TITLE: INOTROPIC EFFECT OF ETOMIDATE ON CARDIAC PAPILLARY MUSCLE IN NORMAL AND CARDIOMYOPATHIC HAMSTERS

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Etomidate (E) has been shown to induce only a slight effect on the intrinsic mechanical properties of rat cardiac papillary muscle (1). However, the effects of E on diseased myocardium remain to be determined. We studied the mechanical effects of E on intrinsic contractility of papillary muscles in normal (NO) and cardiomyopathic (CM) hamsters.

Left ventricular papillary muscles were excised from NO (n=9) and CM (n=10) (Strain BIO 82-62) hamsters (6-month old). They were suspended in a Kreibs-Henseleit solution bubbled with 95% O2-5% CO2 (pH 7.40, 29°C, Ca++ 2.5 mM), field stimulated (3/min), and exposed to 2 concentrations of E (1 then 5 µg.ml-1). The following parameters of inotropy were determined from various afterloaded twitches : the maximum unloaded shortening velocity (Vmax), and the active isometric force normalized per cross-sectional area (AF/s). The Hill's equation was determined from the force-velocity curve to calculate the peak power output (Emax). Analysis was performed using repeated measures analysis of variance and Newman-Keuls test. Data are mean + SD.

E did not modify the inotropic parameters under low (Vmax) and high load (AF/s) in either NO or CM hamsters. In both groups, E did not modify the energetics of papillary muscle (Emax) (Table). It was concluded that E has no significant effect on the intrinsic mechanical properties of diseased myocardium. These results concord with those obtained in patients with cardiac disease (2), and could be important since most anesthetics are myocardial-depressant agents (3).

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References

1. Anesthesiology 72 : 330-340, 1990
2. Br. J. Anaesth. 51 : 551-555, 1979
3. Anesthesiology 69 : 445-447, 1988

Table: Inotropic effect of etomidate.

	NORMAL (n=9)		CARDIOMYOPATHIC (n=10)	
E (µg/m-1)	1	5	1	5
Vmax	102 ± 7	108 ± 15	106 ± 13	109 ± 11
AF/s	100 ± 11	106 ± 26	107 ± 19	101 ± 14
Emax	106 ± 22	111 ± 38	109 ± 27	108 ± 21

Mean percent of control values ± SD. No differences were significant.

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Title: THE ROLE OF THE ENDOTHELIUM IN MODULATING THE VASODILATION INDUCED BY ISOFLURANE IN THE RABBIT BASILAR ARTERY

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The role of the endothelium in modulating the response to volatile agents is largely unknown and available studies have yielded conflicting results (1,2). We studied the ability of isoflurane to relax potassium constricted rabbit basilar arterial segments in the presence and absence of the vascular endothelium.

Basilar arteries from 10 NZW rabbits were studied. The proximal 4.0 mm served as a control and the distal 8.0 mm was cannulated with a 30 gauge needle that was attached to a 95%O_2/5% CO_2 gas source. A gentle stream of this gas was passed through the lumen of the vessel for 17min. The middle 4.0 mm was then cut free. Each of these segments was threaded onto a rigid clip and attached to tension transducer in a plexiglass perfusion chamber. The vessel chambers were continuously supplied with 37°C buffered perfusate (pH 7.40). 95% O_2/5% CO_2 flowing through anesthetic vaporizers was bubbled through the perfusate columns to saturate the perfusate with O_2, CO_2, and controlled concentrations of anesthetic. Each of the segments was perfused for 30min, and were then stretched to 2000 dynes and constricted by changing the perfusate to one containing 30 mM K+ (ISK-30). Functional removal of the endothelium was verified by exposing the segments to 5.0 X 10^{-6} M acetylcholine and observing that relaxation did not occur. The vessels were then exposed to 15min pulses of isoflurane in doses of approximately 0.5, 1.0, 1.5, and 2.0 MAC in a stepwise fashion, with a 30 min period of perfusion with anesthetic-free ISK-30 between each dose.

The dose-response curves for intact and deendothelialized arteries are shown in the figure. ANOVA demonstrated that the lines are indistinguishable, with 50% relaxation achieved at 1.67 MAC for intact vessels and 1.68 MAC for the deendothelialized vessels.

We conclude that isoflurane induced relaxation of rabbit basilar artery segments preconstricted with potassium is not endothelium dependent.

1. Muldoon et al. Anesthesiology 68:31-37, 1988. 2. Stone et al. Anesthesiology 71:126-132,1989