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Prolonged cardiac arrest unveiled silent sick sinus syndrome during general and epidural anesthesia

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Abstract
Patients who have silent sick sinus syndrome (SSS) can show various unexpected arrhythmias during surgery. The severity of these bradyarrythmias is affected by anesthetic methods. We report a unique case of a patient with silent SSS who developed 40 s of asystole under combined general and epidural anesthesia. A 40-year-old woman with no apparent cardiac disease underwent abdominal hysterectomy. General anesthesia was induced and maintained with propofol, fentanyl, and vecuronium combined with thoracic epidural anesthesia. During surgery, severe bradycardia, triggered by peritoneal manipulation, occurred, leading to 40 s of asystole. She was diagnosed as having SSS by a postoperative 24-h Holter electrocardiogram. We propose that the possible existence of SSS should be kept in mind even in a patient who shows no abnormalities on routine preoperative examination, especially in those in whom vagomimetic anesthetic methods are used.

Key words Sick sinus syndrome · Epidural anesthesia · Cardiac arrest · Intravenous anesthetics

Introduction

Sick sinus syndrome (SSS) is an important perioperative complication which can cause various arrhythmias. In some cases clinical symptoms are shown, but in some cases symptoms are silent and develop unexpectedly. The following is a case report describing a patient with silent SSS who developed 40 s of asystole, triggered by peritoneal manipulation, under combined general and epidural anesthesia.

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Case report

A 40-year-old, 153-cm, 52-kg woman was scheduled for total abdominal hysterectomy due to uterine leiomyoma. She had previously undergone several uneventful operations, for congenital ptosis under local anesthesia, and for a brow suspension under general anesthesia, in the past 3 years. With no remarkable systemic complications except for slight anemia (serum hemoglobin, 10.5 $g \cdot dl^{-1}$) due to hypermenorrhea, she had American Society of Anesthesiologists (ASA) physical status classification II. ECG showed a normal sinus rhythm with a rate of 70 $beats \cdot min^{-1}$, PR interval of 0.126 s, and QTc interval of 0.385 s.

Her baseline heart rate in the operating room was 65 $beats \cdot min^{-1}$ in normal sinus rhythm. Atropine 0.3 mg was administered before induction, and the heart rate was increased by 15%. General anesthesia was induced with propofol, fentanyl 0.1 mg, and vecuronium 8 mg, and was maintained by the target controlled infusion of propofol and fentanyl boluses. The effect-site propofol concentration was initially set at 3.0 $\mu g \cdot ml^{-1}$ by Diprifuser infusion pump (TE-371; Terumo, Tokyo, Japan). Laryngoscope insertion and endotracheal intubation succeeded without bradycardia. A total dose of intravenous fentanyl 0.15 mg was given before skin incision. Prior to surgery, 0.75% ropivacaine 8 ml was administered through an epidural catheter, which was inserted at the T12/L1 intervertebral space. No further local anesthetics were administered into the epidural space.

During peritoneal traction, severe bradycardia developed followed by sinus arrest (Fig. 1). Atropine 0.5 mg was immediately administered intravenously and external cardiac massage was performed. The heartbeat recovered in 40 s to 70 $beats \cdot min^{-1}$, and the operative procedure was resumed. Two more episodes of bradycardia of less than 40 $beats \cdot min^{-1}$ occurred during the surgery, but were not related to any operative maneuvers. Atropine 0.5 mg increased the heart rate by 20%

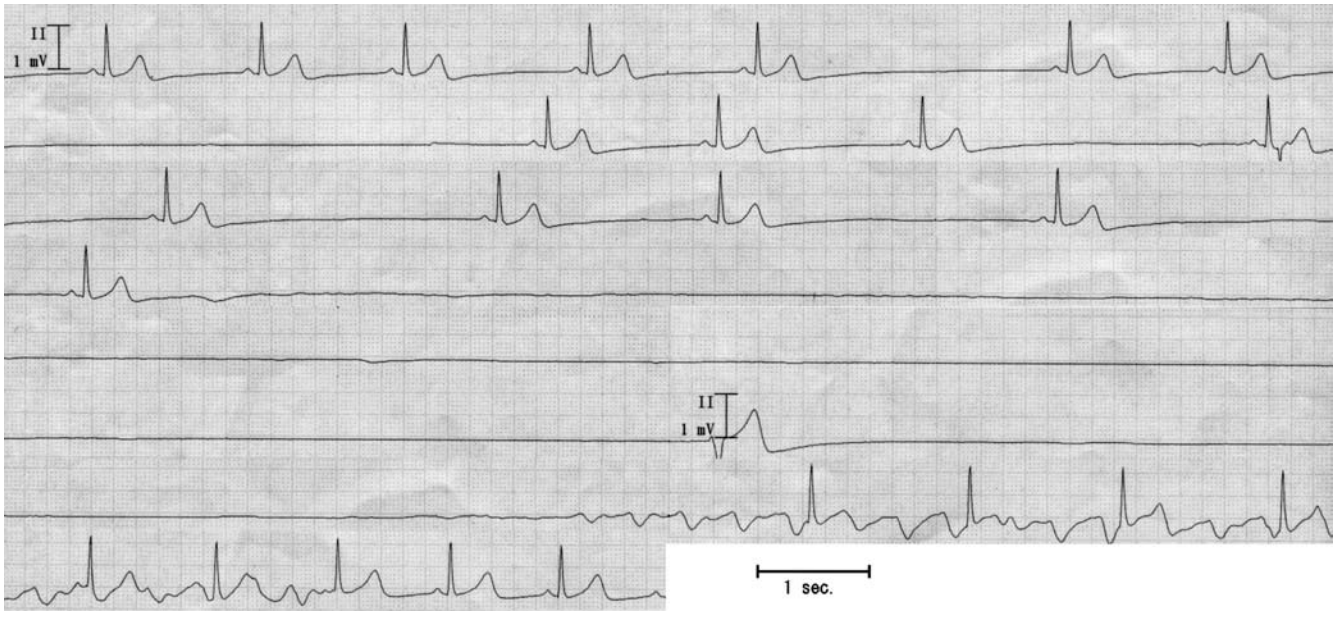

Fig. 1. Electrocardiogram during surgery showed sinus bradycardia, followed by 40-s sinus arrest. Waves due to chest compression were recorded just before recovery of the heartbeat

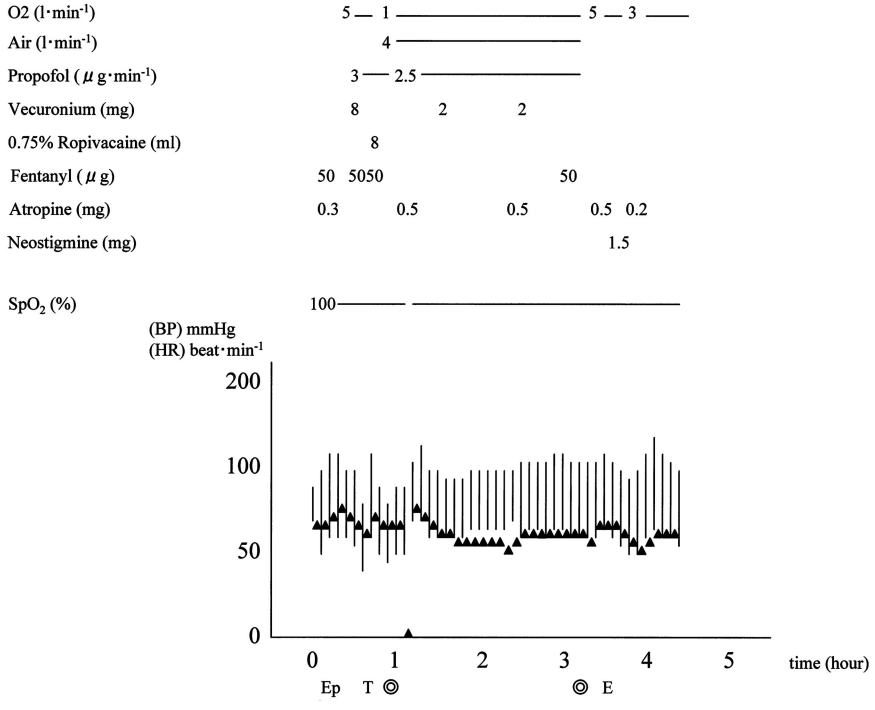

Fig. 2. Time course of anesthesia. *Upper and lower ends of solid lines* show systolic and diastolic blood pressure (*BP*), respectively. *Filled triangles* show heart rate (*HR*). *Ep* shows insertion of epidural catherer. *T* and *E* show intubation and extubation. *Double circles* indicate the beginning and end of surgery

each time. After these episodes, the effect-site propofol concentration was reduced to 2.5 μg·ml^{-1} and no additional epidural local anesthetic was administered. The heart rate was well maintained at about 60 beats·min^{-1}, even after intravenous fentanyl boluses. Systolic and diastolic blood pressure did not change markedly throughout the surgery (Fig. 2).

She emerged from anesthesia without any complications. Durations of surgery and anesthesia were 2 h 15 min and 3 h 57 min, respectively. Blood loss was 150 ml, and the infusion volume of acetate Ringer solution was 1700 ml. The total dose of atropine given during surgery was 2.0 mg. Loss of cold sensation ranged from T7 to L2 at the end of the surgery. Post-

operative 24-h Holter ECG showed a sinus arrest for 6.6 s, indicating silent SSS. She has been under the care of a cardiologist since she was discharged from our hospital. She has not received any further electrophysiological examination and permanent pacemaker implantation yet because of her age and having no apparent symptoms.

Discussion

Although many reports of silent SSS becoming apparent during surgery have been published [1–7], the present case was unique because the cardiac arrest was long-lasting and no objective information about the sinus dysfunction had been obtained preoperatively. To the best of our knowledge, there are no reports describing such a long cardiac arrest with silent SSS. Furthermore, most of the previous cases occurred under either spinal or total intravenous general anesthesia.

Bradycardia is associated with many anesthetic agents. Propofol not only resets the set point of baroreflex control of the heart rate and allows slower heart rates despite decreased arterial pressures [8], but also has a direct effect on sinus activity [9]. Fentanyl is said to have a central vagotonic effect. Vecuronium also causes bradycardia, and the incidence of bradycardia is higher if additional vagotonic agents and/or surgical vagal stimulation exist simultaneously [10,11]. Additionally, even volatile anesthetics attenuate the sinus nodal pacemaker [12]. Bradycardia also results from decreased cardiac sympathetic tone caused by high spinal anesthesia or thoracic epidural anesthesia. In the present patient, the upper level of loss of cold sensation was confirmed as T7 after the surgery, but it might have been higher during the surgery. It is possible that the combination of all these intravenous anesthetics with high epidural anesthesia and surgical stimulation caused instability of the autonomic nervous system. As a result, despite the preanesthetic atropine administration, the relatively vagotonic state made the silent sinus node dysfunction apparent, leading to long-lasting cardiac arrest.

We used atropine for the bradycardia because we did not suspect that the cardiac arrest was caused by SSS, but assumed that it was caused by excessive vagal reflex. Therefore, we used atropine for the bradycardia, reduced the dose of propofol infusion, and did not administer epidural local anesthetics, but gave fentanyl alone after the cardiac arrest. Although atropine increased the heart rate in our patient, more aggressive treatment such as isoproterenol, epinephrine, and external cardiac pacing would have been needed if atropine was not effective, as many patients with SSS did not respond to atropine administration [4–7].

A permanent pacemaker is needed if subjective symptoms are apparent. But according to the ACC/AHA (American College of Cardiology/American Heart Association) guidelines [13], asymptomatic sinus nodal dysfunction is not an indication for pacemaker implantation. The present patient does not have a permanent pacemaker, because the existence of subjective symptoms is the main factor in the need for pacemaker implantation.

Because the sinus node dysfunction of SSS progresses gradually, patients do not always complain of any symptoms in the early stage of this disease. According to several reports [14,15], there are some examinations which can unveil SSS preoperatively, such as routine preoperative 24-h Holter ECG, evaluating cardiovascular responses to beta stimulants or electrical atrial pacing [14], and evaluating the response to carotid massage [15]. But employing these examinations routinely preoperatively has several clinical problems considering cost, time, and invasiveness. The most important thing is to suspect the existence of sinus nodal dysfunction if there is severe bradycardia during anesthesia. And beta-stimulants and an external cardiac pacemaker should be always prepared for immediate use.

We emphasize that the existence of silent SSS should be kept in mind even in patients who show no abnormalities on routine preoperative examination, especially in those with general anesthesia using vagomimetic anesthetics combined with epidural anesthesia.

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