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Undiagnosed sick sinus syndrome manifest during combined general and cervical epidural anesthesia

Tadahiko Ishiyama, Kazuyuki Hanagata, Toshiaki Yamaguchi, Takashi Matsukawa, Satoshi Kashimoto, and Teruo Kumazawa

Department of Anesthesiology, Yamanashi Medical University, 1110 Shimokato, Tamaho, Nakakoma, Yamanashi 409-3898, Japan

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Introduction

Recently introduced computerized anesthesia records and electrocardiogram (ECG) facilitate clinical anesthetic management [1]. The MP-100WS (Physio-Tech, Tokyo, Japan) is a new data acquisition system that allows the performance of ECG data collection and analyses. Sick sinus syndrome (SSS) is difficult to detect preoperatively because it is sometimes asymptomatic. We report a case of sinus arrest associated with SSS and describe the significance of ECG data collection and analyses during anesthesia.

Case report

A 53-year-old, 53-kg, 155-cm woman was scheduled for the Sauvè Kapandji operation, a distal radioulnar arthrodesis with surgical creation of a pseudoarthrosis in the distal ulna, for rheumatoid arthritis of the left wrist. Her medical history was significant for rheumatoid arthritis for 10 years. She had no history of strokes, syncope, or palpitations suggestive of SSS prior to anesthesia. Her only medication was lobenzarit, a nonsteroidal anti-inflammatory drug. She had undergone the same procedure uneventfully on the other wrist under general anesthesia 1 year before. A preoperative ECG revealed sinus rhythm with a pulse rate of 72 beats·min^{-1} and incomplete right bundle branch block.

Premedication consisted of midazolam 2 mg and atropine 0.5 mg administered intramuscularly 30 min before anesthesia. In the operating room, the MP-100WS was used for ECG data collection and analyses. The patient's heart rate was 74 beats·min^{-1} in normal sinus rhythm. An epidural catheter was inserted at the C7-T1 interspace with the patient in lateral position using a midline approach with an 18-gauge Touhy needle. Sensory anesthesia was obtained from C4 to T4 10 min after the administuction of 1.5% lidocaine, 4 ml, and no hemodynamic changes were noted. General anesthesia was induced with propofol, 80 mg, and vecuronium, 8 mg, intravenously. After tracheal intubation, anesthesia was maintained with isoflurane, 0.7%–2.0%, and nitrous oxide, 67%, in oxygen. The patient was hemodynamically stable after the second (50 min after the first injection) and third (80 min after the first injection) epidural injections with 1.5% lidocaine, 4 ml. Although no hemodynamic changes occurred for 11 min after the fourth epidural injection with 1.5% lidocaine, 4 ml (approximately 2 h after the induction of anesthesia), a sudden onset of sporadic sinus arrest was noted on MP-100WS thereafter. Succeeding episodes of sinus arrest were detected by the MP-100WS. Each sinus arrest terminated spontaneously, but the duration gradually increased. Four minutes after the first sinus arrest appeared on MP-100WS, the longest episode occurred, which lasted 16 s, and cardiopulmonary resuscitation was started. Two minuets later, conversion to junctional rhythm was followed by the return of sinus rhythm at 90 beats·min^{-1} with atropine, 0.5 mg, and epinephrine, 0.1 mg. Ten minutes later, sinus arrest recurred. Cardiac rhythm was restored 2 min afterwards with atropine, 0.5 mg (Fig. 1). A temporary pacing wire was inserted via the right internal jugular vein, and ventricular pacing was initiated at 50 beats·min^{-1}. However, sinus arrest did not recur, and the heart rate stabilized at

Address correspondence to: T. Ishiyama
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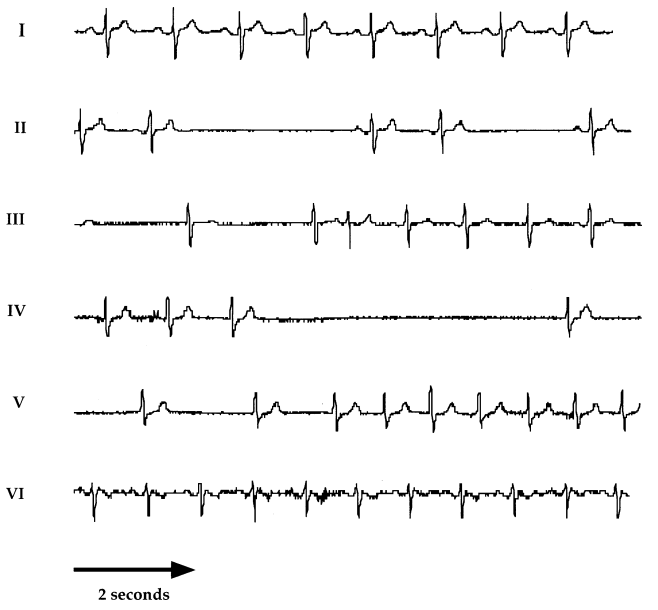

Fig. 1. Computerized continuous acquisition of electrocardiogram with MP-100WS (lead II). I, start of anesthesia; II, first sinus arrest; III, return to atrioventricular junctional rhythm; IV, second sinus arrest; V, return to atrioventricular junctional rhythm; VI, end of anesthesia

75 beats·min^{-1} after those events. The operation time was 3 h 45 min, and the anesthesia time was 4 h 35 min. The MP-100WS indicated SSS, and the temporary pacing was continued.

The patient was extubated and was transferred to the intensive care unit. She was hemodynamically stable when she woke up. However, approximately 15 min after she fell asleep, sinus arrest recurred frequently and temporary pacing worked properly. Cardiologists were consulted and confirmed the diagnosis of SSS with 24-h Holter monitoring. A permanent pacemaker device was placed subsequently.

Discussion

In this case, the patient did not describe symptoms of SSS, and the preoperative ECG was normal except for incomplete right bundle branch block. Furthermore, she had undergone the same operation on the other wrist uneventfully under general anesthesia 1 year before. It is possible that patients with undiagnosed SSS undergo general or local anesthesia. Underwood and Glynn [2] described cardiac arrest under spinal anesthesia in a patient with SSS. SSS may therefore be a cause of cardiac arrest during anesthesia in patients who do not have a history of cardiac disease.

Because cardiac pathology was not suspected preoperatively, the primary cause of sinus arrest in our patient was ascribed to either sympathetic block by cervical epidural anesthesia or inadvertent intrathecal injection. The anesthetist assumed that sinus arrest was transient. However, the MP-100WS indicated SSS. We therefore continued temporary cardiac pacing in the postoperative period when sinus arrest recurred frequently during sleep. Anesthetists should be aware that not all bradycardias are short-term events caused by anesthetic drugs. Patients may have undiagnosed underlying heart disease that may present during anesthesia for the first time. This possibility suggests that ECG data collection and analyses during anesthesia are important in all patients who develop dysrhythmias in the intraoperative period.

It has been reported that spinal anesthesia [3,4] and epidural anesthesia [5,6] may be responsible for cardiac arrest. The balance between sympathetic and parasympathetic tone plays an important role in the control of heart rate. Preganglionic sympathetic nerve fibers are more sensitive to local anesthetic drugs than sensory nerve fibers. In this patient, who had a sensory block from C4 to T4, cardiac accelerator sympathetic fibers could have been blocked. Sinus dysfunction may then have become apparent due to reduced cardioaccelerator activity. In addition, sleep influences the balance between sympathetic and parasympathetic tone and thus may have been a factor in the episodes of sinus arrest in the intensive care unit. This argument could also apply to general anesthesia. Although we did not measure the serum concentration of lidocaine, the possibility may be small that high concentration contributed to the sinus arrest.

The inhalational anesthetics used in this patient were nitrous oxide and isoflurane. Isoflurane has been reported to reduce sinus nodal pacemaker rate [7]. Although heart rate did not change at the start of isoflurane inhalation, we could not exclude the possibility that isoflurane influenced the sinus node and contributed to the arrest.

A case of SSS that presented during combined general/epidural anesthesia is reported. Epidural anesthesia and isoflurane may have impaired sinus node function. Computerized ECG data collection and analyses are useful in the diagnosis of new arrhythmias that develop intraoperatively.

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