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LETTER TO THE EDITOR

Sudden cardiac arrest in head and neck surgery: a case report

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To the Editor:

Perioperative malignant arrhythmia, which is pathognomonic in head and neck surgery, has been reported [1]. We encountered a case of sudden cardiac arrest in head and neck surgery under general anesthesia, associated with baroreceptor reflex (BRR).

The patient was a 66-year-old woman, 152 cm tall, weighing 45 kg. She was diagnosed with left maxillary carcinoma, and scheduled for left radical neck dissection and tumor resection after preoperative chemotherapy. She had a history of hypertension, and had been taking a β-blocker (atenolol 50 mg/day) in preparation for treatment. The laboratory data and physical examination did not show any marked abnormalities.

Anesthesia was induced with 50 mg i.v. propofol and 0.25 μg/kg/min remifentanil. Neuromuscular blockade was produced with 35 mg i.v. rocuronium. Tracheal intubation was completed without difficulty and without significant variations in heart rate or blood pressure. Anesthesia was maintained with 40% oxygen, 1.5% sevoflurane, and remifentanil 0.2–0.3 μg/kg/min. Systolic blood pressure (100–130 mmHg) and heart rate (50–60 beats/min) were stable. In addition to standard monitors, continuous arterial blood pressure was monitored through an intraarterial catheter in the right radial artery, and a Bispectral Index monitor was used.

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Two hours after starting the surgery, when part of the left carotid artery was slightly pressed by surgical manipulation, the ECG showed severe bradycardia. Surgery was interrupted immediately, and 0.5 mg atropine was administered, but the ECG pattern showed asystole (Fig. 1), with loss of waveform on pulse oximetry and invasive arterial blood pressure. Chest compressions were started immediately. Systolic blood pressure increased gradually about 50 s after asystole. At 70 s after asystole, we stopped the chest compressions, the heart rate had increased to 30 beats/min, and systolic blood pressure to 60–70 mmHg. At 90 s after asystole, blood pressure, heart rate, and ECG had returned to baseline values. Because the arterial blood gas values were normal and the patient's vital signs were stable, the operation was restarted. The subsequent surgical and post-anesthetic course was uneventful.

BRR is a negative feedback reflex that regulates the circulation by decreasing the heart rate and strength of heart contractions and vasodilation throughout the peripheral circulatory system. Baroreceptors are located on the blood vessel wall of each internal carotid artery (an area known as the carotid sinus) and the wall of the aortic arch.

In our case, we consider that the cause of asystole was BRR induced by stretching of the carotid sinus during surgery, causing the maximum negative feedback to the heart; however, a very severe reflex that requires cardiopulmonary resuscitation is very uncommon. In a previous study, the incidence of BRR during radical neck dissection was about 10%, but cardiac arrest was not reported [2]. We therefore consider that the cardiac arrest in our patient was related to particular factors of perioperative management in addition to BRR.

Remifentanil is an ultra-short-acting μ-opioid receptor agonist. It was reported that remifentanil contributed to severe bradycardia and asystole during anesthesia induction

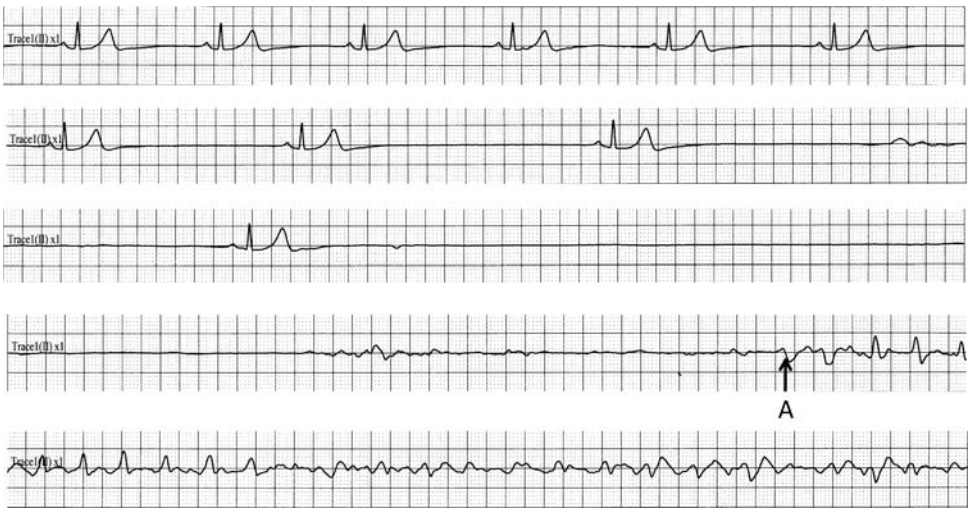

Fig. 1 Continuous electrocardiogram showing asystole in this patient; *A* indicates when chest compressions were started

[3, 4]. Hypotension and bradycardia caused by remifentanil seem to be induced by the exertion of a central vagotonic action and by stimulation of the peripheral μ-opioid receptor [5]. In previous studies, this action was reported to have been exacerbated by preexisting cardiac disease, β-blockers, and/or calcium channel blockers [3, 4]. In our patient, it was speculated that asystole was precipitated by BRR, in which central vagotonic action was exerted by remifentanil and the adrenergic blockade induced by the β-blocker that had been administered to treat hypertension.

We conclude that the cardiac arrest in our patient was induced by BRR that was likely to have been potentiated by remifentanil and a β-blocker. We suggest that anesthesiologists should be well informed about the potential of adverse reactions to these medicines, as well as being aware of cardiac reflexes during anesthesia.

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