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Atrioventricular Conduction in Dogs during Anesthesia with Isoflurane

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The effects of 1.25, 2.0, and 2.5 MAC isoflurane on atrioventricular conduction were studied by His-bundle electrocardiography during atrial pacing in ten dogs. No effect upon atrioventricular conduction as evidenced by changes in A–H interval (the time of conduction from low right atrium to His-bundle, representing primary AV nodal conduction) was found at these concentrations. Atrial pacing to 200 beats/min did not influence the A–H interval at the three anesthetic concentrations. The stability of cardiac rhythm observed clinically with isoflurane may be related to this lack of effect upon the AV node. (Key words: Anesthetics, volatile: isoflurane. Heart: atria; conduction; arrhythmias; electrocardiography; His-bundle.)

IT IS WELL ESTABLISHED that halogenated anesthetics can alter cardiac rate and rhythm. Halothane anesthesia inconsistently produces a slight increase in heart rate in man,[1] can produce nodal rhythm and sinus arrhythmia, and consistently lowers the threshold to ventricular arrhythmias produced by exogenous and endogenous catecholamines.[2,3] Enflurane produces a marked increase in heart rate,†† and much more β-adrenergic stimulation is needed to produce ventricular arrhythmias with enflurane than with halothane.[3],‡‡ Isoflurane has been reported to increase heart rate[4] and produces a stable cardiac rhythm concurrent with epinephrine injection.[2] Transient abnormalities of rhythm have been reported to occur with isoflurane. Since alterations of cardiac conduction and automaticity may be factors in the production of cardiac arrhythmias during anesthesia,[5] we examined the effects of isoflurane upon atrioventricular (AV) conduction, utilizing His-bundle electrocardiography.[6,7]

Methods

Ten unpremedicated mongrel dogs of either sex (14 to 22 kg) were anesthetized with isoflurane§§ and oxygen, utilizing an Ohio anesthesia machine with a standard circle absorber apparatus and a Vernitrol vaporizer. A foreleg intravenous route was established with a slow infusion of dextrose, 5 per cent, in lactated Ringer's solution. Intubation of the trachea was accomplished without muscle relaxants or adjuvant drugs. No drug other than the volatile anesthetic agent was administered at any time. Ventilation was assisted or controlled to maintain end-tidal carbon dioxide between 4.5 and 5.5 per cent as measured by a calibrated Beckman infrared gas analyzer, model LB-2, sampling via a small-gauge catheter placed into the endotracheal tube. End-tidal anesthetic concentration was measured in a similar manner. Temperature (measured with an esophageal thermistor) was kept between 35.5 and 38 C by heating lamps. Anesthetized dogs were secured in the supine position and bilateral femoral venous cutdowns were performed. A tripolar catheter¶¶ was positioned under fluoroscopic control across the tricuspid valve and a stable His-bundle electrogram was obtained. A second tripolar catheter was passed via the opposite femoral vein and positioned near the junction of the superior vena cava and right atrium for atrial pacing. A Medtronic 5837 R-wave coupled pulse generator delivering a square-wave monophasic pulse of 2-msec duration was used for all atrial pacing studies.

The His-bundle catheter electrodes were connected to an Ele-cath switch box that allowed the selection of any combination of two electrodes. The output of the switch box was connected via a Burr-Brown model 8809 isolation amplifier, a model 8860 ECG control

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†† Calverley RK, Smith NT, Prys-Roberts C: Cardiovascular effects of prolonged enflurane anesthesia in man (abstr). Annual Meeting, American Society of Anesthesiologists, 1975, pp 57–58.

‡‡ Johnston RR, Eger EI: A comparative interaction of epinephrine with halothane, enflurane, and isoflurane in man (abstr). Annual Meeting, American Society of Anesthesiologists, 1974, pp 53–54.

§§ Isoflurane supplied by Ohio Medical Products.

¶¶ United States Catheter & Instrument Corporation, Billerica, Mass.

unit, and a model 8861 interface unit to an Electronics for Medicine DR-12 recorder with band-width cutoff frequencies of 50–400 Hz. Arterial (aortic root) and left ventricular pressures were recorded using a model 350 Millar-tip transducer catheter introduced via the femoral artery. The catheter was connected to the DR-12 recorder via a pressure signal conditioning unit.***

After satisfactory anesthesia had been achieved and end-tidal carbon dioxide and isoflurane concentrations had been stable for at least 15 min, baseline recordings of heart rate, blood pressure (systolic, diastolic, mean), His-bundle electrogram, and Frank X-Y-Z surface electrocardiogram were made. Three alveolar concentrations of isoflurane, 1.9, 3.0, and 3.7 per cent, corresponding to 1.25, 2.0, and 2.5 MAC,[8] respectively, were studied. After baseline recordings were obtained, the right atrium was paced to 200/min

*** Henry Koerner Research, Inc., Model 747.

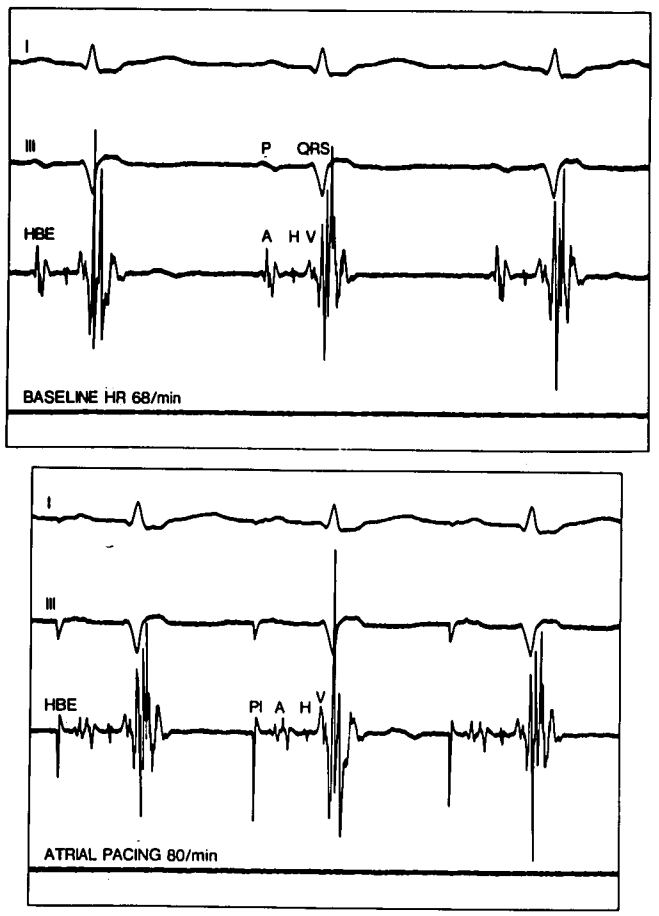

FIG. 1. Surface leads I and III and His-bundle electrogram (HBE) with (*above*) and without (*below*) atrial pacing. A = atrial firing; H = His bundle firing; V = ventricular firing; PI = pacing impulse.

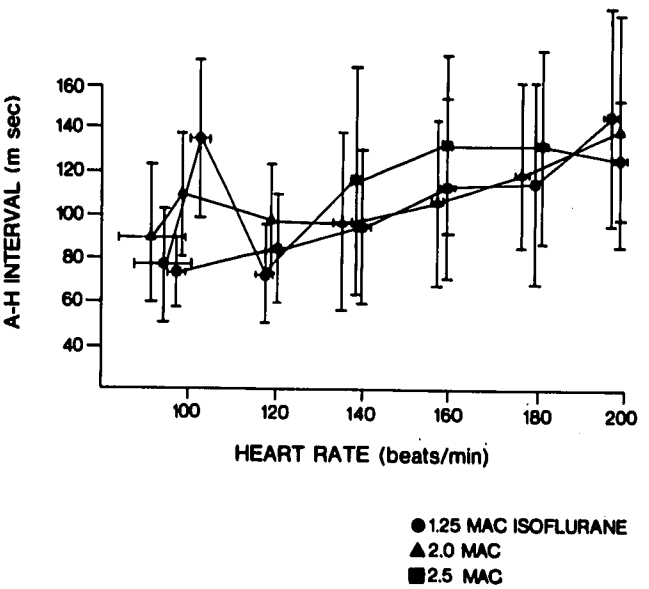

FIG. 2. A–H interval plotted against heart rate at three MAC multiples of end-tidal isoflurane. Initial points represent spontaneously beating hearts prior to the onset of atrial pacing. All values are means ± SEM.

in 20-beat/min increments above the baseline rate and recordings repeated at each paced rate. Pacing was then discontinued and the dog equilibrated with the next higher MAC value, with baseline and paced recordings being repeated as before. Each paced rate was maintained continuously for 3 min (with no evidence of loss of atrial capture) before recordings were made. At least ten consecutive beats were recorded at a paper speed of 100 mm/sec.

Three deflections are seen on a His-bundle electrogram, representing portions of AV conduction (fig. 1). The first is the atrial deflection (A-wave), reflecting low right atrial depolarization that occurs during the surface P wave. The His-bundle spike (H) is seen next, and is produced by rapid passage of the impulse through this structure. Last, the ventricular (V) deflection is produced by ventricular myocardial depolarization. When atrial pacing is being used a pacing impulse (PI) deflection is seen just preceding the A-wave. Two intervals were investigated in this study. The first was the A–H interval, the time of conduction from low right atrium to His-bundle, representing primary AV nodal conduction. The second was the H–V interval, representing conduction time from depolarization of the His bundle to the beginning of myocardial firing. These intervals were measured and plotted against increase in heart rate at each MAC level to compare the effects of isoflurane on AV conduction. All data were analyzed by an analysis of variance.

Results

There was no effect on A–H interval with the three anesthetic concentrations studied, nor was any effect of atrial pacing demonstrated at any anesthetic concentration (fig. 2). No change in the H–V interval was found in any of the studies. H–V intervals (in msec) were 25.8 ± 5.1 (SD), 24.5 ± 4.8, and 24.5 ± 5.0, at 1.25, 2.0, and 2.5 MAC isoflurane, respectively. A dose-related depression of mean arterial blood pressure by isoflurane was observed. At end-tidal isoflurane concentrations of 1.25, 2.0, and 2.5 MAC, mean arterial blood pressures were 91 ± 17 55 ± 16, and 53 ± 17 torr, respectively. Arterial blood-gas values for O_2, CO_2 and pH were 315–510 torr, 30–39 torr, and 7.38–7.51, respectively.

Discussion

The His-bundle electrocardiogram allows subdivision of the P–R interval into two components.[9,10] Conduction time between the atrial depolarization potential and the His-bundle deflection (A–H interval) primarily represents impulse propagation in the region of the AV node. Conduction to the distal bundle of His and Purkinje network (H–V interval) is measured from the His deflection to the beginning of the QRS complex. Atlee and Rusy described a dose-dependent depressant effect of halothane on AV conduction.[11,12] Additionally, atrial pacing has been shown to progressively lengthen the A–H interval in dogs and in man during halothane anesthesia.[11–13] Atlee has also shown that enflurane prolongs AV nodal conduction time in both the spontaneously beating and the incrementally paced dog heart.[14]

Our data showed no effect upon AV conduction of the three concentrations of isoflurane used. Standard errors in A–H intervals in our study are larger than those previously found during enflurane and halothane anesthesia.[12–14] We interpret this as being due to the marked variability of effects of isoflurane on the AV node compared with halothane or enflurane. Isoflurane, then, does not affect the AV node in a dose-related manner as does halothane or enflurane. Altered conduction through the AV node may be a contributing mechanism to cardiac arrhythmias during general anesthesia. Lack of depressed conduction within the AV node would tend to hinder the re-entry of a sinus impulse. The antiarrhythmic properties of isoflurane, as well as the stability of cardiac rhythm in patients anesthetized with isoflurane when challenged by administration of catecholamines, may be related to the lack of effect of isoflurane on the AV node.

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