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CASE REPORT

Maintained consciousness during witnessed asystole after spinal anesthesia for Cesarean section [version 1; peer review: 3 approved with reservations]

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Abstract

Despite its low incidence, cardiac arrest after spinal anesthesia carries a high mortality. Counterintuitively, young and healthy patients with low resting pulse are at increased risk. We report the case of a healthy 24 yr G₂P0 at term scheduled for elective Cesarean section whose heart rate decreased to 30 bpm, followed by more than 30 seconds of asystole 3 minutes after spinal anesthesia with a T4 level block. Following atropine and epinephrine administration, the patient had several single heart beats when startled by the anesthesiologist's loud voice and when touching her chest to prepare for chest compressions. Eventually, regular sinus rhythm returned with a heart rate of up to 160 bpm. The patient was rapidly prepped, and within 5 minutes, the fetus was delivered surgically with Apgar scores of 8 and 9. Most unusually, the patient remained responsive during the entire event and denied having lost consciousness. Supine position and volume loading may have contributed to venous pooling within the cerebral vasculature, so even in the absence of cerebral blood flow during asystole venous blood may still have been present and delayed cerebral hypoxia. Therefore, loss of consciousness in the supine position may occur considerably after the onset of asystole which may reduce the time available for treatment and contribute to its high mortality. Inspiration during the two startle reactions may have decreased vagal tone and permitted enough spontaneous cardiac activity to circulate the resuscitative drugs without CPR.

Keywords

obstetrics, cesarean section, spinal anesthesia, asystole, consciousness

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Any reports and responses or comments on the article can be found at the end of the article.

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Introduction

Cardiac arrest after spinal anesthesia is rare, recently reported to occur in 1.5/10,000 anesthetics¹. Despite its low frequency, it was believed to be associated with a high mortality rate, reported as >40% mortality in the closed claims analysis by Caplan *et al.*². We present a unique case of cardiac arrest after spinal anesthesia for Cesarean section. We discuss relevant physiological mechanisms to explain why this patient never lost consciousness during the asystolic episode, which could mislead clinicians into delaying treatment.

Case Report

A healthy Spanish speaking 24 year old G₂P₀ female (70 kg, 65", BMI 26.5) at term was scheduled for an elective Cesarean section for breech presentation. The patient's preoperative heart rate (HR) was 62 beats per minute (bpm), blood pressure was 126/70 mmHg. After administration of 1000 ml Ringer's lactate solution, spinal anesthesia was performed with 1.6 cc of 0.75% bupivacaine (12 mg) in a sitting position, resulting in a bilateral T4 sensory tested by pinprick. No sedatives or prophylactic vasopressors were administered. The patient was laid supine immediately after spinal anesthesia with a left lateral tilt to prevent aorto-caval compression. SpO₂ remained at 98%. Three min later her HR decreased to 30 bpm, followed by 15 sec of asystole, which was immediately noticed via pulse oximeter and EKG. 1 mg atropine was administered intravenously (IV). The anesthesiologist's loud voice shouting for resuscitative help appeared to startle the patient who turned towards him. Now, 3 beats at 40 bpm were noted, followed again by asystole for 30 sec. 70 mcg of epinephrine IV was given, the anesthesiologist repeated the call for help, and removed the blanket and gown from her chest to begin chest compressions. The patient again appeared startled by this action based on facial expressions while turning towards the anesthesiologist, and a single spontaneous heart beat reappeared on the monitor. Another beat followed within 2 sec, rapidly accelerating to 160 bpm (sinus tachycardia). The patient was rapidly prepared, and the fetus was delivered within 5 min with Apgar scores of 8 and 9. Afterwards, the events were discussed with her with the help of an interpreter. She had been unaware of the cardiac arrest, and denied having lost consciousness.

Discussion

Spinal anesthesia is associated with an increased risk of hypotension and bradycardia³. ASA 1 status, baseline HR <60 bpm, a prolonged PR-interval, beta-blockade, age <50 years, a sensory blockade above T6 and male gender have been described as independent risk factors²⁻⁵. The differential diagnosis for asystole in this setting includes hypoxia due to respiratory depression, high or total spinal, local anesthetic toxicity when an epidural dose is administered IV, myocardial ischemia, vasovagal reaction, and bradycardic reflexes after neuraxial anesthesia. Our

patient was unsedated, and oxygenation maintained prior to the arrest. In fact, in contrast to earlier studies that suspected oversedation², it is difficult to invoke hypoxemia as the primary cause of cardiac arrests during this case or historical reports because most asystolic arrests occurred in the setting of 95–100% oxygen saturations⁶. Spinal anesthesia itself is associated with bradycardia in 9–13% of patients³. Most of these effects are related to sympathetic blockade during spinal anesthesia. Sympathetic blockade is often two to six levels higher than the sensory level, so that a patient with a T4 sensory block may have completely blocked cardioaccelerator fibers originating from T1 to T4⁷. However, in a prospective study of 952 patients, Carpenter *et al.*³ found that the traditional risk factor peak block height had the weakest correlation with the severity of bradycardia.

A more important effect of sympathetic inhibition is a significant decrease in venous return to the heart that enhances cardiac vagal tone⁸. The decrease in venous return could further be accentuated by aorto-caval compression despite the application of a left lateral tilt position, since no particular position has been shown to completely abolish caval compression⁹. Cardiac vagal tone may be enhanced by three different mechanisms (4): 1) Decreased venous return results in decreased stretch of atrial pacemaker cells resulting in bradycardia; 2) Firing of low pressure baroreceptors in the right atrium and vena cava and 3) A Bezold-Jarisch reflex, in which left ventricular mechanoreceptors are stimulated and paradoxically cause bradycardia. Earlier studies suggested that vasodilation caused by sympathetic blockade may be best treated by preemptive fluid administration^{7,8}. However, more recent data suggest that crystalloids alone (without vasopressors) are inconsistent in preventing hypotension¹⁰.

A high or total spinal anesthesia was unlikely, since our patient responded appropriately to pinprick sensations at or above the T4 level. Nevertheless, a T4 level block could completely block cardioaccelerator fibers originating from T1 to T4 resulting in severe bradycardia. Caplan *et al.* in their closed claims analysis noted the median time from local anesthetic administration to cardiac arrest to be 36±18 minutes (2); however, most of these cases were non-obstetric. The onset of asystole in our case was 3 min from initiation of spinal anesthesia which could have resulted from a sympathetic block accentuated by at least partial aorto-caval compression by the gravid uterus. Supine hypotensive syndrome of pregnancy has been well described in the literature and can cause a marked bradycardia with a reduction in cardiac output and severe hypotension in a few subjects¹¹.

While women are 11 to 50 times less likely to develop bradycardia during neuraxial anesthesia than men¹², parturients typically have a higher HR and are therefore even less likely

to develop bradycardia or cardiac arrest during neuraxial anesthesia⁵. Carpenter *et al.* reported a low baseline heart rate (<60 bpm), ASA 1 status (when compared to ASA 3 and 4) and beta-blockade as the strongest predictors of bradycardia³. With her baseline HR of 62 bpm our patient may have been at higher risk.

Case reports exist in which patients were amnesic for episodes of unconsciousness, as documented with carotid sinus syndrome¹³. Despite the language barrier, our patient exhibited signs of consciousness during asystole via her startle reactions and turning her head to unexpected shouting and light tactile stimulation. This demonstrates that a patient could remain conscious for up to 30 sec despite being asystolic in isolated cases. This delay in loss of consciousness could substantially delay recognition of a life threatening event, especially if a patient is temporarily disconnected from monitoring. It therefore underscores the importance of continuous monitoring during spinal anesthesia with pulse oximetry and EKG at all times.

There are several theoretical mechanisms that may be postulated to explain the maintenance of cerebral oxygenation in this scenario. First, supine position and volume loading may have contributed to venous pooling within the cerebral vasculature. Studies have also demonstrated that stroke volume and CVP increase during vagally induced bradycardia¹⁴, so that a few heart beats during the asystolic episode may have produced a disproportionately large amount of blood flow in this setting. Whether aortic compression by a gravid uterus enhances cerebral blood flow at the expense of lower extremity blood flow remains unknown. Through their experiments on healthy controls Ide *et al.* showed that an impaired ability to increase cardiac output during exercise with a large muscle mass appears to limit blood flow distribution not only to active muscle¹⁵ but also to such a vital organ as the brain, and that this flow restriction was by way of the sympathetic nervous system¹⁶. During central blood volume depletion, the increase in sympathetic nerve activity shifts the cerebral autoregulation curve to the right¹⁷ and the vasoconstrictor sympathetic nerve activity overrides vasodilatation¹⁸. Ide *et al.* also showed that sympathetic blockade at the level of the neck eliminated the limitation to the increase in cerebral blood flow following cardio-selective β -1 adrenergic blockade¹⁶. Although a theoretical hypothesis, a T4 level blockade in our patient may have blunted the effect of reduced cardiac output on cerebral vasoconstriction, and may have helped maintain cerebral perfusion prior to the asystolic events.

An auditory startle stimulus produces a transient tachycardia 2 to 5 sec after the stimulus, consistent with our findings; the reflex is mediated by the vagus nerve and sympathetic ganglia¹⁹.

The initial startle response was followed by 3 spontaneous heart beats and probably occurred before the atropine had time to circulate through the bloodstream. The second startle reaction perhaps permitted enough cardiac activity to circulate the resuscitative drugs without CPR. A deep inspiration accompanying the startle response may have further reduced vagal tone by a mechanism similar to that seen with respiratory sinus arrhythmia (RSA)²⁰. RSA is a cardiorespiratory phenomenon characterized by HR or R-R interval (RRI) fluctuations in phase with inhalation and exhalation. Typically, HR accelerates during inspiration and slows during expiration, and is prominent during high resting vagal tone; increased sympathetic stimulation attenuates it. Rate and depth of respiration also influence the magnitude of RSA with slow deep breathing producing maximal and rapid shallow breathing greatly attenuating RSA. Unfortunately, no EKG strips are available to confirm the above assumptions as the anesthesiologist was busy resuscitating the patient.

Conclusion

We present a unique case of sudden cardiac arrest after spinal anesthesia in an apparently healthy patient. Vagal predominance and sympathetic blockade along with activation of baroreceptor reflexes may contribute to bradycardia during spinal anesthesia. Cerebral perfusion may be maintained by venous pooling and a partial sympathetic blockade blunting the effect of reduced cardiac output on cerebral vasoconstriction. Transient tachycardia could be caused by a startle response and the effect of slow, deep respirations on RSA magnitude, allowing sufficient spontaneous cardiac activity to circulate the resuscitative drugs. Consciousness may be maintained even after asystole has occurred, rendering insufficiently monitored patients at high risk.

Consent

This case report is in accordance with institutional IRB-guidelines. Despite several attempts, contacting the patient was impossible so we have been unable to obtain explicit written consent.

Author Contributions

All authors were involved in the draft and revision of the manuscript and have agreed to its final content.

Competing Interests

None of the authors have a relevant competing interest.

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Bernard Wittels

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The authors describe one 15-second and one 30-second episode of transient asystole after spinal anesthesia for cesarean delivery in a woman with a baseline heart rate of 62 and an intravenous preload of 1 liter of crystalloid over an undefined period of time. In their manuscript, they note correctly that bradycardia is a common dysrhythmia with spinal anesthesia and that asystole and sudden death are relatively rare. Much of the article centers on the purported physiologic mechanisms involved, yet there are no data in this case to support any specific argument.

In their manuscript title, the authors imply that their patient's persistent consciousness throughout two transient episodes of asystole is unusual or unique, but most clinicians would view this as normal in a young, healthy individual; in their manuscript, they describe treatment with IV atropine, IV epinephrine, and shouting at the patient, but this omits airway evaluation, oxygenation, ventilation and ventricular pacing that are BLS and ACLS measures to treat asystole in the non-obstetric patient.

The authors mention the Bezold-Jarisch reflex only once in the manuscript as a possible contributing influence; and their clinical responses ignored this physiology. Many authors agree that even two liters of IV crystalloid may be insufficient to prevent hypotension with spinal anesthesia, so the authors should have anticipated both hypotension and hypovolemia-induced changes in cardiac physiology. Resuscitation should have included rapid IV fluid administration and raising the patient's legs to increase venous return to the heart without increasing the cephalad spread of the spinal anesthetic. As in any resuscitation, communication with the patient ("Annie, Annie, are you OK?"), providing 100% oxygen by face mask, assisting with ventilation if necessary, and frequently checking the blood pressure (the ABC's) should have been performed. There is no role for shouting or frightening the patient who has severe bradycardia or transient asystole. In fact, sudden frightening has been known to induce a transient myocardial stunning and LV dysfunction in young, healthy individuals. By not explicitly doing maneuvers to increase venous return to the heart, the anesthesiologists delayed the patient's recovery and her response to IV atropine and epinephrine.

Most obstetric anesthesiologists know to prevent these types of episodes by administering IV fluids in amounts of 2 liters or more prior to spinal anesthesia, as well as administering pressor agents (ephedrine or phenylephrine) at the earliest sign of hypotension or bradycardia. Continuous calm communication, especially to distressed obstetric patients is an art all obstetric anesthesiologists should master. Consciousness in unmedicated, young, healthy patients without any peripheral vascular disease has no clearly defined relationship to heart rate alone, so that awareness can only be assessed by direct communication.

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

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Although not a scientific article, this is a well written and interesting case report of an exceptionally rare event. Although there is nothing special about management it is of educational interest due to the incompletely explained physiological events and clinical presentation and course.

Did the patient have a history of syncope? It would have been very helpful to have captured the ECG sequence and to have reported on the final block height (was this checked again?), as very high spinal block remains a strong possible explanation. The case report does not mention when the inspiratory effort, that is later discussed, was made. The decision to give epinephrine prior to preparing to instituting external cardiac massage, when the second episode of asystole occurred, warrants mention given it is contrary to guidelines. Clearly consciousness can be maintained with minimal or episodic cardiac output, and has been described during CPR ([Bihari S, Rajavee V. Neurocrit Care 2008](#)).

In the conclusion, reference is made to a transient tachycardia in the patient – but this would be more accurately described as transient myocardial conduction or activity or similar.

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of

expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

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John Pollard

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I believe that this report should be Approved with Reservations. One concern is the assumption that this patient did not have relatively high levels of spinal blockade. The authors describe a 'T4 level block' 3 minutes after initiating spinal anesthesia. Since the block would be expected to continue to spread upwardly after the initial 3 minutes, it is very likely that this patient had higher levels of sensory blockade after the 3 minute mark. Since the effective dose is significantly decreased in pregnancy, the authors should remind the reader that 12 mg of bupivacaine was a relatively large dose for a short (5 ft 5 inch tall) woman undergoing cesarean section.

A second concern is the speculation the discussion based primarily on physiologic studies that either had no sympathetic blockade or in one case unilateral sympathetic blockade. A spinal block that caused a T4 sensory block in 3 minutes would be expected to cause a near if not total sympathetic block in the ensuing minutes so physiologic studies following complete sympathetic blockade would likely be applicable to this case. Despite these reservations, the authors do highlight a very important point. Physicians and nurses are often falsely reassured by a patient who is still awake or even talking despite severe decreases in heart rate or blood pressure. In such cases, there appears to be enough vasoconstriction from endogenous adrenaline or vasopressin to shunt blood flow to the heart and brain despite the low cardiac output. This case demonstrates that in the supine position, the cardiac output needed to maintain consciousness is far less than what is needed to provide adequate perfusion to all of the other key organs. Treating hemodynamically unstable patients aggressively with fluids, atropine and vasopressors while they still conscious improves the likelihood that resuscitation medications will enter the central circulation more expeditiously and should result in better outcomes as observed in this case.

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

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