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## Sudden Cardiac Arrest during Epidural Anesthesia

*To the Editor:*—We report a case of sudden cardiac arrest during epidural anesthesia to remind practitioners of this rare but potentially devastating complication.

The patient, a 35-yr-old woman with non-Hodgkins lymphoma was scheduled for bone marrow harvesting. She had initially presented in 1985 and had been successfully treated with cytotoxics and radiotherapy. Her current admission was occasioned by a relapse with retroperitoneal involvement. She denied any history or symptoms of cardiovascular disease, and had undergone general anesthesia in the past without complications. There was no history of drug allergy, and all her preoperative laboratory tests (including ECG and chest roentgenogram) were within acceptable limits.

No preanesthetic medication was administered, and an epidural catheter was inserted using a midline approach at the L<sub>3</sub>–L<sub>4</sub> interspace with the patient in the sitting position. The patient was placed prone on the operating table, and a total of 23 ml 2% lidocaine without epinephrine was injected through the catheter over a 30-min period. During and after the performance of the epidural, a total of 150 µg fentanyl and 2 mg midazolam were administered intravenously for sedation. Monitoring included five-lead ECG, pulse oximetry (SpO<sub>2</sub>), and automated blood pressure by cuff. Oxygen at 4 l/min *via* nasal cannulae was administered throughout.

The patient's blood pressure on admission to the operating room was 100 mmHg systolic. During and after the administration of epidural lidocaine, her pressure decreased to 90 mmHg systolic. ECG revealed normal sinus rhythm at 77–90 beats per min. SpO<sub>2</sub> remained in the 98–100% range throughout the procedure and up to the events described below.

Fifty minutes into the procedure, and 45 and 55 min after administration of lidocaine and sedation, respectively, the patient's cardiac rhythm was noted by the authors suddenly to change from sinus at a rate of 80 beats per min to complete atrioventricular block followed almost immediately by complete cardiac arrest. Immediately prior to the arrest, SpO<sub>2</sub> was 99%, and blood pressure was 85 mmHg systolic. The patient, while not spontaneously conversing at this point, was easily arousable by verbal stimuli alone. A total of 1,200 ml bone marrow had been aspirated to this point, and volume replaced with one and one half units of packed red cells and approximately 1,500 ml crystalloid.

The patient was immediately turned onto her back, her lungs ventilated by mask with 100% oxygen, and external cardiac compressions commenced in the standard fashion. One milligram of atropine was intravenously administered at this time. Within 2 min, spontaneous cardiac activity was noted on the ECG monitor. The patient started to breathe spontaneously and opened her eyes. The sensory level of the block was assessed at this time and found to be at T<sub>5</sub>. No further perturbations of blood pressure or cardiac rhythm were noted over the next 24 h. No neurologic sequelae ensued.

Sudden and unexplained cardiac arrest constitutes a rare but significant complication during spinal anesthesia.<sup>1,2</sup> The ASA Closed Claim Study reported 14 cases of unexpected and sudden cardiac arrest during spinal anesthesia, arrests which "seemed to evolve with unexpected speed against a background of apparently stable hemodynamics."<sup>1</sup> Of these 14 cases, 6 died and 7 sustained permanent neurologic injury. Potential causes for this dysrhythmia include intrinsic atrioventricular

nodal disease, hypotension-induced myocardial ischemia, coronary artery disease, and unopposed vagal tone due to spinal induced sympathectomy.<sup>2</sup> The role of respiratory depression secondary to the administration of opioids and/or sedative-hypnotic agents in sudden cardiac arrests in these patients also has received mention.<sup>1,3</sup> Caplan *et al.* suggested that respiratory changes produced by sedation may have played a role in approximately one half of their reported cases,<sup>1</sup> and Knill, in a Letter to the Editor in response, amplified on this theme.<sup>3</sup> The appearance of this complication during epidural anesthesia also has been reported,<sup>4</sup> the causation and mechanism presumably the same as those for spinal anesthesia.

While the event described herein occurred 55 min after the administration of fentanyl and midazolam, and while the patient was easily arousable by verbal stimulation alone, the possibility of the existence of hypercarbia as a contributing factor in the patient's cardiac arrest is real. This possibility is not negated by the existence of normal SpO<sub>2</sub> immediately prior to the arrest. The routine use of capnography in this setting, in addition to the often-recommended SpO<sub>2</sub>, thus appears to be reasonable. Both of these monitoring modalities also serve to detect the effects of the prone position on respiration, another possible complicating factor in this case.

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