Accelerated Idioventricular Rhythm Associated with Desflurane Administration

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Ventricular arrhythmia occurs with halothane administration (1), and may be complicated by ventricular tachycardia, especially in the presence of hypoxemia or hypercarbia (2). Sevoflurane and desflurane are volatile anesthetics used for their rapid onset and offset of action and cardiovascular stability. Sevoflurane caused fewer ventricular arrhythmias than halothane during anesthesia for dental extraction (1). Although desflurane does not facilitate ventricular arrhythmia (3), the induction of anesthesia with desflurane can result in sympathetic stimulation, which may induce cardiac arrhythmias, including ventricular arrhythmias (4,5). We report a case of accelerated idioventricular rhythm (AIVR) related to the administration of desflurane.

Case Report

A 69-yr-old woman, weighing 69 kg, was scheduled for pelvic surgery for urinary incontinence. She had previously undergone multiple surgical procedures for urinary incontinence with no anesthetic complications. A preoperative examination revealed no signs or symptoms of cardiovascular disease. The arterial blood pressure was 126/75 mm Hg. The preoperative electrocardiogram (ECG) showed a sinus rhythm at 85 bpm, a QRS duration of 0.06 s, and no ventricular ectopic beats.

After premedication with hydroxyzine 100 mg, general anesthesia was induced with sufentanil 20 μg and propofol 200 mg. Cisatracurium 8 mg was given to facilitate intubation of the trachea. The lungs of the patient were ventilated with N_2O 50% in O_2 and desflurane. Arterial blood pressure was 150/80 mm Hg, heart rate was 60 bpm, end-tidal CO_2 was 34 mm Hg, and peripheral SaO_2 by pulse oximetry was 100%. A few minutes later, at a desflurane end-tidal concentration of 5.4%, episodes of ventricular arrhythmia occurred with wide QRS complexes indicative of AIVR (ventricular rhythm at this time was 80 bpm) alternating with sinus rhythm. The arterial blood pressure was 130/70 mm Hg. Desflurane administration was stopped and AIVR disappeared spontaneously within 5 min, at a desflurane end-tidal concentration of 0.8%. At this time, the ECG was similar to the preoperative one (Fig. 1). After discontinuation of desflurane, the patient showed signs of light anesthesia, and the arterial blood pressure increased to 160/80 mm Hg, so desflurane was restarted at 3.2% end-tidal concentration without N_2O. AIVR reappeared immediately (Fig. 2). The desflurane was again discontinued, but the AIVR persisted for 5 min, until the end-tidal desflurane concentration decreased to 0%. Anesthesia was continued with a target-controlled IV infusion of propofol with a target concentration at 5 μg/mL, and 20% N_2O in O_2. The patient remained in a stable sinus rhythm during the remainder of the procedure.

Plasma troponin Ic concentrations measured at the end of surgery and 24 h later were in the normal range (0.02 μg/L). Plasma sodium, potassium, calcium, and magnesium concentrations were also normal. During the following 24-h period, continuous ECG monitoring showed no cardiac rhythm disorder. Echocardiography performed 2 days later showed no evidence of valvular heart disease or any evidence of coronary artery disease.

Discussion

During anesthesia, ventricular arrhythmias have been documented in patients with previous cardiac disease, electrolyte disturbances, or in relation to drugs given intraoperatively (2). AIVR is characterized by an ectopic ventricular rhythm with 3 or more consecutive ventricular premature beats at a rate faster than ventricular escape but slower than 100 to 125 bpm (6). The electrophysiologic characteristics of AIVR suggest a mechanism of abnormal automaticity such as enhanced phase 4 depolarization of ventricular muscle fibers, rather than reentrant arrhythmia. AIVR is usually well tolerated and no specific antiarrhythmic treatment is recommended apart from removing the suspected cause. AIVR often occurs in patients with acute myocardial infarction, especially when thrombolysis results in reperfusion (6). It has also been described in association with cocaine abuse (7). In this setting, the arrhythmia is thought to be caused by the

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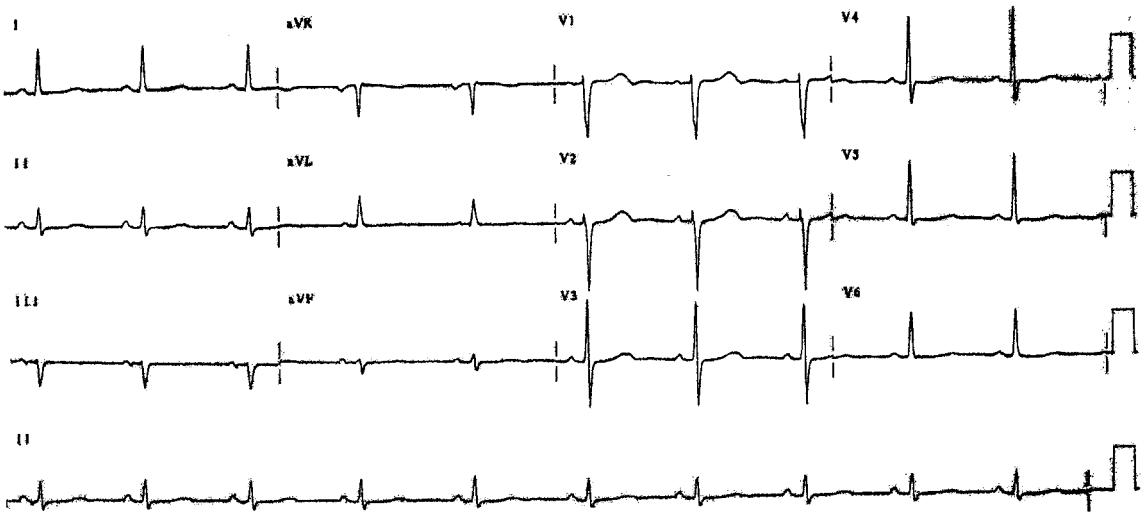

Figure 1. Perioperative electrocardiogram after the first administration of desflurane documents sinus rhythm.

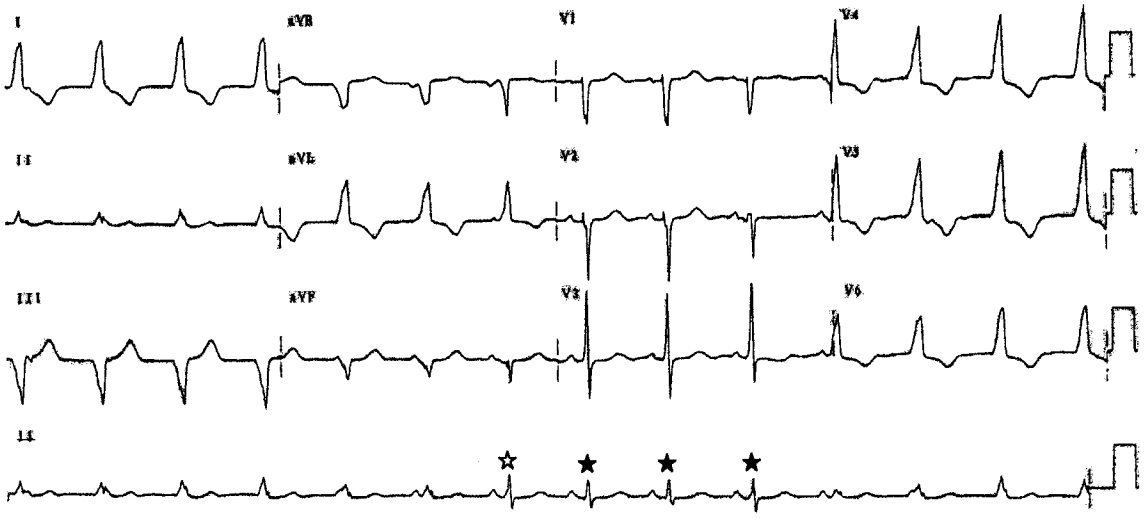

Figure 2. Perioperative electrocardiogram during the rapid desflurane readministration (end-tidal concentration: 3.2%) documenting accelerated idioventricular rhythm with a ventricular rhythm at 80 bpm. One fusion complex (white star) and three sinusal complexes (black stars) were recorded.

sympathomimetic effects of cocaine on the heart. Cocaine blocks the reuptake of norepinephrine at nerve endings. Subepicardial infusion of norepinephrine has also been reported to cause reversible ventricular arrhythmia (5). Thus, increased cardiac sympathetic activity may precipitate ventricular arrhythmia.

Volatile anesthetics, such as halothane, induce ventricular arrhythmia (1). In the presence of certain inhaled anesthetics, the dose of sympathomimetic drugs that will produce cardiac arrhythmia is markedly decreased (8). Epinephrine normally increases the rate of phase 4 depolarization in automatic cells. The incidence of catecholamine-induced dysrhythmias was most frequent with halothane than other inhaled anesthetics (9). The rapid administration of desflurane up to 1.5 minimum alveolar anesthetic concentration has been associated with systemic hypertension and tachycardia (10). This is related to a rapid increase in sympathetic activity and norepinephrine and epinephrine release (11). *In vitro* studies have also shown that desflurane may induce intramyocardial catecholamines release (12,13). Submucosal epinephrine administration induced premature ventricular contractions in patients given 1.0–1.3 minimum alveolar anesthetic concentration desflurane (3). Thus, we speculate that the two desflurane rapid sequences of administration were responsible for catecholamines release and precipitated the AIVR episodes in our patient. The second episode of AIVR started at a smaller desflurane end-tidal concentration than the

first one. At this time, the patient showed signs of awakening, which would have increased sympathetic tone. Propofol and N_2O, also administered to the patient, also increase the incidence of ventricular arrhythmia in response to epinephrine injection, and they may have contributed to the first AIVR episode (14,15). However, no episode of AIVR was noted during the remainder of the surgical procedure, when anesthesia was maintained with propofol and N_2O.

We conclude that a rapid administration of desflurane, resulting in transient hypertension and tachycardia, when a patient has a sympathetic imbalance such as a period of inadequate anesthesia, may precipitate ventricular arrhythmias such as AIVR.

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