INTRODUCTION: Halothane lowers the dose of epinephrine needed to induce ventricular arrhythmias in experimental animals and in patients. A previous study in dogs\(^1\) suggested that a higher concentration of halothane (2.0 MAC) appeared to sensitize the heart less to epinephrine arrhythmias than a lower concentration (1.2 MAC); however no statistical analysis was performed. Our study was designed to definitively determine whether varying halothane concentration over a clinically relevant range (0.5% to 2.0%) alters the arrhythmogenic dose of epinephrine (ADE).

METHODS: Anesthesia was induced and maintained in unpremedicated dogs with halothane in oxygen. Tracheal intubation was performed in the absence of muscle relaxants and ventilation was controlled to achieve normocapnia. Maintenance fluids consisted of 5% dextrose in 0.2% NaCl supplemented with 89 mg/L of NaHCO\(_3\) to prevent the metabolic acidosis observed with repeated epinephrine infusions. End-tidal CO\(_2\) and halothane, arterial blood pressure, and lead II of the EKG were continuously recorded.

The ADE was measured according to the method of Pace et al.\(^2\). A fresh 100 mg/mL solution of epinephrine in 5% dextrose was infused via a syringe pump for three minutes at a constant rate. If the arrhythmia threshold (4 or more ventricular ectopic beats in a 15 second period or any tachycardia producing a life-threatening drop in blood pressure) was not achieved, the animal was allowed to recover from the epinephrine infusion before a logarithmically spaced higher dose was infused. This process of progressively larger doses of epinephrine was continued until the ADE was established.

MAC for halothane in the dog is 0.9%; therefore the ADE assessment at 0.5% halothane may be affected by endogenous epinephrine release secondary to light anesthesia. Na\(_2\)O\(^3\) and barbiturates\(^4\) were avoided because both of these supplemental anesthetic agents sensitize the myocardium to epinephrine; etomidate has not previously been investigated in this regard. We sought to demonstrate that etomidate does not enhance epinephrine arrhythmogenicity so that it could be used as a supplemental agent in this study. Therefore, 10 dogs were anesthetized with halothane as described above. After the baseline ADE was determined with halothane alone, an intravenous loading dose of etomidate, 1 mg/kg, was given and followed by a continuous infusion of 4 mg/kg/hr. Blood samples taken at intervals confirmed that therapeutic levels of etomidate were achieved.\(^5\) After a steady-state of etomidate was established, the ADE was again determined. Statistical analysis by the \(t\) test for paired data revealed no significant difference in the ADE values with and without etomidate (\(p > 0.05\)).

Therefore at anesthetic concentrations of halothane, anesthesia was supplemented with an intravenous infusion of etomidate.