Paroxysmal atrioventricular block in a healthy patient receiving spinal anesthesia: A case report

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The presentation of type 1, second-degree atrioventricular block (AVB) in a young, healthy patient is rare. This article describes such an event in a 36-year-old white male under subarachnoid block (SAB) anesthesia.

At 25 minutes into surgery, the pulse oximeter and the ECG monitor both alarmed for nonsensing and asystole, respectively. The patient had experienced a complete AVB. "Cough cardiopulmonary resuscitation (CPR)" was begun, and the patient was instructed to take deep breaths and cough as hard as possible. During the entire duration of the AVB, the patient never lost consciousness and continued cough CPR, with encouragement from the author. Ephedrine 12.5 mg in two doses was administered through the rapidly infusing intravenous line. The patient was continued on oxygen, as described, and the pulse oximeter continued to demonstrate a saturation greater than 96% during the entire event. After approximately 40 seconds, the patient's rhythm changed to a type 1, second-degree AVB. After an additional 15 minutes, the patient's sinus rhythm returned to normal.

This event emphasizes the value of vigilance. Continuous monitoring, including pulse oximetry, blood pressure measurements, and continuous ECG, should be used on all patients receiving any form of anesthesia.

Key words: Atrioventricular block, complications, spinal anesthesia.

Introduction
Although a variety of atrioventricular blocks (AVB) occur during the course of an anesthetic in patients with preexisting cardiovascular disease, the presentation of a type 1, second-degree AVB in a young, healthy patient is rare and accounts for only a small number of closed insurance claims. This is an account of a healthy 36-year-old male who developed a second-degree AVB during a subarachnoid block (SAB) anesthetic.

Case presentation
A 36-year-old white male presented in the emergency room with a trimalleolar fracture to his right ankle, which resulted from a fall from his farm tractor in which he twisted his ankle.

A preoperative anesthesia evaluation revealed a healthy, thin adult white male with no prior exposure to either general or regional anesthesia. His only exposure to anesthetic agents had been during dental procedures which, he recalled, resulted in no untoward effects from the local anesthesia. In fact, no member of his immediate family had ever experienced an untoward response during anesthesia as far as the patient could recall. The patient...
was on no medications, and he used alcohol infrequently.

An SAB anesthetic technique was selected for this patient, and because he had eaten only 3 hours prior to presentation, sodium citrate 30 mL by mouth was given as premedication prior to his transfer to the operating room; no other preoperative medications were given.

After the patient arrived in the operating room, standard monitors were applied, including noninvasive blood pressure that had been set to cycle every 2½ minutes, continuous electrocardiograph (ECG), continuous pulse oximeter with waveform display and waveform tone-activated, and a temperature strip.

The initial vital signs were systolic blood pressure 118 mmHg, diastolic blood pressure 62 mmHg, pulse rate 71 per minute, and respiratory rate 10 breaths per minute. Initial examination of the ECG revealed a normal sinus rhythm at 71 per minute. There was no evidence of ectopic pacemaker activity or conduction aberration. An intravenous infusion of lactated Ringer’s with 5% dextrose was begun, and the patient received 400 mL prior to induction of the spinal anesthetic. Oxygen at 2 L/min by nasal prongs was administered.

At this time, midazolam 2.5 mg was administered intravenously to provide sedation, and the patient was turned on his left side for the induction of the spinal anesthetic. The patient remained alert and oriented during this time.

A 25-gauge spinal needle was introduced at a level of the L3-4 interspace. After a free flow of cerebral spinal fluid was seen, 70 mg of a hyperbaric solution of lidocaine 5% with dextrose 7.5% was injected to produce spinal anesthesia, and the patient was returned to a supine position. The level of anesthesia achieved was approximately at the T12-L1 level. The patient was alert, oriented, and talking quite freely during this period of time.

After the induction of SAB, the patient’s vital signs remained stable, with the systolic blood pressure maintained at 120 mmHg and the diastolic pressure at 70 mmHg. The heart rate remained at 70 beats per minute, with the ECG demonstrating a normal sinus rhythm with no evidence of aberration or ectopy. The respiratory rate remained at 10 breaths per minute. The level of anesthesia remaining was reevaluated and determined to be at the T12-L1 level so the surgeon began the operative procedure. Meanwhile, the patient actively continued a conversation with the surgical staff and the anesthetist.

At 25 minutes into surgery (42 minutes into anesthesia), the surgeon was closing the subcutaneous tissue layer. At this time, there was a paroxysmal onset of a sinus bradycardia (30 per minute) with a complete AVB. The patient became dizzy but remained alert. The pulse oximeter and the ECG monitor both alarmed for nonsensing and asystole, respectively. The noninvasive blood pressure monitor showed a systolic blood pressure of 60 mmHg, with a diastolic pressure of 40 mmHg. (The ECG strip recorder was not on automatic, so there is no hard copy record of the complete AVB.)

"Cough cardiopulmonary resuscitation (CPR)" was begun, with the patient instructed to take deep breaths and cough as hard as possible. During the entire AVB, the patient never lost consciousness and continued cough CPR with encouragement from the author. Ephedrine 12.5 mg in two doses was administered through the rapidly infusing intravenous line. The patient was continued on oxygen, as described, and the pulse oximeter demonstrated a saturation greater than 96% during the entire event. After approximately 40 seconds, the patient’s rhythm changed to a type 1, second degree AVB (Figure 1). Fifteen minutes later, the patient’s sinus rhythm had returned to normal. (Figure 2). However, as a prophylactic measure, an external cardiac pacemaker was applied in the event that there were repeat episodes of the bradycardia.

The remainder of the anesthesia process was uneventful, and the patient was transferred to the cardiac care unit (CCU) for monitoring and evaluation.

Upon his admission to the CCU, an ECG was obtained from the patient that showed no evidence of acute injury or dysrhythmia; however, after 5 more hours a repeat ECG showed ST segment elevation over the inferior wall leads. This elevation had resolved by the time another ECG was obtained 2 hours later. Cardiac enzymes remained within normal limits.

Consultation with a cardiologist produced no evidence of a cardiac abnormality, and the patient was transferred from the CCU on the second postoperative day, having experienced no further episodes of dysrhythmias.

Discussion

The progression of a first degree AVB to a second degree AVB through the blockade of cardiac sympathetic neurons has been offered as an explanation for some presentations of AVB under SAB.4 Atrioventricular block may be present when the level of block achieved includes the cardiac accelerators that arise from the upper four or five thoracic sympathetic ganglia or if a high spinal anesthetic is accompanied by the administration of
fentanyl. In this case, the level of spinal anesthesia was maintained at the T12-L1 level, and there was no narcotic administered.

Greene has suggested that the peripheral vasodilation produced by hypoxia and hypercarbia can result in a sufficient decrease in venous return and atrial filling. When this decrease is accompanied by the unopposed vagal tone produced by a high spinal, the combination might produce bradycardia and cardiac arrest. In this case, the cardiac arrest was not accompanied by a decrease in oxygen saturation, and the patient demonstrated none of the alterations in vital signs or mental acuity that might be seen with hypercarbia.

Caplan and associates have noted the impact sedation has on patients under SAB who suffer cardiac arrests during surgery, pointing out that, in a group of 14 patients, nine had received sufficient narcotic and sedatives to produce a "... sleep-like state in which there was no spontaneous verbalization." The patient described in this case report remained fully alert and carried on a conversation throughout the procedure until the occurrence of the bradycardia and during the complete AVB period.

Brown and colleagues have demonstrated that some sudden bradycardias are actually of longer duration. The patient in this case maintained an adequate heart rate and rhythm and a blood pressure consistent with preinduction values.

Most of the literature reviewed concerned cardiac arrests in patients under SAB. Although this episode had an excellent outcome, if treatment had not been initiated quickly with excellent patient response, the outcome could well have been cardiac arrest and death.

Ephedrine, a sympathomimetic agent, was chosen over a parasympatholytic agent such as atropine, because of the nature of the patient's conduction abnormality. The case was most notable because of the complete lack of any subordinate pacemaker. Normally, subordinate cardiac pacemaker cells in
the atria, junctional apparatus, or ventricle will produce escape impulses in the absence of any "higher" pacemaker activation (Figures 3-5). In this case, the loss of normal sinoatrial pacing and the complete absence of atrioventricular conduction were made more critical by the total lack of any atrial, junctional, or ventricular escape mechanism. Because parasympathomimetic innervation to the ventricles is sparse, the reason for this episode of bradycardia and complete AVB with no ventricular escape mechanism must include some component of decreased sympathetic tone, as described by Caplan and associates.6

Ephedrine was the initial choice for intervention because of its immediate availability; it was provided in the spinal tray used in this case. It was given within seconds of the onset of the event. Caplan and associates suggest that alpha agonists such as ephedrine and epinephrine produce the most satisfactory outcomes in cases of cardiac arrest in patients under SAB anesthesia. They further suggest that epinephrine may produce a more satisfactory result. In this case, the lack of any subordinate pacemaker in this patient during the entire period of complete loss of atrioventricular conduction and secondary cardiac pacemaker activity suggests that the immediate introduction of a sympathetic agent would produce better results, and the patient did have an excellent response to the administration of ephedrine.

Figure 3
Normal sinus rhythm at 72 per minute with a sinus arrest of 1.28 seconds terminated by an atrial escape complex

Figure 4
Normal sinus rhythm at 72 per minute with a sinus arrest of 1.28 seconds terminated by a junctional escape complex
Figure 5
Normal sinus rhythm at 72 per minute with a sinus arrest of 1.28 seconds terminated by a ventricular escape complex

P wave should have appeared here.

1.28 seconds

**Conclusion**

Although profound cardiac events can occur in patients under SAB who are heavily sedated, have a high level of spinal anesthesia, are compromised in their ventilations, or who experience a gradual decline in heart rate or blood pressure, this case demonstrates that AVB can occur in patients whose vital sign values remain excellent, who do not have high levels of spinal anesthesia, and who are awake and alert.

This event further emphasizes the value of vigilance. Caplan and associates suggested that, at a minimum, pulse oximetry should be employed when the patient's ability to communicate is impaired and that epinephrine should be available for early treatment of bradycardias. Continuous monitoring, including pulse oximetry, blood pressure measurements, and continuous ECG should be used on all patients receiving any form of anesthesia.

**REFERENCES**


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Michael Mitton, CRNA, graduated from St. Joseph Hospital School of Anesthesia in 1979. He is currently providing anesthesia in Shelbyville, Indiana. Mr. Mitton is the founder of Professional Education Services and has written a number of computer programs designed for the active anesthesia clinician. Mr. Mitton has lectured extensively on electrocardiography, dysrhythmias, regional anesthesia, and invasive monitoring techniques.