CASE REPORT

Inhibition of Premature Ventricular Contractions by Desflurane

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THE NEED FOR ANESTHESIA support outside the traditional operating room is increasing rapidly1, including a substantial increase in the number of procedures performed in the cardiac electrophysiology (EP) laboratory. Currently, no consensus or evidence-based recommendations exist regarding the type of anesthetic or anesthetic agents and drugs used to best facilitate radiofrequency (RF) ablation of tachyarrhythmias. Additionally, little unambiguous data are available on the impact of specific anesthetics on the success of various procedures performed in the EP laboratory. Peters et al2 reported the increase of the defibrillation threshold by adding lidocaine to propofol sedation to minimize injection pain during internal cardiac defibrillator placement, which possibly could cause unnecessary revision of the lead system. The authors present a case of premature ventricular contraction (PVC) RF ablation in which the use of desflurane led to the inhibition of the arrhythmia.

CASE REPORT

A 44-year-old, 188-cm, 109-kg man without significant cardiovascular history presented to his electrophysiologist’s office with recurrent syncpe associated with palpitations. Transthoracic echocardiography showed normal left ventricular function without significant valvular abnormalities. Subsequent Holter monitoring and treadmill stress test showed frequent monomorphic PVCs without evidence of reversible ischemia. The patient was started on metoprolol, but this failed to control his arrhythmia. Before this intervention, the patient had undergone two EP studies at another institution under conscious sedation where the earliest activation was mapped to the ventricular outflow tract region. Radiofrequency ablation in both right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) had temporarily suppressed the PVCs, but the arrhythmia returned once RF energy was turned off, consistent with an intramyocardial focus. Because of his continued clinical symptoms, the patient was referred for a repeat ablation.

Preprocedural electrolyte laboratory values were all within normal limits (sodium 142 mEq/L, potassium 4.1 mEq/L, calcium 8.6 mg/dL, magnesium 1.8 mg/dL). The patient had taken his regular dose of metoprolol, 25 mg, on the morning of the procedure.

According to institutional practice for this type of procedure, no preprocedure anxiolysis with benzodiazepines was administered because the electrophysiologists at this institution believe that benzodiazepine administration may lead to suppression of the arrhythmia. In the procedure room, the patient was sedated with propofol titrated to effect for the vascular access part of the procedure (up to 100 μg/kg/min), and monitored according to American Society of Anesthesiologists (ASA) guidelines including end-tidal CO₂ measurement. No lidocaine was added to the propofol at any point during the procedure as an adjunct to decrease injection pain. Propofol was discontinued once femoral venous access was obtained and intracardiac catheters had been placed. Ten minutes after discontinuing the propofol infusion, the patient was conversant, appropriately following commands, and the neurologic status was at baseline. The electrocardiogram (ECG) showed a ventricular bigeminy with the PVC morphology of a left bundle-branch block with QRS transition in leads V2/V3 with right superior axis (Fig 1).

Electrophysiologic mapping was performed using an electroanatomic mapping system (CARTO, Biosense-Webster, Diamond Bar, CA), and the arrhythmogenic focus (earliest activation) was mapped to the lateral aspect of the RVOT just below the pulmonic valve. Before commencement of RF ablation, a continuous propofol infusion was restarted at 100 μg/kg/min to deepen sedation and to prevent patient movement and discomfort during the ablation. The patient soon presented with signs of upper airway obstruction and intermittent coughing, which interfered with the RF ablation procedure. To achieve an adequate level of sedation allowing for mapping and ablation without patient movement and to relieve the airway obstruction, general anesthesia was induced with propofol (150 mg intravenous [IV] bolus) and fentanyl (100 μg IV bolus). Vecuronium (7 mg IV bolus) was administered for neuromuscular blockade and endotracheal intubation performed. General anesthesia was maintained with desflurane (targeted end-tidal concentration 5%). A few minutes after desflurane was started, the PVCs completely disappeared and electrical stimulation failed to reproduce any PVCs (Fig 2). Desflurane was discontinued and a total intravenous anesthetic technique using a continuous propofol infusion at a rate of 125 μg/kg/min was initiated. The PVCs returned as ventricular bigeminy within a few minutes after desflurane had been discontinued. When PVCs returned, no end-tidal desflurane was detected in the breathing circuit. There were no significant changes in vital signs, including arterial blood pressure, heart rate, or arterial oxygen saturation. Depth of anesthesia monitoring using the BIS monitor (Aspect Medical Systems, Newton, MA) was also unchanged from when desflurane was used. As a proof of the theory, desflurane was again added at an end-tidal concentration of 1% (with titration of the propofol down to 90 μg/kg/min), which inhibited the arrhythmia. In accordance with the authors’ prior observation, discontinuation of desflurane (with reinstitution of the original propofol dose) then

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produced the arrhythmia once more. Given the history of failed RF ablation in both the RVOT and LVOT region, bipolar ablation was performed in this region with two ablation catheters causing temporary suppression of the arrhythmia. However, the PVCs returned once the RF energy was turned off. Given the lack of efficacy of the ablation, the procedure was terminated with the patient remaining in ventricular bigeminy. The patient was discharged home on metoprolol.

**DISCUSSION**

Providing anesthesia care in the EP laboratory requires a high level of collaboration and planning between the electrophysiologist and anesthesiologist. The diversity of cases performed, the large variety of treatment strategies available, the patient’s underlying cardiac function and comorbidities, and procedural requirements, as well as the patient’s expectations all need to be weighed in when deciding what type of anesthetic and which anesthetic drugs to choose for a specific procedure. Naturally this will vary between practitioners and institutions, and no general guidelines or recommendations have been published. General anesthesia has been associated with greater procedural success for atrial fibrillation ablations, presumably because of less patient movement during critical lesion placement and the long duration of the procedure. Ablation procedures for supraventricular tachycardias other than atrial fibrillation are often performed with light sedation or monitored anesthesia care (MAC) only. Although complicated scar-related ventricular tachycardia (VT) ablations are lengthy procedures that typically require general anesthesia, including invasive hemodynamic monitoring, PVC ablation or VTs originating in the right or left ventricular outflow tract usually are performed with minimal or no sedation. In the VT ablation setting, a thorough understanding of the type of ventricular arrhythmia is required. Unlike supraventricular tachyarrhythmias, there are no consistent anatomic conduction pathways that can be studied and the effects of the various anesthetics evaluated. Multiple etiologies of VT have been described. In general, an arrhythmogenic substrate (eg, scar tissue), modulating, and triggering factors are involved. Although the arrhythmogenic substrate cannot be changed by an anesthetic technique, the interaction of anesthetic agents with the autonomic nervous system, neuraxial sympathetic blocks, as well as sympathetic ganglion blocks, all have been clearly shown to suppress or terminate VT (triggering and modulating effects). General anesthesia alone, regardless of a specific drug, with the possible exception of ketamine, generally will reduce the sympathetic output and may impair arrhythmia induction. It is for this reason that despite conflicting data, the apparent consensus among most electrophysiologists remains that deeper sedation and general anesthesia impair arrhythmia induction and should be avoided if possible.

Only scarce and often conflicting data are available regarding specific effects of anesthetic drugs on suppressing VTs. Sevoflurane and isoflurane have been shown to prolong action potential duration and QT interval. Desflurane has been shown to prolong QT interval as well. On the other hand, propofol did not exhibit similar effects. General anesthesia and sedation with propofol, however, is often used in patients presenting in VT storm unresponsive to antiarrhythmia treatment to blunt the sympathetic output and help terminate or control the VT. Dexmedetomidine has been used to successfully treat a case of malignant VT in a child, possibly because of the significant sympatholytic properties associated with central α₂-receptor stimulation. Potent inhalation anesthetic agents have been widely used during SVT ablation procedures, apparently with minimal effect on the inducibility of supraventricular tachyarrhythmias. However, in the VT ablation setting, isoflurane was shown to suppress the ability to induce VT. Desflurane, an ether derivate closely related to isoflurane, increases sympathetic nervous system activity independently of a baroreceptor reflex–mediated effect. This sympathetic activation has been postulated to facilitate the

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**Fig 1.** Electrocardiogram tracing showing a ventricular bigeminy with the premature ventricular contraction: left bundle branch block morphology with QRS transition in leads V2/V3 with right superior axis. PR interval 190 msec, heart rate 76 beats/min, QRS 100 msec, QT 482 msec. QTc (using Bazett’s formula—QT interval divided by the square root of the preceding RR interval expressed in seconds) 544 msec.

**Fig 2.** Electrocardiogram tracing showing sinus rhythm without any premature ventricular contraction after the administration of desflurane. PR interval 184 msec, heart rate 61 beats/min, QRS 98 msec, QT 464 msec. QTc (using Bazett’s formula—QT interval divided by the square root of the preceding RR interval expressed in seconds) 468 msec.
induction of arrhythmias. In this case report, the authors describe for the first time, possible evidence that desflurane, despite its sympathomimetic properties, can suppress arrhythmias of ventricular origin. Future studies may be able to elicit a possible mechanism for this suppression. Although a number of other medications were administered to the patient, the temporal relationship of suppression of the arrhythmia and the administration of desflurane remains fascinating. Interestingly, the QT interval was shorter when desflurane was used, unlike what was reported by Yildirim and colleagues.

Studies on the effect of anesthetic agents on the inducibility or suppression of arrhythmias during ablation procedures are needed to further elaborate this observation. Although the authors cannot be absolutely sure that other factors besides desflurane did not contribute to the suppression of the arrhythmia in this patient, the presented case nevertheless greatly advances knowledge regarding the potential inhibition properties of desflurane and its effect during an outflow ventricular tachycardia RF ablation. Furthermore, because the relationship among sympathetic tone, genetics, and underlying arrhythmogenic substrate are likely to be different among patients, studies on the effects of anesthetic gases on these arrhythmias is likely to yield a large variety of results. Perhaps a registry of techniques and effects might be of greater value. In the absence of more specific data, practitioners are encouraged to tailor anesthesia technique and anesthetic agents to the individual patient’s response to facilitate tachyarrhythmia RF ablation. A high level of collaboration and planning between the electrophysiologist and the anesthesiologist is imperative for optimal care to be delivered.

REFERENCES