Non-sustained ventricular tachycardia resolving under general anesthesia: A Case Presentation

Lance S. Patak MD, MBA1; David Bradley, MD2; Bishr Haydar MD1
1Department of Anesthesiology, 2Division of Pediatric Cardiology at the University of Michigan Medical School, Ann Arbor, MI

Abstract

A 14-year-old competitive gymnast presented for a left distal femur epiphysiodesis. She had previously sustained a right femur fracture and was noted to have asymptomatic NSVT perioperatively, which improved while under general anesthesia. The patient had normal exercise testing, and her NSVT terminated under GA. She returned 11 months later for a right distal femoral osteotomy. Her baseline NSVT again improved after induction and returned five minutes after emergence.

Case Presentation

This case presents repeated temporary resolution of NSVT under GA followed by NSVT with a short-term increase in VT from baseline of 50% to 40% with normal hemodynamics. During both surgeries, the patient received lidocaine and propofol for induction, and isoflurane for maintenance anesthesia. Her NSVT decreased immediately after induction, prior to initiation of isoflurane. The etiology of this may be due to a short-term decrease in sympathetic activity. In the ETIOT, this may be due to the antiarrhythmic effect of lidocaine initially. However, the effect greatly outlasted the half-life of lidocaine. It is quite likely, as seen with various anesthesia combinations in the electrophysiology lab, that ectopy subsided primarily due to sympathetic withdrawal or vagal tone.

Discussion

As illustrated in Table 1, isoflurane itself may exhibit pro- and antiarrhythmic effects via multiple cardiac ion channel effects. Specifically, its effects on sodium channels may extend ‘recovery’ time of the channels (Figure 2), and thus increase the channels’ antiarrhythmic effect, which may have specific utility for ventricular ectopy. Other possible etiologies include changes in CNS or SNS outflow during the state of general anesthesia, neurohormonal changes resulting from anesthetic agents or the state of general anesthesia, or mechanical changes in the heart due to anesthetic-induced changes in preload, afterload, and myocardial performance. Furthermore, her NSVT is a benign ventricular tachycardia and cardioversion would not be very useful as it will resume immediately if shocked. Rather, a better treatment for such NSVT would be amiodarone. However, if the heart is normal and the tachycardia is not too fast and not polymorphic, NSVT is generally well tolerated.

Table 2. Clinical Significance of NSVT

<table>
<thead>
<tr>
<th>NSVT Type</th>
<th>Clinical Significance</th>
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<tbody>
<tr>
<td>Null</td>
<td>Polymorphic NSVT</td>
</tr>
<tr>
<td>Low</td>
<td>Asymptomatic NSVT</td>
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<tr>
<td>High</td>
<td>Symptomatic NSVT</td>
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References


Figure 1. Postop (a), Intraop (b), Postop (c) representative EKG tracings

Figure 2. Cardiac action potential (AP) and the contribution of diverse ion channels.

Table 1. Summary of the etiologic factors during anesthesia on cardiac action potential.