Effects of Volatile Anesthetics on Atrial and AV Nodal Electrophysiological Properties in Guinea Pig Isolated Perfused Heart

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Background: Knowledge of the anesthetic effects on atrial and atrioventricular (AV) nodal electrophysiologic properties is fundamental to understand the modulatory role of anesthetics on the pathogenesis of supraventricular tachyarrhythmias, and to individualize the perioperative management of patients with supraventricular tachyarrhythmias or AV nodal conduction disturbances. Therefore the authors studied the effects of three commonly used volatile anesthetics on the electrophysiologic properties of the atrium and AV node.

Methods: The concentration-dependent electrophysiologic effects of halothane, isoflurane, and desflurane (0–2 minimum alveolar concentration [MAC]) were studied in guinea pig Langendorff-perfused hearts fit with instruments to simultaneously measure atrial and AV nodal conduction times and atrial monophasic action potential duration. Atrial and AV nodal effective refractory periods were measured simultaneously using a computer-assisted premature stimulation protocol. The concentrations of anesthetics in the gas phase were monitored by an infrared gas analyzer.

Results: Volatile anesthetics caused markedly different concentration-dependent effects on atrial conduction, repolarization, and refractoriness, and on AV nodal function. At equianesthetic concentrations, halothane depressed atrial conduction the most, whereas desflurane caused the greatest shortening of atrial monophasic action potential duration. Halothane had no significant effect on atrial refractoriness, whereas at 2 MAC desflurane significantly shortened and isoflurane significantly prolonged atrial effective refractory periods by 18.1 ± 13.5% and 13.2 ± 14.7%, respectively. On an equi-MAC basis, the rank order of potency for the anesthetics to prolong AV nodal conduction time and AV nodal ERP was halothane > desflurane > isoflurane.

Conclusion: The different electrophysiologic effects of volatile anesthetics in the atrium and AV node suggest that these agents may modulate atrial dysrythmogenesis in distinctly different ways. (Key words: Anesthesia; atrial wavelength; perioperative dysrhythmias.)

SUPRAVENTRICULAR dysrhythmias such as premature atrial complexes, junctional rhythms, and atrial flutter-fibrillation have been observed in 60–80% of patients undergoing anesthesia and surgery.1,2 Although most of these dysrhythmias have only minimal hemodynamic effects, some of them (e.g., sustained atrial fibrillation with fast ventricular rate) may cause severe impairment of cardiovascular function. The electrophysiologic mechanism(s) underlying supraventricular tachyarrhythmias (SVT) include alterations in normal or abnormal automaticity, triggered activity, and reentrant excitation, or a combination of these.3,4 Although some perioperative SVTs may be caused by altered automaticity, triggered activity,5,6 or both, most sustained SVTs (e.g., atrial flutter or fibrillation) are caused by reentrant excitation.4,7

Although the results of several studies indicate that volatile anesthetics may directly or indirectly (e.g., by interacting with catecholamines) modulate cardiac automaticity and triggered activity,8–16 their effects on the substrates of reentrant SVTs are not fully understood. Electrophysiologic factors critical to the development and perpetuation of a reentrant SVT are changes in atrial conduction velocity, repolarization, and refractoriness.9–11 We have previously shown that thiopental, propofol, and ketamine modulate these parameters in distinctly different ways.12 In addition these intravenous anesthetics cause significantly different effects on fre-
frequency-dependent atrioventricular (AV) nodal conduction delay, a protective mechanism whereby the AV node regulates ventricular rate during SVTs. In the present study, the effects of three commonly used volatile anesthetics, desflurane, halothane, and isoflurane on atrial conduction time, monophasic action potential (MAP) duration, and refractoriness were measured simultaneously with AV nodal conduction time and the effective refractory period (ERP) in guinea pig isolated perfused hearts. Similar to our previous study and those of Smeejs et al. and Rensma et al., these results were used to predict the effects of volatile anesthetics on the initiation and perpetuation of reentrant SVTs and regulation of ventricular rate during atrial tachydysrhythmias.

Materials and Methods

Chemicals
Desflurane, halothane, and isoflurane were purchased from Ohmeda PPD (Liberty Corner, NJ), Halocarbon Laboratories (River Edge, NJ), and Anaquest Inc. (Liberty Corner, NJ), respectively. Desflurane, halothane, and isoflurane were delivered to the heart by bubbling the anesthetics through the perfusate solution using calibrated Tec 6, Fluotec 4, and Isotec 4 vaporizers (Ohmeda, West Yorkshire, UK), respectively. The concentrations of anesthetics in the gas phase were monitored continuously using a calibrated Ohmeda 5250 RGM (Englewood, CO) infrared gas analyzer.

Isolated Perfused Hearts
All experimental protocols were reviewed and approved by the Animal Use Committee of the University of Florida Health Sciences Center. Guinea pig hearts isolated from animals weighing 450–500 g were perfused according to the Langendorff technique, as described previously. After completion of dissection and instrumentation, the hearts were allowed to equilibrate at least for 20 min before the experiments were begun. Unless otherwise indicated, hearts were paced (3-ms pulses at twice-threshold intensity) at an atrial cycle length of 250 ms (240 beats/min) via a bipolar electrode placed on the high atrioseptal area. An analog-to-digital data acquisition board and Axotape (Axon Instruments Inc., Foster City, CA) data acquisition software (for typical examples of the recordings, see figure 1).

His Bundle Electrogram. The His bundle electrogram was recorded by placing a unipolar polytetrafluoroethylene-coated stainless steel electrode in the AV nodal area through a small right ventricular incision adjacent to the right atrium. The stimulus-to-atrium (S-A) and atrium-to-His bundle (A-H) intervals were used...
as indices of atrial and AV nodal conduction time, respectively. These intervals were measured from the digitally stored His bundle electrogram using cursor measurements in the Axotape program. If an intervention caused a high degree AV block, the longest stable A-H interval before the onset of AV block was considered the maximum dromotropic effect, and that value was used for data analysis.

**Monophasic Action Potentials.** The MAPs were recorded according to Franz using a pressure contact silver-silver chloride electrode (EP Technologies, Sunnyvale, CA) positioned and held on the surface of the left atrium using a Mitutoyo micromanipulator (Sutter Instrument Corp., Novato, CA). The atrial MAP duration at 50% (MAPD₀₅) and 90% (MAPD₀₉₀) repolarization and basic conduction time (BCT), defined as the time from the stimulus artifact to the initial upward deflection of the MAP, were determined from the digital recordings using a custom-made computer template written for Microsoft Excel, version 7.0 (Microsoft Corporation, Redmond, WA).

**Effective Refractory Periods.** Atrial and AV nodal ERPs were measured simultaneously using the following premature stimulus protocol. After a train of 15 stimuli (S₁) at a basic cycle length of 250 ms, a single premature stimulus (S₂) was introduced. The coupling interval (S₁S₂) between the last S₁ and the test stimulus (S₂) was progressively shortened in 3-ms steps after every train of stimuli. The longest S₁S₂ and A₁A₂ interval for a stimulus that failed to produce an atrial and His bundle response was defined as atrial and AV nodal ERPs, respectively. If an anesthetic caused second-degree AV block during baseline stimulation, the basic cycle length (atrial cycle length = 250 ms) was considered the ERP. Some electrophysiologically complex antiarrhythmic agents (e.g., amiodarone) exert their "class III antiarrhythmic actions" by both voltage- and time-dependent mechanisms.

Therefore, to determine whether volatile anesthetics cause any postrepolarization refractoriness, we calculated the atrial ERP:MAPD₀₉₀ ratios.

**Pharmacological and Pacing Protocols**

The effects of volatile anesthetics on atrial conduction (S-A interval, BCT) and repolarization (MAPD₀₅, MAPD₀₉₀), AV nodal conduction (A-H interval), and atrial and AV nodal ERP were examined for a concentration range of 0.2 minimum alveolar concentration (MAC). The concentrations of the anesthetics were normalized to the guinea pig MAC values of approximately 6.4% for desflurane and 1.15% and 1.01% for isoflurane and halothane, respectively. After baseline electrophysiologic parameters were measured, the anesthetics were administered at successively higher concentrations (1 and 2 MAC) until steady state was reached (i.e., approximately 20 min) and the measurements were repeated. Thereafter, the perfusion was returned to a drug-free medium, and washout values were measured 30 min later.

**Data Analysis**

All measurements are reported as mean ± SD. All statistical tests were performed on the raw, untransformed data using SPSS version 7.5 (SPSS, Chicago, IL). Before parametric statistical testing, the normal distribution of the experimental data was validated using the Kolmogorov-Smirnov test with Lilliefors' correction. Differences among means were tested by two-way repeated measures analysis of variance with one-way replication followed by Student-Newman Keuls testing. *P* < 0.05 was considered significant.

**Results**

Table 1 shows the baseline values for all the electrophysiologic measurements. Subsequent data here in the Results section are expressed as the percentage change from the baseline value (mean ± SD).

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**Table 1. Baseline Values of the Electrophysiologic Measurements in Guinea Pig Isolated Perfused Heart**

<table>
<thead>
<tr>
<th></th>
<th>Desflurane (n = 6)</th>
<th>Halothane (n = 7)</th>
<th>Isoflurane (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HBE measurements</td>
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<tr>
<td>S-A interval (ms)</td>
<td>10.1 ± 1.5</td>
<td>9.6 ± 1.6</td>
<td>9.4 ± 1.9</td>
</tr>
<tr>
<td>A-H interval (ms)</td>
<td>51.7 ± 5.0</td>
<td>50.2 ± 6.5</td>
<td>46.7 ± 5.5</td>
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<tr>
<td>Atrial MAP measurements</td>
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<tr>
<td>BCT (ms)</td>
<td>21.2 ± 3.9</td>
<td>18.6 ± 3.2</td>
<td>18.6 ± 2.1</td>
</tr>
<tr>
<td>MAPD₀₅ (ms)</td>
<td>42.7 ± 5.6</td>
<td>47.7 ± 4.4</td>
<td>42.6 ± 4.8</td>
</tr>
<tr>
<td>MAPD₀₉₀ (ms)</td>
<td>67.2 ± 6.9</td>
<td>72.0 ± 7.1</td>
<td>67.1 ± 7.3</td>
</tr>
<tr>
<td>Refractory periods</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrial ERP (ms)</td>
<td>77.1 ± 9.1</td>
<td>70.7 ± 4.5</td>
<td>78.7 ± 6.7</td>
</tr>
<tr>
<td>AV nodal ERP (ms)</td>
<td>137.7 ± 7.1</td>
<td>137.1 ± 8.1</td>
<td>133.0 ± 6.6</td>
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</tbody>
</table>

Values are mean ± SD for the number of experiments in parentheses. There were no statistically significant differences between the groups.

HBE = His bundle electrogram; MAP = monophasic action potential; MAPD₀₅ = atrial monophasic action potential duration at 50% repolarization; MAPD₀₉₀ = monophasic action potential duration at 90% repolarization; ERP = effective refractory period.

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Effects of Volatile Anesthetics on Atrial Conduction Time

Figure 2 shows the effects of the volatile anesthetics on atrial conduction times. None of the anesthetics caused significant changes in the S-A interval or BCT at 1 MAC. However, at higher concentrations (2 MAC), halothane (n = 7) significantly prolonged the S-A interval and BCT by 24.3 ± 18.4% and 20.7 ± 15.7%, respectively. Isoflurane (n = 7) also prolonged the S-A interval (14.7 ± 16.7%, P < 0.05) and tended to increase the BCT (9 ± 16.6%) at 2 MAC. In contrast, desflurane (n = 6) had no significant effects on atrial conduction times. Thus, on an equi-MAC basis, the rank order of potency for the volatile anesthetics to depress atrial conduction was halothane > isoflurane > desflurane. The anesthetic-induced prolongation of the S-A interval and BCT was completely reversed during the 30-min washout period in all groups.

Effects of Volatile Anesthetics on Atrial Repolarization and Refractoriness

The volatile anesthetics caused significantly different effects on early atrial repolarization (MAPD50) but interestingly did not significantly influence late repolarization (MAPD90, fig. 3). Desflurane and halothane caused

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Fig. 2. Effects of volatile anesthetics on atrial conduction times. Shown are concentration-dependent effects of halothane, isoflurane, and desflurane on (A) the stimulus-to-atrium interval and (B) basic conduction time of the atrial monophasic action potential. Each bar represents a mean ± SD of seven experiments for the halothane and isoflurane groups and of six experiments for the desflurane group. Significant changes (P < 0.05) are indicated: *control versus 1 or 2 minimum alveolar concentration (MAC) for a given anesthetic; †halothane or isoflurane versus desflurane at a given MAC; ‡halothane versus isoflurane at a given MAC; and §1 versus 2 MAC for a given anesthetic.

Fig. 3. Effects of volatile anesthetics on atrial monophasic action potential duration. Shown are concentration-dependent effects of halothane, isoflurane, and desflurane on atrial monophasic action potential duration at (A) 50% (MAPD50) and (B) 90% repolarization level (MAPD90). Each bar represents a mean ± SD of seven experiments for the halothane and isoflurane groups and of six experiments for the desflurane group. Significant changes (P < 0.05) are indicated: *control versus 1 or 2 minimum alveolar concentration (MAC) for a given anesthetic; †halothane or isoflurane versus desflurane at a given MAC; ‡halothane versus isoflurane at a given MAC; and §1 versus 2 MAC for a given anesthetic.
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Fig. 4. The effects of volatile anesthetics on the atrial effective refractory period (ERP). Shown are concentration-dependent effects of halothane, isoflurane, and desflurane on atrial ERP. Each bar represents a mean ± SD of seven experiments for the halothane and isoflurane groups and of six experiments for the desflurane group. Significant changes ($P < 0.05$) are indicated: *control versus 1 or 2 minimum alveolar concentration (MAC) for a given anesthetic; †halothane or isoflurane versus desflurane at a given MAC; ‡halothane versus isoflurane at a given MAC; §1 versus 2 MAC for a given anesthetic.

concentration-dependent shortening of atrial APD, whereas isoflurane had no effect. Desflurane ($n = 6$) and halothane ($n = 7$) shortened MAPD$_{50}$ values at 1 MAC by 19.1 ± 9.5% and 15.2 ± 6% and at 2 MAC by 28.6 ± 11.2% and 26.6 ± 11.7%, respectively. Isoflurane had no significant effect on MAPD$_{50}$. In contrast, none of the anesthetics significantly altered MAPD$_{90}$. Unlike halothane and desflurane, isoflurane did not significantly shorten MAPD$_{50}$ or MAPD$_{90}$. In all anesthetic groups, no significant differences between baseline and washout values of MAPD$_{50}$ and MAPD$_{90}$ were found.

The anesthetics also caused significantly different effects on atrial refractoriness. As shown in figure 4, halothane had no significant effect, whereas desflurane and isoflurane caused a concentration-dependent shortening and prolongation of atrial ERP, respectively. Specifically, at 1 MAC none of the anesthetics caused significant changes in atrial ERP. At 2 MAC, halothane ($n = 7$) had no significant effect, whereas desflurane ($n = 6$) significantly shortened atrial ERP by 18.1 ± 13.5%, and isoflurane ($n = 7$) significantly prolonged atrial ERP by 13.2 ± 14.7%. When these changes are compared with those in atrial MAPD$_{50}$, it is apparent that isoflurane, unlike halothane and desflurane, may have caused some postre polarization refractoriness. That is, the changes in terminal APD predicted well the effects of halothane and desflurane on atrial ERP, whereas isoflurane caused significant prolongation of atrial ERP, although it did not significantly affect atrial MAPD$_{90}$. At 2 MAC, isoflurane tended to increase the ERP:MAPD$_{50}$ ratio (from $1.16 ± 0.09$ to $1.34 ± 0.08$, $P = 0.23$). There were no significant differences between the baseline and washout values of atrial MAPD$_{50}$, MAPD$_{90}$, and ERP in any of the anesthetic groups.

Effects of Volatile Anesthetics on Atrioventricular Nodal Conduction

In guinea pig hearts paced at an atrial cycle length of 250 ms, all three anesthetics prolonged AV nodal conduction time in a concentration-dependent manner (fig. 5). At 1 MAC, desflurane ($n = 6$) and halothane ($n = 7$) significantly prolonged the A-H interval by 17.3 ± 6.1% and 20.9 ± 12.9%, respectively, whereas isoflurane did not significantly prolong the A-H interval. The depressant AV nodal effects of the anesthetics were sig-
Discussion

The major finding of this study was that volatile anesthetics exerted significantly different effects on the electrophysiologic properties of the atrium and AV node in guinea pig isolated perfused hearts. These results should enhance our understanding of the effects of volatile anesthetics on reentrant SVTs and may also help to explain at least some of the differences in the pro- and antidysrhythmic actions of volatile anesthetics.\textsuperscript{18,19}

Atrial Effects of Volatile Anesthetics

Although limited data on the effects of volatile anesthetics on atrial conduction time are available, the results of many earlier studies support our findings. The observations that halothane and isoflurane depress conduction (and contractility) are in keeping with previous results in Purkinje fibers, papillary muscles, and ventricular preparations\textsuperscript{20–24} whereas the failure of 2 MAC desflurane to prolong atrial conduction intervals is a new finding. Similarly, in general our MAP data are consistent with previous intracellular action potential recordings in various cardiac tissues.\textsuperscript{25,26} For example, similar to our results, Gallagher et al.\textsuperscript{27} found that halothane caused differential effects in canine Purkinje fibers on the plateau and terminal phases of the action potential (i.e., it decreased APD\textsubscript{50} and slightly lengthened APD\textsubscript{90}).

Although most sustained and paroxysmal SVTs are caused by reentrant excitation,\textsuperscript{3,4} the effects of volatile anesthetics on the electrophysiologic factors critical to the initiation and perpetuation of reentrant SVTs have remained unclear. According to Rensma et al.,\textsuperscript{10} not only shortening of atrial refractoriness but also slowing of atrial conduction velocity predispose the myocardium to the development of reentrant tachycardias. Thus, notwithstanding the authoritative work of Atlee and Yeager,\textsuperscript{28} who studied the effects of volatile anesthetics on atrial refractoriness in dogs fitted with instruments for long-term monitoring, the current results substantially improve our understanding of the effect of volatile anesthetics on the pathogenesis of reentrant atrial tachydysrhythmias.

Previously we\textsuperscript{12} found marked differences in the actions of intravenous anesthetics on atrial wavelength (i.e., $\lambda$, the product of atrial conduction velocity and ERP), a factor that accurately predicts the likelihood of developing atrial tachydysrhythmias.\textsuperscript{10} In the current study no direct measurements of atrial conduction velocity were made and the exact values of $\lambda$ were not
Table 2. Summary of the Supraventricular Effects of Volatile Anesthetics in Guinea Pig Isolated Perfused Heart

<table>
<thead>
<tr>
<th>Anesthetic</th>
<th>Conduction Time</th>
<th>ERP</th>
<th>Wavelength (λ)*</th>
<th>AV Node</th>
<th>A-H Interval</th>
<th>ERP</th>
</tr>
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<tbody>
<tr>
<td>Desflurane</td>
<td>**</td>
<td>↓</td>
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<td>1.0 MAC</td>
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<td>2.0 MAC</td>
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</tr>
<tr>
<td>Halothane</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
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<td>1.0 MAC</td>
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<td>2.0 MAC</td>
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</tr>
<tr>
<td>Isoflurane</td>
<td>↑</td>
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<td>1.0 MAC</td>
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<td>2.0 MAC</td>
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</table>

** = no change; ↑ and ↓ = tendency to increase and decrease, respectively; ↑↑ and ↓↓ = statistically significant increase and decrease, respectively. *While estimating the anesthetic-induced changes in atrial wavelength (λ), changes in conduction time were assumed to correlate negatively with conduction velocity. Atrial and AV nodal effective refractory periods (ERP) were measured as described in Materials and Methods.

calculated. Nevertheless, because changes in cardiac conduction times usually mirror those of conduction velocity (i.e., the longer the conduction time, the slower the conduction velocity), a relative comparison between the effects of volatile anesthetics on atrial wavelength can be made. None of the volatile anesthetics produced changes likely to affect λ at the lower concentration (1 MAC), whereas their significantly different effects on atrial conduction intervals and ERP suggests that they may exert disparate actions on atrial λ at 2 MAC (table 2). Accordingly, halothane and desflurane may tend to decrease λ at 2 MAC by slowing atrial conduction velocity and by shortening atrial refractoriness, respectively. Isoflurane, on the other hand, caused opposite effects on atrial conduction velocity and refractoriness, which is likely to minimize its effects on λ. In conclusion, although no atrial tachydysrythmias were induced during the experiments, the differences between the actions of the volatile anesthetics on atrial electrophysiologic properties suggest that these agents may cause distinctly different actions on the initiation and perpetuation of perioperative reentrant atrial tachydysrythmias.

Atioventricular Nodal Effects of Volatile Anesthetics

Our rank order of potency for the negative dromotropic effects of volatile anesthetics (halothane > desflurane > isoflurane) corresponds with previous findings from other laboratories. For example, Atlee et al. 29,30 found that halothane and enflurane prolonged AV nodal conduction time significantly more than did isoflurane in dogs fitted with instruments for long-term monitoring, and Nakaigaawa et al. 31 showed that halothane caused greater negative dromotropic effects than did isoflurane in pentobarbital-anesthetized dogs. Our study was the first to compare the dromotropic effects of desflurane with those of halothane and isoflurane. In keeping with their depressant effects on the AV nodal conduction (i.e., A-H interval lengthening), halothane, desflurane, and isoflurane caused concentration-dependent prolongations of AV nodal ERP. In this study, we elected to use AV nodal ERP rather than Wenckebach cycle length determinations as a measure of frequency-dependent drug effects on AV nodal conduction because AV nodal ERP correlates well with Wenckebach cycle length. 12,32 allows the simultaneous determination of atrial ERP, and is less stressful to the isolated heart preparation.

Despite the limitations of the study, these findings may have clinical implications for the anesthetic management of patients with a history of supraventricular tachydysrythmias or preexisting AV nodal conduction disturbances. Similar to antidysrhythmic agents, 33,34 anesthetics that depress AV nodal conduction in a frequency-dependent manner would effectively filter rapid supernumerary impulses during atrial tachydysrythmias. Therefore, our data suggest that halothane would be more protective against an excessive ventricular rate during atrial tachydysrythmias than would either desflurane or isoflurane. On the other hand, halothane was also the most conducive to cause high-degree AV nodal block. When assessing the clinical relevance of this observation, several important points should be consid-
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ereed. First, because of the phenomenon of frequency dependence whereby AV nodal conduction is modulated by atrial rate, a slower heart rate would significantly mitigate the AV nodal effects of volatile anesthetic in humans. Second, in unpaced animals (or humans) the AV nodal effects of the volatile anesthetic would be modulated by the chronotropic actions of the drugs. Third, all three volatile anesthetics prolonged AV nodal ERP, which suggest that their negative dromotropic effect is frequency dependent. Thus they may markedly depress conduction during tachycardias but have little or no effect on AV nodal conduction during normal heart rates. Together these findings indicate that it is unlikely that any of the contemporary volatile anesthetics would cause high-degree AV block in patients with normal heart rate and without preexisting AV nodal conduction disturbances. Consistent with this interpretation, two recent clinical studies showed that isoflurane caused minimal to no effects on AV nodal function in children undergoing routine electrophysiologic examination for Wolf-Parkinson-White syndrome. However, experimental data do indicate that interactions with other AV blocking agents (e.g., calcium channel blockers and amiodarone) may significantly augment the negative dromotropic effects of volatile anesthetics and thus increase the risk of conduction disturbances.

Limitations of the Study and Conclusions

In the present study we used guinea pig-denervated, isolated perfused hearts. Given the important anesthetic–catecholamine interactions and findings that activation of M1-muscarinic cholinergic receptors and adrenergic stimulation shortens atrial action potential and modulates AV nodal conduction, it is likely that the autonomic nervous system tone would modify the atrial and AV nodal effects of volatile anesthetics. Similarly, species-dependent differences may limit extrapolation of these results to the clinical setting. Notwithstanding, the paramount role of local changes in atrial conduction velocity, repolarization, and refractoriness in the pathogenesis of reentrant atrial tachydysrhythmias, temporal and spatial dispersion of these changes, and their alterations under pathologic conditions also may be important. Indeed, halothane increases regional differences of Purkinje fiber APD and promotes reentry in the superfused infarcted canine heart. Furthermore, no conclusions can be reached from the results of this study regarding the effects of anesthetics on SVTs initiated by mechanisms other than reentry.

Despite this, we believe that the effects of volatile anesthetics reported here may, at least to some extent, be used to predict their actions on the development and perpetuation of reentrant SVTs. Because of its counteracting effects on atrial conduction velocity and refractoriness, isoflurane may be less likely to facilitate development of reentrant atrial tachydysrhythmias than halothane and desflurane, which tend to decrease atrial wavelength. On the other hand, the more pronounced frequency-dependent negative dromotropic effects of halothane and desflurane suggest that these agents would be more protective against fast ventricular response during SVTs than isoflurane. If applicable to humans, these findings provide the anesthesiologist a more rational basis for selecting volatile anesthetics in patients with a history of SVTs or AV nodal conduction disturbances. However, before any specific recommendation can be made, randomized, prospective clinical studies need to be done to assess the effects of volatile anesthetic on atrial and AV nodal electrophysiologic properties.

References

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