Preoperative Confirmation of Epidural Catheter Siting by Epidural Electrical Stimulation (Tsui) Test Precipitates Vasovagal Pre-Syncope in a 61-Year-Old Woman Undergoing Surgical Repair of a Ventral Hernia: A Case Report

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Objective: Challenging differential diagnosis

Background: Thoracic epidural analgesia is a commonly applied technique for perioperative pain management in major thoraco-abdominal surgery, but is complicated by high failure rates. The epidural electrical stimulation test (EEST) or “Tsui test” is a safe and effective method whereby low electrical current is used to confirm correct epidural catheter position and decrease the variability in analgesic effectiveness of the neuraxial technique.

Case Report: We present the case of a 61-year-old woman with no prior cardiac or pulmonary comorbidities who was scheduled to undergo a ventral and parastomal hernia repair with component separation. The patient was offered a low thoracic epidural for perioperative analgesia. Technical aspects of the siting of the epidural catheter were uneventful and a confirmatory Tsui test was planned. At the initiation of electrical stimulation via the epidural catheter to confirm optimal catheter positioning, the patient experienced symptomatic bradycardia and hemodynamic instability that persisted despite terminating the electrical stimulation, and required pharmacologic intervention.

Conclusions: This report describes a rare case of vasovagal pre-syncope associated with the EEST or Tsui test. Although vasovagal reactions can be commonly associated with neuraxial procedures due to augmented venous return or severe emotional stress, we raise the possibility that through direct electrical stimulation in the epidural space, the EEST may have the potential to trigger such a physiologic response.

Keywords: Anesthesia • Anesthesia, Epidural • Perioperative Medicine • Syncope, Vasovagal

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Background

Thoracic epidural analgesia remains a mainstay of perioperative pain management for major thoraco-abdominal surgery. Evidence suggests that effective epidural analgesia has the potential to limit the surgical stress response, reducing cardiovascular, respiratory, and gastrointestinal morbidity. However, reported epidural failure rates as high as 32% highlight the variability in analgesic effectiveness associated with the neuraxial technique [1,2]. Epidural failure has been attributed to variables such as provider experience, incorrect siting of the catheter during insertion, and post-procedural catheter migration [2]. In practice, many clinicians rely on subjective indicators related to the epidural technique to inform the likelihood of success of epidural catheter placement, such as a convincing “loss of resistance” or minimal force required for catheter threading. Confirmation of the catheter’s position can be further validated through the use of a standard epidural test dose (3 mL lidocaine, 1.5%, with epinephrine, 1: 200,000) [3]. However, while the response to the test dose may be sufficient to identify an intrathecal or intravascular catheter insertion, it is often inadequate to accurately assess the onset of a sensory blockade originating from local anesthetic administered in the epidural space [4].

Recognizing the challenges associated with epidural catheter siting using the LOR technique, alternative methodologies, such as ultrasonography, fluoroscopy, and epidural electrical stimulation, have been explored as facilitators of procedural success. Image guidance, using either ultrasonography or fluoroscopy, offers a visual representation of anatomical landmarks that may increase first-attempt success rates [5,6]. While potentially beneficial, the widespread adoption of imaging techniques to assist with epidural catheter placement in the perioperative environment has lagged due to the time and resources required to employ these tools in preprocedural locations [7].

First described in 1998, the epidural electrical stimulation test (EEST) or “Tsui test,” describes the use of low-current stimulation through a saline-primed coil-reinforced epidural catheter to evoke a truncal motor response and confirm epidural catheter placement. Muscle movement in response to electrical stimulation, generally between 1 to 10 milliamps (mA), indicates the catheter tip is in the epidural space [8]. The technique offers a sensitivity of 80-100% in confirming correct lumbar and thoracic epidural catheter tip location, and can also be used to identify inadvertent subarachnoid, subdural, or intravascular catheter positioning with tools readily available in the perioperative environment [8-13].

We present a case of symptomatic bradycardia and hemodynamic instability, presumably a vasovagal response, induced by the initiation of the EEST in a patient undergoing surgical repair of a ventral hernia.

Case Report

A 61-year-old woman with a history of rectal cancer, treated surgically over 10 years prior with a low-anterior resection and end-colostomy, was scheduled to undergo a ventral and para-stomal hernia repair with component separation. The patient reported no prior cardiac or pulmonary comorbidities and was not prescribed any cardiovascular medications. A pre-procedural cardiac rhythm strip demonstrated a normal sinus rhythm at a rate of 67 beats per minute.

As part of a perioperative multimodal analgesic plan, the patient was offered a low thoracic epidural. After obtaining written consent in the pre-procedural area, the patient was placed in a sitting position on a transport stretcher with the assistance of a mobile epidural positioning device. Heart rate, electrocardiography, blood pressure, and pulse oximetry were monitored in accordance with the American Society of Anesthesiologists’ Standards for Basic Anesthetic Monitoring. Supplemental oxygen was delivered via nasal cannula at a rate of 2 L/min. Prior to initiating the procedure, 50 mcg of intravenous fentanyl was administered for patient comfort.

Anatomical landmarks assisted with the identification of the desired T9-T10 thoracic vertebral interspace for insertion of the epidural catheter. Skin overlying the site was prepared using a chlorhexidine solution and draped in a sterile fashion. Three milliliters of 1% lidocaine were used to anesthetize the skin and subcutaneous tissue. A 17-gauge Touhy needle was advanced with loss of resistance to saline at 5 cm on the first attempt. A spring-loaded 19-gauge PERIFIX® FX epidural catheter (B. Braun, Bethlehem, PA) was advanced without resistance into the epidural space to 20 cm at the proximal end of the Touhy needle. The Touhy needle was removed and the epidural catheter was pulled back to a depth of 15 cm at the patient’s skin. Following a negative aspiration of the epidural catheter, a Johan adapter (Figure 1) was attached to the epidural catheter connector. A sterile syringe of normal saline was then used to establish a continuous column of saline and allow for the conduction of a low-amplitude current delivered by a nerve stimulator (SunStim™, SunMed, Grand Rapids, MI) through the epidural catheter (Figure 2). The current intensity was increased by increments of 10% (7 mA) by an assistant outside of the sterile field. A positive evoked motor response of the abdominal muscles was detected at 21 mA by palpating the patient’s upper abdomen. During stimulation, the catheter was slowly pulled back to an optimal catheter tip position of 12 cm at the skin.
Several seconds after the initiation of current by the nerve stimulator, the patient’s electrocardiogram (ECG) changed from a normal sinus rhythm of 61 beats per minute to an idioventricular rhythm, absent of p-waves, at a rate of 38 beats per minute. Electrical anomalies, similar to pacing spikes, were visible on the ECG adjacent to each QRS complex (Figure 3). Blood pressure concomitantly dropped from 153/78 to 92/38 mmHg, and the patient reported feeling unwell. Oxygen saturation measured by pulse oximetry remained stable at 99%. The nerve stimulator was immediately turned off, which correlated with observed electrical interference disappearing from the ECG. The patient remained bradycardic following the cessation of the electrical stimulation prompting the administration of 0.2 mg of intravenous glycopyrrolate. The patient received an incremental epidural test dose of lidocaine 1.5% with epinephrine 1: 200,000 for a total volume of 5 mL (3 mL followed by 2 mL). The test dose was negative for signs of an intrathecal or intravascular injection. Following the test dose, decreased sensation was detected bilaterally at the dermatomal level of T10 in response to ice. The epidural provided adequate analgesia perioperatively, and the patient was discharged to home on postoperative day 3 following the removal of the epidural catheter without any additional adverse events.

**Discussion**

Although decades of clinical investigation have validated the EEST as a safe and effective confirmatory tool for epidural catheter placement, we raise the potential for the technique to precipitate a vasovagal response. We are unaware of any previous reports of symptomatic bradycardia attributed to the electrical stimulation technique.

Bradycardia and hypotension is a well-documented adverse event associated with neuraxial anesthesia [14-17]. However, it is most commonly encountered after the administration of local anesthetic where a medication-induced sympathectomy can leave parasympathetic tone unopposed. Vasovagal syncope or pre-syncpe, represents a similar constellation of signs that is not uncommon in peri-procedural settings. This neurally mediated reflex activation of sympathetic and parasympathetic loops is not completely understood, but can be generated...
from both the activation ofafferent neural pathways through the vagus nerve and emotional stress. While activation of the vagus nerve is the more well-described source of the observed vasodepressor response, significant emotional stress can produce similar physiological effects through the stimulation of the limbic sympatho-inhibitory center [18,19].

While vasovagal syncope and pre-syncope can be encountered during a neuraxial procedure prior to any pharmacologic intervention, the temporal onset of this patient’s symptomatic bradycardia and hypotension suggests a probable association with the EEST. The patient had yet to receive any local anesthetic in the epidural space and physiologic parameters changed only after the initiation of electrical stimulation.

Anatomically, the observed bradyarrhythmia could be described by 2 mechanisms: direct activation of sympathetic cardiac nerve fibers resulting in cardiac pacing or secondarily via vagus nerve stimulation. Given the depth of insertion of the epidural catheter at the time of initiation of the electrical stimulation and the proximity of the preganglionic sympathetic cardiac neurons (which originate primarily from the thoracic spinal segments between T1-T5), it is theoretically possible that the EEST induced a dysrhythmia. However, since the frequency of the delivered nerve stimulator output (1 stimulation every 0.5 seconds) translates to a rate of 120 stimulations per minute, it is more likely that the pacing spikes observed during the period of bradycardia represent electrical artifact. Direct stimulation of the vagus nerve may be a more plausible explanation of the findings, particularly given the peak current of 21 mA used to initially obtain a truncal motor response. Previous studies have demonstrated that epidural electrical stimulation can reduce arterial concentrations of norepinephrine and epinephrine, implying suppression of sympathetic tone [20]. Noting that the patient’s symptomatic bradycardia did not immediately resolve with cessation of the epidural stimulation test, it is likely that the electrical stimulation initiated a neural cascade that altered the autonomic balance.

Conclusions

This report describes a rare case of vasovagal pre-syncope associated with the EEST or “Tsui test.” While vasovagal reactions are not uncommon during neuraxial procedures, we raise the possibility that the EEST may have the potential to directly trigger such a physiologic response. We recommend vigilance regarding hemodynamic parameters during the administration of the confirmatory test and the immediate availability of sympathomimetic or anticholinergic agents to respond to neurally mediated cardiovascular events.

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