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Introduction
Spinal cord stimulators (SCS) are a non-pharmacologic, minimally invasive therapy for chronic neuropathic and ischemic pain refractory to conventional medical treatments. The most common indication for SCS placement is chronic pain from failed back surgery syndrome with radicular pain. Other indications include complex regional pain syndrome, peripheral neuropathy, phantom limb pain, angina and ischemic leg pain (3). Complications of SCS range from 5.3% to 40% and include lead migration, lead fractures, infection, seroma, tolerance, inadequate pain relief and dural puncture (4). The most severe potential complication is spinal epidural hematoma, which can be life threatening. The procedure is carried out by first inserting wire leads onto the epidural space under fluoroscopy to the appropriate spinal level. After successful placement of the lead or leads an incision is made to create a pocket for placement if the generator. The leads are then tunneled to the pocket and connected to the generator. The incisions are then sutured closed. Our report describes an SCS lead advancement through an adhesion/obstruction, transmitting pressure to the posterior thoracic epidural space, resulting in an immediate bradycardia.

Case Presentation
A 43 year old female with a history of chronic refractory lumbar back pain and obesity presented for revision of spinal cord stimulator. The leads from her initial SCS insertion had migrated and she was not receiving adequate pain relief from the therapy. Preoperative assessment was positive only for bilateral lower extremity radiculopathy. Induction was uneventful, and the patient was placed in the prone position. During the procedure, the surgeon’s attempt to advance the lead through scar tissue elicited severe bradycardia (HR 28) which resolved in about ten seconds. Glycopyrrolate (0.2 mg) was administered intravenously prior to resolution of the bradycardia. The remainder of the procedure was uneventful and the patient was discharged to the PACU without any further complications.

Discussion & Conclusion
Compression of spinal cord secondary to difficult lead placement could be the cause of this cardiovascular event. A search of current literature was unable to yield any other reported cases similar to ours. Our hypothesis is that the compression on the spine lead to an imbalance in the peripheral nervous system leading to the bradycardia. A known complication of spinal

Compression is hypotension and bradycardia (1), although in our case the event did not last long enough to determine if the patient became hypotensive. Bradycardia associated with spinal cord injury has been extensively described. The mechanism of the bradycardia has been described as disruption of descending pathways from central control centers to spinal sympathetic neurons, originating from T1-L2, which can lead to loss of supraspinal control over the sympathetic nervous system leading to unopposed parasympathetic activity through the vagus nerve (2). Anesthesiologists need to be aware that severe bradycardia can occur during spinal cord stimulator implantation.

References

This research was supported in whole or in part by HCA and/or an HCA affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA or any of its affiliated entities.