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Bradycardia and Asystole during Spinal Anesthesia: A Report of Three Cases Without Morbidity

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Although bradycardia has long been accepted to be a typical cardiovascular response to high spinal anesthesia, the phenomenon of abrupt, extreme bradycardia and/or asystole has recently received renewed attention. For example, Caplan *et al.* reported 14 cases of sudden cardiac arrest in healthy patients who had received spinal anesthesia.[1] These authors stated that the potential for sudden cardiac arrest in the setting of apparent hemodynamic stability is present, yet poorly understood. They speculated that unappreciated respiratory insufficiency may have been an important factor in approximately 50% of their cases. We report one case of extreme bradycardia and two cases of asystole during spinal anesthesia that were not associated with hypoxemia, obvious respiratory depression, or adverse outcome.

CASE REPORTS

Case 1. A 42-yr-old, 52-kg woman was scheduled for amputation of the distal phalanx of the left great toe because of osteomyelitis. She had a 20-yr history of cigarette smoking and a 5-yr history of diabetes mellitus complicated by peripheral vascular disease and neuropathy. Medications consisted of insulin and dicloxacillin. Physical examination, laboratory evaluation, and ECG were unremarkable.

Upon arrival in the operating room, ECG, automated blood pressure cuff, and pulse oximeter (Sp_{O_2}) monitors were applied. The preoperative blood pressure was 116/80 mmHg and the heart rate was 84 beats/min with sinus rhythm. Fentanyl 50 μg and midazolam 1 mg intravenously (iv) were given, and nasal oxygen supplementation at 3 l/min was begun. Spinal anesthesia was administered with hyperbaric lidocaine 60 mg and epinephrine 0.2 mg, while the patient was in the left lateral decubitus position, with the table horizontal. Five minutes later the heart rate abruptly decreased to 37 beats/min with intermittent junctional complexes, and the blood pressure decreased to 72/45. This rhythm persisted for approximately 15 s before slowing to a rate of 15 beats/min, at which time ventricular escape beats appeared. Atropine 0.6 mg and ephedrine 7.5 mg were administered iv, with a prompt increase in heart rate to 80 beats/min, a return of normal sinus rhythm, and a blood pressure of 140/95. The patient remained conscious throughout (total duration less than 1 min), and the Sp_{O_2} was never less than 96%. Sensory block level to pinprick was determined immediately after return of normal sinus rhythm and found to be T_3 bilaterally. The remainder of the anesthetic course and hospitalization was unremarkable. A postoperative ECG was unchanged.

Case 2. A 60-yr-old, 69-kg woman was scheduled for rectal examination under anesthesia and excision of anal fissure. Three years prior to admission she experienced an episode of rapid heartbeat, was found to have paroxysmal atrial fibrillation with ambulatory Holter monitoring, and digoxin 0.25 mg/day was begun with no additional symptoms and no additional ECG evidence of dysrhythmia. The digoxin level at this dose was subtherapeutic (0.5 ng/ml). The patient also had a history of hypertension, well controlled with atenolol and hydrochlorothiazide. Preoperative evaluation revealed blood pressure of 144/78 and heart rate of 82 beats/min. Physical examination and laboratory evaluation were normal. The preoperative ECG showed normal sinus rhythm with nonspecific T-wave changes.

After iv premedication with fentanyl 50 μg and midazolam 1 mg, ECG, automated blood pressure cuff, and pulse oximeter monitors were applied. Spinal anesthesia was administered with hyperbaric tetracaine 6 mg and epinephrine 0.2 mg. Twenty minutes later the patient was placed in the jackknife position. Sensory level to pinprick was noted to be T_4 bilaterally at that time. Supplemental oxygen 3 l/min was administered and the patient received no additional sedative medication. During the initial 15 min of the operation the blood pressure remained approximately 100/60 mmHg, and the heart rate decreased gradually from 60 to 48 beats/min. At that point (35 min after the spinal was administered) the patient complained of nausea and the heart rate fell abruptly to 27 beats/min for 10 s, followed by 10 s of asystole. Atropine 0.4 mg iv was administered. Initially, P-waves without associated QRS complexes were observed, but 5 s after atropine, sinus rhythm returned with a rate of 60 beats/min and a stable blood pressure. Verbal communication with the patient was maintained up to the point of asystole, and the Sp_{O_2} was consistently 100%. Prior to asystole, no ECG abnormalities were present other than sinus bradycardia. Immediately following the return of stable pulse and blood pressure, the patient's sensory block level to pinprick was again noted to be T_4 bilaterally. The remainder of the anesthetic course and hospital stay were unremarkable. A postoperative ECG was unchanged, and 18 h of ambulatory telemetry revealed no abnormalities.

Case 3. An 85-yr-old, 50-kg woman with a history of normal pressure hydrocephalus, peptic ulcer disease, and depression was scheduled for removal of a dynamic hip screw. Medications consisted of ranitidine and lorazepam. Physical examination, laboratory evaluation, and ECG were unremarkable.

Upon arrival in the operating room, ECG, automated blood pressure cuff, and pulse oximeter monitors were applied. The preoperative blood pressure was 125/55 mmHg and the heart rate was 70 beats/min with sinus rhythm. After iv sedation with thiopental 40 mg and nasal oxygen supplementation at 3 l/min, spinal anesthesia was administered with hyperbaric lidocaine 50 mg and epinephrine 0.2 mg. No additional sedation was given. Immediately afterwards, prophylactic ephedrine

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25 mg intramuscularly (im) was given, and the Sp_{O_2} was noted to be 98%. Five minutes later the sensory block level to pinprick was found to be T_4 bilaterally. Eight minutes after performing the spinal anesthetic, the patient abruptly complained of nausea and then lost consciousness. ECG revealed P-waves without associated ventricular complexes for 6 s. This abrupt change occurred while the patient was conversing with her anesthesiologist. Just prior to cardiac arrest, the Sp_{O_2} was 98%, the blood pressure was 120/50 mmHg, and the heart rate was 68 beats/min (fig. 1). External cardiac compression was immediately initiated and atropine 0.2 mg and ephedrine 20 mg iv were administered. This was followed by the immediate return of sinus rhythm at a rate of 85 beats/min and a blood pressure of 130/85 mmHg. Spinal anesthesia level to pinprick was again noted to be T_4. The patient complained of continued nausea and was given droperidol 0.625 mg iv, with subsequent relief. The remainder of the anesthetic course and hospitalization was uneventful.

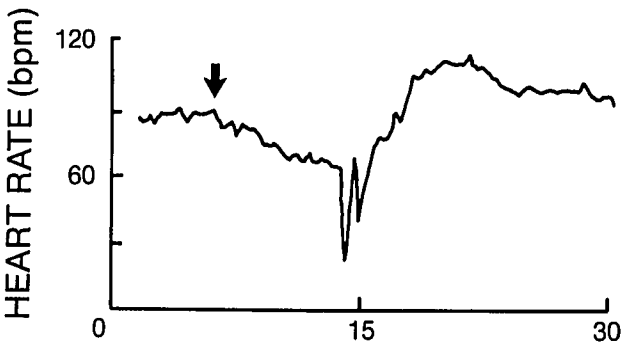

FIG. 1. Trended output of heart rate (from the ECG monitor) for the first 30 min of anesthetic care for case 3. Spinal anesthesia was administered at the arrow. Note the gradual slowing of heart rate prior to the abrupt decline. Atropine 0.2 mg and ephedrine 20 mg were administered iv and chest compressions were immediately instituted. The heart rate rapidly responded to this therapy.

DISCUSSION

The bradycardia typically associated with spinal anesthesia is slow in onset, moderate in severity, and easily treated. The onset of this bradycardia has been attributed both to loss of cardiac sympathetic stimulation and to decreased venous return to the heart.[2] A spinal anesthetic with a sympathetic blockade level of T_1 or higher results in unopposed cardiac vagal input, with its negative chronotropic, dromotropic, and inotropic effects.[3,4] Decreased venous return to the heart may induce bradycardia by either: 1) activating a reflex arc with stretch receptors located in the junction between the superior vena cava and the right atrium[5]; or 2) an intracardiac reflex in which heart rate is proportional to the degree of pacemaker stretch.[6]

The precipitous onset of severe bradycardia or asystole that we report also appears to be a clinical event that can occur during spinal anesthesia. Although this is considered an uncommon event, it may not be rare. The three cases we report occurred over 6 wk, during which time we performed approximately 180 spinal anesthetics. We are not certain whether this incidence merely reflects an increased awareness and reporting of these events or whether this is a random grouping of rare events.

Why should some spinal anesthetics produce asystole or extreme bradycardia? The abrupt onset suggests a reflex process. Although the two reflex mechanisms listed above have been previously suggested, we suggest that the Bezold-Jarisch reflex should also be considered. The classic Bezold-Jarisch reflex arises from mechanoreceptors and chemoreceptors located primarily in the inferoposterior wall of the left ventricle.[7,8] Stimulation of these receptors by stretch or chemical substances increases parasympathetic activity and inhibits sympathetic activity, producing bradycardia, systemic vasodilation, and hypotension.[7] Normally, a decrease in left ventricular end-diastolic volume results in a decrease in receptor activity. However, a rapid decrease in ventricular volume can actually stimulate and increase the activity of these receptors, presumably due to vigorous ventricular contraction around an almost empty chamber.[9] This "paradoxical" stimulation of the ventricular receptors in the face of hypovolemia results in a paradoxic Bezold-Jarisch reflex.[7-11] We speculate that a rapid decrease in ventricular volume during spinal anesthesia can paradoxically activate the Bezold-Jarisch reflex and thereby induce acute bradycardia or asystole. Similarly, paradoxical stimulation of the Bezold-Jarisch reflex has been suggested as the etiology for profound bradycardia, asystole, and sudden death associated with a variety of clinical syndromes including vasovagal syncope,[7-10] aortic stenosis,[12] and recurrent pulmonary embolism.[13]

Another possible mechanism for asystole or profound bradycardia would be preexisting autonomic imbalance or dysfunction. Syncope, profound bradycardia, and sudden cardiopulmonary arrest have been reported in unanesthetized patients with autonomic dysfunction (*e.g.*, patients with diabetes,[14] AIDS,[15] or the aged[16]). Because spinal anesthesia represents a challenge to the autonomic mechanisms of homeostasis, preexisting derangement in autonomic nervous system function may increase the propensity for bradycardia or asystole. Autonomic dysfunction may have played a role in two of our patients because one was diabetic and another was 85 yr old.

The possibility that premedicants or therapeutic drugs may contribute to the bradycardia cannot be excluded.[17,18] Two of our patients received fentanyl as a premedicant and one was treated with digitalis and atenolol. These drugs may have contributed to slowing of the heart rate.

One conclusion we can make is that patients who receive spinal anesthesia require constant monitoring and vigilance. Our patients developed bradycardia or asystole at 5, 8, and 35 min after the spinal anesthetic was performed. Immediately prior to the abrupt decrease in heart

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rate, these patients were hemodynamically stable, well oxygenated, and were not heavily sedated. Two of our patients had a slow decline in heart rate prior to the sudden decrease (fig. 1). This gradual decrease in heart rate supports our original speculation that the abrupt and unexpected nature of the cardiac arrest is partially explained by a sudden onset and partially by sudden recognition.[19] We emphasize that our patients had no morbidity associated with their slowed heart rate. All were easily resuscitated using atropine with or without ephedrine, and only one required closed chest cardiac massage.

Previous authors[20,21] have speculated that excessive sedation and unrecognized hypoxemia may have caused the cardiac arrests reported by Caplan *et al.*[1] These three cases indicate that neither factor is required; cardiac arrest occurred in patients in whom Sp_{O_2} was normal and who were minimally sedated. In contrast to Caplan's report,[1] our patients were easily resuscitated, did not require treatment with iv epinephrine, and recovered without morbidity, despite the fact that they were older and more infirm. Perhaps our patients were better able to communicate their distress because they were lightly sedated and the resulting early recognition and treatment contributed to the prevention of an adverse outcome.

In summary, we report the sudden onset of profound bradycardia and asystole during spinal anesthesia in patients who are not hypoxic. Although the exact pathophysiology of this phenomenon is unknown, the etiology is probably multifactorial. The abrupt onset of severe bradycardia or asystole suggests a reflex mechanism, and we speculate that the Bezold-Jarisch reflex may be responsible. Preexisting autonomic dysfunction may increase the incidence and/or severity of this reflex response.

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