the level of blockade slowly and utilizing divided doses of anaesthetic injected after careful tests of aspiration. The depressant effects of narcotics on the central ventilatory response to carbon dioxide must also be kept in mind. The lowest possible concentration of epidural fentanyl should be employed and naloxone should be available for immediate administration if signs of narcotic-induced respiratory depression become apparent.

In summary, status asthmaticus in the parturient carries a high risk of maternal and fetal morbidity and mortality.

Production of fetal asphyxia through maternal arterial hypoxaemia and respiratory alkalosis must be avoided, utilizing if necessary controlled mechanical ventilation. The addition of aerosolized atropine to a medical regimen consisting of methylxanthines, corticosteroids and inhaled beta-sympathomimetic agents may decrease cholinergic tone in the maternal bronchial smooth muscle and appears to have little effect on the fetus. Properly instituted epidural analgesia may also alleviate bronchospasm brought on by the maternal response to a painful labour. In the absence of intravenous or subarachnoid injection, fentanyl added to the epidural local anaesthetic mixture appears to produce profound analgesia with minimal effect on maternal respiratory effort or fetal well-being.

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Résumé

L'anesthésie régionale est une technique convenable pour traiter une parturiente asthmatique. Nous rapportons le cas d'une asthmatique en travail chez qui l'administration rapide d'une analgésie péridurale au bupivacaine-fentanyl a été associée à une amélioration de l'efficacité d'une thérapie médicale concourante pour bronchospasme. Avant de débiter le blocage péridural, on a employé de l'atropine par inhalation dans une tentative de réduire le tonus parasympathique des muscles lisses bronchiques. Une amélioration clinique soutenue ne s'est produite qu'après la sortie du fœtus et du placenta.