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CLINICAL REPORT

# Cardiac arrest after spinal anesthesia in a patient with neurally mediated syncope

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**Abstract** We present the case of cardiac arrest in a patient with neurally mediated syncope (NMS). A 66-year-old male patient was scheduled to undergo right inguinal hernioplasty. He had a history of syncope, which occurred a few times a year in childhood and once a year recently. One minute after the second spinal injection, cardiac arrest (asystole) developed. Sinus rhythm was restored by cardiac massage and intravenous administration of atropine and ephedrine. The operation was cancelled. The patient was diagnosed as NMS by a cardiologist. Four months later, right inguinal hernioplasty was performed, uneventfully, under general anesthesia. High sympathetic blockade due to spinal anesthesia and transient withdrawal of sympathetic tone and increase in vagal discharge due to NMS could be the main causes of the cardiac arrest. If the patient has any possibility of NMS, anesthesiologists should consider the possibility of cardiac arrest after spinal anesthesia.

**Keywords** Cardiac arrest · Spinal anesthesia · Neutrally mediated syncope

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## Introduction

Neurally mediated syncope (NMS) is a sudden and transient loss of consciousness, hypotension, and bradycardia caused by disorder of autonomic regulation [1]. Other terms have been used to describe this type of syncope including vasovagal syncope and neurocardiogenic syncope [2, 3]. Because the incidence of NMS is reported to be 3–3.5% [3], we sometimes encounter surgical patients with this disorder. However, severe complications such as cardiac arrest associated with NMS could be rare [2]. We present a case of cardiac arrest after spinal anesthesia in a patient with NMS.

## Case report

A 66-year-old male patient (165.5 cm, 55 kg) was scheduled to undergo right inguinal hernioplasty. He had a history of syncope, which occurred a few times a year in the childhood and once a year recently. He also had rheumatoid arthritis, and his medications for this included prednisolone 1 mg daily, mizoribine 50 mg three times daily, and methotrexate 2 mg daily. Preoperative assessments, including echocardiography and 24-h Holter electrocardiogram were normal.

The patient received no premedication. On arrival in the operating room, electrocardiogram (ECG), an automated blood pressure cuff, and oxygen saturation monitor were applied. Before spinal anesthesia, his blood pressure was 130/70 mmHg and heart rate 70 beats/min (Fig. 1). With the patient in the lateral position, a 25-gauge Quincke spinal needle was inserted into the subarachnoid space at the L3/4 interspace and 12 mg hyperbaric bupivacaine and 10 μg fentanyl were administered. Blood pressure temporarily

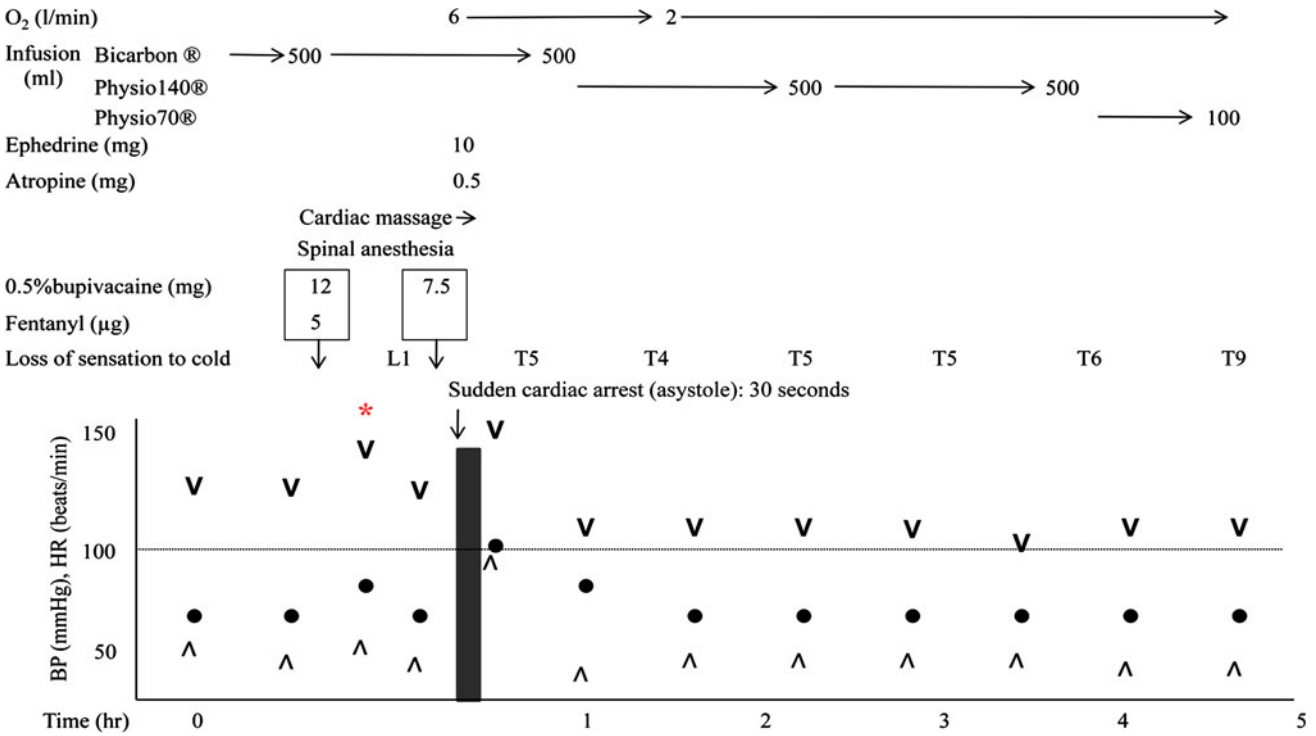


**Fig. 1** Anesthetic chart. Blood pressure and heart rate temporarily increased after the first spinal injection (*asterisk*). Then, sudden cardiac arrest (asystole) developed and the patient lost consciousness 1 min after the second spinal injection. This biphasic hemodynamic change was compatible with neurally mediated syncope. Cardiac massage was initiated and atropine 0.5 mg and ephedrine 10 mg were administered. Approximately 30 s after the start of cardiac arrest, sinus rhythm restored and the patient recovered consciousness. *BP* blood pressure, *HR* heart rate, *v symbols* systolic blood pressure, *inverted v symbols* diastolic blood pressure, *filled circles* heart rate

increased from 125/70 to 140/100 mmHg and heart rate slightly increased from 80 to 90 beats/min 5 min after the injection. Sensory anesthesia to cold had reached only L1 10 min after the spinal injection. The lumbar puncture was repeated and 7.5 mg hyperbaric bupivacaine was injected at the L3/4 interspace through a 25-gauge Quincke needle. After the spinal injection, the patient returned to supine position. One minute after the spinal injection, sudden cardiac arrest (asystole) developed and the patient lost consciousness. Cardiac massage was initiated and atropine 0.5 mg and ephedrine 10 mg were administered. Approximately 30 s after the start of cardiac arrest, sinus rhythm was restored and the patient recovered consciousness with blood pressure of 150/100 mmHg and heart rate of 100 beats/min. Sensory block level extended to T5. Maximum sensory block level of T4 was observed 40 min after the last spinal injection. After the event, the patient was hemodynamically stable and cardiac arrest did not recur. The operation was cancelled and the patient was transferred to the ward. Cardiologists were consulted and they ruled out autonomic nerve disorders such as severe DM, Shy–Drager disease, etc., by means of a thorough medical history, careful physical examination, and laboratory data analysis. They also excluded cardiac disorders such as coronary artery disease, sick sinus syndrome, and arrhythmias by coronary angiography, echocardiography, and 24-h Holter electrocardiography. They then performed a head-up tilt-table test and found characteristic biphasic hemodynamic reaction. They diagnosed his illness as NMS.

Four months later, right inguinal hernioplasty was scheduled again. Before surgery, a temporary pacemaker was implanted. In the operating room, continuous ECG, an automated blood pressure cuff, and oxygen saturation were instituted and his left radial artery was cannulated for invasive blood pressure monitoring. Anesthesia was induced by propofol, using target-controlled infusion with a plasma concentration of 3 μg/ml, and a continuous intravenous infusion of remifentanil 0.3 μg/kg/min, and was maintained by propofol 2.0–2.6 μg/ml, remifentanil 0.15–0.3 μg/kg/min, and intermittent bolus injection of fentanyl 50 μg. After induction of anesthesia, invasive blood pressure decreased from 120/70 to 70/30 mmHg and heart rate decreased from 70 to 55 beats/min. Ephedrine 10 mg was effective and blood pressure and heart rate increased to 100/70 mmHg and 75 beats/min, respectively. After the cardiovascular event, dopamine 3 μg/kg/min was given, and blood pressure and heart rate were stable during anesthesia. The operation was performed uneventfully. Pacing was not necessary and there were no complications after the operation.

## Discussion

Risk factors for bradycardia during spinal anesthesia have been reported as follows: baseline heart rate <60 beats/min, ASA physical status I, use of beta-blockers, sensory level above T6, age <50 years, prolonged PR interval [4]. When two or more of these factors are present, the patient may be considered high-risk for bradycardia and cardiac arrest during spinal anesthesia [4]. In our case, only sensory block level above T6 was presented. In addition, worsening bradycardia has often preceded the onset of cardiac arrest during spinal anesthesia [4]. Nevertheless, bradycardia was not observed and cardiac arrest occurred suddenly. It is unlikely that spinal anesthesia was the only cause of cardiac arrest. In NMS, one of the triggers of syncope is blood displacement in the lower extremities [1]. Fluid shift in the lower extremities induces reduced venous return that results in a variety of reduced cardiac output. Then sympathetic nerves are activated and blood pressure and heart rate increase. Cardiac mechanoreceptors are stimulated followed by transient withdrawal of sympathetic tone and transient increase in vagal discharge. As a result, severe bradycardia or asystole and syncope occur. This characteristic bi-phasic change of blood pressure and heart rate is a feature of NMS [1]. In this case, first spinal anesthesia may have caused fluid shift in the lower extremities that may have triggered the NMS mechanism. Blood pressure and heart rate increased after the first spinal anesthesia. Then cardiac arrest occurred after the second spinal anesthesia. This biphasic hemodynamic change was consistent with NMS. After the second spinal anesthesia, transient withdrawal of sympathetic tone and transient increase in vagal discharge due to NMS may have developed. Simultaneously, complete blockade of cardiac accelerator fibers due to spinal anesthesia could have occurred, because sympathetic blockade level is often 2–6 levels higher than sensory blockade. Combination of the NMS mechanism and spinal anesthesia-induced sympathetic blockade should be the main cause of the cardiac arrest in this case. Spinal anesthesia should be avoided in patients with NMS, because it may be likely to aggravate the risk of cardiovascular instability caused by NMS.

In the second general anesthesia, we used propofol, remifentanil, and fentanyl, however, hypotension and mild bradycardia occurred after induction. It has been reported that asystole occurred after propofol and fentanyl administration in an anxious patient [5]. Propofol causes reduction in sympathetic activity that leads to a decrease in arterial blood pressure [6]. On the other hand, fentanyl and remifentanil induce bradycardia which is primarily mediated by stimulation of the central vagal nucleus [7]. Therefore, use of propofol and fentanyl and/or remifentanil induces inhibition of sympathetic nerve activity and activation of parasympathetic nerve activity. Because sympathetic withdrawal and increased parasympathetic output are involved in the development of NMS, a combination of propofol and opioid analgesics may not be suitable for patients with NMS. Volatile anesthetics, for example sevoflurane and isoflurane, also depress sympathetic activity [8]. However, they attenuate parasympathetic nervous system function [9]. Sympathetic and parasympathetic nervous activity seem to be equally depressed during sevoflurane and isoflurane anesthesia. We should have used sevoflurane or isoflurane instead of propofol in this case.

Some studies have suggested that pacemaker therapy is not effective for NMS patients [10]. In contrast, other studies have supported cardiac pacing as effective therapy for NMS [11]. Pacing therapy for prevention of syncope could be controversial. However, a pacemaker should be effective for cardiac arrest during anesthesia. Therefore, we implanted a temporary pacemaker before the second surgery, although its use was not necessary during the second operation.

In NMS, bradycardia and vasodilation occur due to parasympathetic activation and sympathetic withdrawal [1]. Therefore, atropine alone may not be effective for hypotension in NMS, although it is usually used for treating bradycardia. In addition to atropine, sympathomimetic drugs may be necessary. Because ephedrine has $\alpha$ and $\beta 1$ adrenergic stimulating effects, it is helpful in treating hypotension accompanied by bradycardia. Commonly, adrenaline must be given in cardiac arrest. Nevertheless, in our case cardiac rhythm was restored from asystole with external cardiac massage and ephedrine and atropine without administering adrenaline. In addition, withdrawal of sympathetic tone and increase in vagal discharge are transient in NMS [12]. Ephedrine combined with atropine may be first-line agents for NMS. After early initiation of cardiopulmonary resuscitation, adrenaline may not be necessary in asystole due to NMS.

Premedication was not given before spinal anesthesia. NMS is triggered by precipitating events such as fear, severe pain, and emotional distress [3]. The patient's nervousness might have affected the cause of cardiac arrest after the spinal anesthesia. Premedications, for example a minor tranquilizer, should have been applied in this case.

In summary, we report a case of asystole after spinal anesthesia in a patient with NMS. Spinal anesthesia could have triggered the NMS mechanism. The combination of NMS mechanism and spinal anesthesia-induced sympathetic blockade should be one of the possible causes of cardiac arrest. Spinal anesthesia should be avoided in patients with NMS. If the patient has any possibility of NMS, anesthesiologists should consider the possibility of cardiac arrest after spinal anesthesia.

## References


1. Zaqqa M, Massumi A. Neurally mediated syncope. Tex Heart Inst J. 2000;27:268–72.
2. McConachie I. Vasovagal asystole during spinal anaesthesia. Anaesthesia. 1991;46:281–2.
3. Molnar J, Somberg JC. Neurocardiogenic syncope. Cardiology. 2009;114:47–9.
4. Pollard JB. Cardiac arrest during spinal anesthesia: common mechanisms and strategies for prevention. Anesth Analg. 2001;92:252–6.
5. Guise PA. Asystole following propofol and fentanyl in an anxious patient. Anaesth Intensive Care. 1991;19:116–8.
6. Ebert TJ. Sympathetic and hemodynamic effects of moderate and deep sedation with propofol in humans. Anesthesiology. 2005;103 :20–4.
7. Fukuda K. Intravenous opioid anesthetics. In: Miller RD, editor. Miller's anesthesia. 6 ed. Philadelphia: Churchill Livingstone; 2005. p. 379–437
8. Yamazaki M, Stekiel TA, Bosnjak ZJ, Kampine JP, Stekiel WJ. Effects of volatile anesthetic agents on in situ vascular smooth muscle transmembrane potential in resistance- and capacitance-regulating blood vessels. Anesthesiology. 1998;88:1085–95.
9. Picker O, Scheeren TW, Arndt JO. Inhalation anaesthetics increase heart rate by decreasing cardiac vagal activity in dogs. Br J Anaesth. 2001;87:748–54.
10. Connolly SJ, Sheldon R, Thorpe KE, Roberts RS, Ellenbogen KA, Wilkoff BL, Morillo C, Gent M, Investigators VI. Pacemaker therapy for prevention of syncope in patients with recurrent severe vasovagal syncope: Second Vasovagal Pacemaker Study (VPS II): a randomized trial. JAMA. 2003;289:2224–9.
11. Brignole M, Sutton R. Pacing for neurally mediated syncope: is placebo powerless? Europace. 2007;9:31–3.
12. Alboni P, Brignole M, Degli Uberti EC. Is vasovagal syncope a disease? Europace. 2007;9:83–7.