Declarations of interest

None.

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Syncope after administration of epidural analgesia in an obstetric patient with a vagus nerve stimulator

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ABSTRACT

Vagus nerve stimulation (VNS) is an adjunctive therapy for medically refractory epilepsy and depression. Vagus nerve stimulation is generally well-tolerated, but cardiac arrhythmias or asystole are rare complications that have been reported. This case report describes an obstetric patient who received epidural analgesia and subsequently experienced two episodes of syncope synchronous with stimulation from her VNS device. These resolved after deactivating the device. This is the first report of a suspected arrhythmia during VNS in the setting of epidural analgesia.

Keywords: Vagus nerve stimulation; Epidural analgesia; Syncope

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Introduction

Vagus nerve stimulation (VNS) is a treatment modality for medically refractory epilepsy and depression. A generator is surgically implanted into a subcutaneous pouch in the chest wall and connected to a tunneled electrode that attaches around one vagus nerve in the carotid sheath.[1] The device is programmed to stimulate at a certain time interval and current output, titrated to maximize efficacy while minimizing side effects. Bradycardia or other arrhythmias are rare complications.[2] This report describes an obstetric patient with a VNS who experienced syncope during stimulation events while receiving labor analgesia via continuous epidural infusion of local anesthetic.

Case report

A 34-year-old female, G1P0 at 32 + 5 weeks of gestation presented with decreased fetal movements and was diagnosed with intrauterine fetal demise. Fetal echocardiogram had previously demonstrated a two-vessel umbilical cord and a left superior vena cava, and a diagnosis of polyhydramnios had been made at 32 weeks-of-gestation with an amniotic fluid index of 26 cm.

Her medical history included severe, medically refractory depression treated with bupropion and a left-sided vagal nerve stimulator, which had been placed 10 years previously (Cyberonics, Model # 104, Houston, TX). She was otherwise healthy and had received general anesthesia without complication for the VNS placement, and for a generator battery change one year prior to presentation. The most recent device check had been two months prior to presentation.

She was admitted to the labor floor for induction of labor and requested epidural analgesia. Admission blood pressure was 128/77 mmHg and complete blood count, coagulation studies, liver enzymes, creatinine level, and urine proteins were all unremarkable.

After obtaining informed consent, an epidural catheter was placed in the sitting position via a 17-gauge Tuohy needle, using a loss-of-resistance to saline technique at the L3-4 level. Aspiration of the epidural catheter was negative for blood or cerebrospinal fluid, and the patient was asymptomatic after a test dose of 3 mL of lidocaine 1.5% with epinephrine 1:200,000. Patient-controlled epidural analgesia (PCEA) was started with an initial bolus of 15 mL of 0.0625% bupivacaine with 2 μg/mL fentanyl, followed by an infusion at 10 mL/h with a demand bolus of 5 mL and a 10-min lock-out. Nursing staff checked her blood pressures at two minute intervals for the first 20 minutes after epidural placement, and no abnormalities were noted.

Approximately 40 minutes after the initial dose, while the obstetrician was placing an intra-cervical Foley bulb for cervical ripening, the patient experienced a transient loss of consciousness. She regained full consciousness without any disorientation within a few seconds. She described tinnitus immediately prior to the loss of consciousness, but denied circumoral numbness or metallic taste; her blood pressure was unchanged from baseline. The epidural infusion was paused. On examination, the patient had a bilateral T10 sensory level of anesthesia, no motor block, and there was no nystagmus. The presumed diagnosis was vasovagal syncope related to Foley bulb placement. However, five minutes later, the patient again experienced brief loss of consciousness, this time observed by the anesthesia team and with no obvious causative event. As she regained consciousness, she realized that the syncopal episode immediately followed the onset of electrical stimulation from her VNS, which she senses by a change in her voice, and a "tickling pressure sensation." Ephedrine 10 mg was administered, continuous electrocardiography and pulse oximetry monitoring were initiated, and the next vagal nerve stimulus occurred without event. The patient and medical team agreed to disable the VNS using her device magnet. The epidural infusion was restarted, and labor and delivery proceeded uneventfully with no further episodes of syncope. Fetal autopsy confirmed the earlier echocardiogram findings, maternal anti-cardiolipin and lupus anticoagulant antibody screens were negative and no definitive diagnosis to explain the fetal death was made.

Discussion

Vagus nerve stimulation was initially approved for the management of epilepsy in the United States of America (USA) in 1997, after which it was observed that patients reported better mood, independent of the degree of seizure control.[3] This led to pilot studies using VNS for the treatment of resistant depression, and it was approved for this indication in 2005. The mechanism of action has yet to be elucidated. The VNS device consists of a pulse generator/stimulator, placed in a subcutaneous pouch in the chest wall, which connects to an electrode wrapped around the left vagus nerve in the carotid sheath via a single subcutaneous lead. The VNS generator can be programmed and interrogated via an externally placed programming wand. A typical setting is 30 second pulses of electrical current (1–2 mA at 20–30 Hz) every five minutes.[4]

The most commonly reported adverse effects include hoarseness, cough, dyspnea, and neck pain.[3] Side effects tend to be dose-dependent, decrease with time, and are only present during stimulation.[5] Cardiac arrhythmias, including atrio-ventricular (AV) block, bradycardia and asystole, are rare complications. The left vagus nerve is chosen to minimize cardiac complications; the right vagus nerve supplies the sinoatrial node, while the left innervates the AV node.[1] A cohort study of 22 patients found that VNS-induced arrhythmias were

more likely in older patients, and were not dose-dependent, suggesting the mechanism is unlikely due to direct vagal nerve stimulation, but rather a more complex modulation of cardiac conduction.[2]

Our patient had not experienced an episode of symptomatic bradycardia or syncope during the 10 years of VNS therapy. She did note occasional dyspnea during stimulation periods, and she routinely suspended VNS activity during exercise. She was not receiving any medications other than the epidural infusion that might have predisposed her to syncope. She was in the early stages of induction of labor, with no clinical or laboratory evidence to suggest pre-eclampsia, seizure, placental abruption, or sepsis.

During the initial presentation, local anesthetic systemic toxicity was among the differential diagnoses of the syncope. Such toxicity most typically presents with central nervous system signs within a few minutes of local anesthetic injection, however signs can be non-specific and the onset may vary, occasionally presenting substantially later.[6] The local anesthetic infusion was temporarily discontinued and resuscitative supplies, including Intralipid, were kept in close proximity until the clinical picture became more evident.

Normally, arterial baroreceptor control leads to a compensatory increase in heart rate in response to vasodilation. However, during epidural block a reduction in venous return can increase vagal activity, secondary to decreased right atrial filling, leading to reduced signal output from intrinsic chronotropic stretch receptors (a reverse Bainbridge reflex).[7] We suspect that this physiological change, combined with transient vagal nerve stimulation, led to a reversible arrhythmia. We also suspect that a further increase in vagal tone from intra-cervical Foley bulb placement contributed to the first episode of syncope. Temporarily discontinuing the epidural infusion, administering ephedrine as a temporizing measure, and disabling the VNS device may have prevented the occurrence of any further episodes for our patient.

In general, there is no current recommendation to disable a VNS during the peri-operative period, if side effects are absent.[4] Housmans et al. reported one case of bradycardia and asystole in a patient with a VNS during a general anesthetic, years after initial stimulator placement.[8] This group similarly hypothesized that the lack of sympathetic reflexes due to anesthetic agents may have contributed to VNS-induced asystole.

Neuromodulatory devices are gaining in popularity, and will likely be seen with increasing frequency in obstetric patients.[9] To our knowledge, this is the first report of VNS-induced syncope in the context of epidural analgesia or anesthesia. Other case reports and case series have reviewed VNS during pregnancy, and their consensus is that VNS use should be continued throughout pregnancy and delivery, but these publications have not included discussions of anesthetic management.[10–12] There is one report describing the use of a VNS during labor epidural analgesia, without complications.[13] As this current report represents only the second case report in this setting, conclusions about safety cannot be drawn. Our patient was undergoing induction of labor for intrauterine fetal demise, so fetal wellbeing was not a concern. However, in a patient with a living fetus, perturbation of uteroplacental perfusion during an arrhythmia would have to be considered. If VNS is to be continued during epidural analgesia, continuous electrocardiography monitoring should be considered. The manufacturer supplies VNS patients with a bar-shaped 50 gauss magnet with a wristband and belt clip to keep the magnet readily available. Brief application of the magnet over the generator for less than two seconds will lead to delivery of an extra stimulation, which can be used by patients or caregivers to terminate a seizure. Placement of the magnet without removal and taping it in place will reversibly suspend therapy,[14] and it may be advisable to do so if new onset syncope or cardiac arrhythmias occur during administration of epidural analgesia.

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Anaesthetic management for caesarean section of a parturient with a known difficult airway and closed spinal dysraphism

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ABSTRACT
Many anaesthetists consider patients with existing neurological deficits, untreated spinal pathology or those having undergone major spinal intervention to be precluded from undergoing neuraxial anaesthesia. While this is partly rooted in fears of litigation there is also a lack of consensus of the best practice in the anaesthetic management of these patients.
We present our management of a parturient who attended our institution, having a number of anaesthetic complexities including a known difficult airway, spinal fusion and persistent spinal cord tethering. She successfully underwent delivery under neuraxial blockade for the delivery of her fourth child.
We believe that by undergoing a thorough multidisciplinary clinical evaluation, including the extensive use of neuroimaging and ultrasound, it may be possible to plan and perform safe neuraxial anaesthesia.

Keywords: Neuraxial anaesthesia; Scoliosis; Harrington rods; Spinal dysraphism; Spinal cord tethering

Case report

We present the case of a 36-year-old para 3, gravida 2^{+1} woman who presented for the delivery of her fourth child. She had undergone a two-stage scoliosis repair using Harrington rods, inserted from the sixth cervical to the second lumbar vertebrae at the age of 12. She had associated closed spinal dysraphism, requiring multiple surgeries to de-tether her spinal cord at the fifth lumbar and first sacral levels. Neurologically she had persistent unilateral left leg numbness, however bladder, bowel and motor function were subjectively normal.

Obstetrically her first two pregnancies were normal, having delivered spontaneously without neuraxial blockade. Her third pregnancy, however, was complicated by the diagnosis of a heterotopic pregnancy requiring laparoscopy. Intubation was difficult, with two failed attempts using an Airtraq video laryngoscope and ultimately, she was intubated using a McCoy laryngoscope. She was documented as a Cormac and Lehane grade three view.[1] Her remaining viable pregnancy was a delivery by emergency caesarean section at 28 weeks' gestation, due to concerns about an evolving uterine scar dehiscence. This was performed under general anaesthesia following an awake fibreoptic intubation. Unfortunately, due to neonatal prematurity, this child did not survive past 36 hours. Our patient described the whole event, including awake fibreoptic intubation, as "horrible".

Anaesthetic management

This patient was assessed at our high-risk anaesthetic clinic. She had reduced neck extension, poor mouth opening of 2 cm and a Mallampati score of 4.[1] Thyromental distance was 6.5 cm and prognathism was normal. Examination of her back revealed a scar from the C6 to the L2 vertebrae, corresponding to her previous scoliosis repair; and a scar overlying the L5/S1 vertebral levels, from her previous spinal cord

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