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Cardiovascular and Pulmonary Responses following Etomidate Induction of Anesthesia in Patients with Demonstrated Cardiac Disease

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Cardiovascular and pulmonary effects following the administration of 0.3 mg/kg of etomidate were studied in patients with documented cardiac disease. The only significant change was a slight elevation (2 torr) in arterial carbon dioxide tension.

Key Words: ANESTHETICS, Intravenous: etomidate; INDUCTION, Anesthetic: etomidate.

AN intravenous drug that produces rapid onset of hypnosis without adverse effects on the respiratory and cardiovascular systems would be appreciated by anesthesiologists. Such a drug would fill an existing gap in our current armanentarium of anesthetic induction agents. Previous reports have suggested that etomidate, a new rapid acting nonbarbiturate hypnotic, lacks significant adverse cardiovascular and respiratory effects.[1-8] However, these studies did not include patients with preexisting cardiovascular disease.

In order to assess the cardiovascular and respiratory effects of etomidate in high risk patients we undertook such a study in surgical patients with demonstrated cardiac disease.

Materials and Methods

Twenty-two patients (18 men and four women, mean age 55 ± 9 years), A.S.A. classes III and IV, were studied. (Approval for the study was given by the Use of Human Subjects Committee at Tulane University School of Medicine. Informed consent was obtained from each subject.) All had existing cardiac disease documented by prior catheterization. Two patients were scheduled for peripheral vascular surgery, eight for valvular heart surgery, and 12 for coronary artery bypass grafting. Premedication consisted of 5 mg of droperidol 1 hour prior to anesthetic induction in every patient. Balloon flotation, thermistor-tipped, pulmonary artery catheters as well as radial artery catheters were inserted percutaneously. Control measurements of heart rate (HR), mean arterial pressure ($\overline{AP}$), central venous pressure (CVP), mean pulmonary artery pressure ($\overline{PAP}$), pulmonary artery occlusion pressure (PAOP), cardiac output (CO), and arterial and mixed venous blood gas tensions were obtained. Etomidate was then injected intravenously in a dose of 0.3 mg/kg in 60 seconds. Oxygen was administered preceding and during the induction period. The above measurements were repeated between 1 and 3 minutes following etomidate

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TABLE

Effect of Etomidate on Cardiac and Pulmonary Variables (n = 22)*

	Before etomidate	After etomidate
HR	69 ± 12	70 ± 9
AP (torr)	91 ± 18	88 ± 21
CVP (torr)	6 ± 5	7 ± 4
PAP (torr)	20 ± 5	20 ± 5
PAOP (torr)	14 ± 5	15 ± 5
CI ($L \cdot min^{-1} \cdot m^{2-2}$)	2.6 ± 0.7	2.7 ± 0.6
Pa_{O_2} (torr)	290 ± 68	292 ± 70
Pa_{CO_2} (torr)	41 ± 4	43 ± 5†
$C(a - \bar{v})_{O_2}$ (ml %)	4.6 ± 1	4.7 ± 1
$\dot{Q}_{sp}/\dot{Q}_t$ (%)	21 ± 0.05	21 ± 0.05

* Values are means ± SD. See text for abbreviations.
† $p < 0.05$.

injection. Using the above data and body surface area, cardiac index (CI), arteriovenous oxygen content difference ($C(a-\bar{v})O_2$), and total intrapulmonary shunt ($\dot{Q}_{sp}/\dot{Q}_t$) were calculated.

Results

Due to rapid onset and brief duration of action of etomidate (at a dose of 0.3 mg/kg, sleep is produced in a single arm-brain circulation time and inactive metabolites peak by 7 minutes[6, 9]), it was elected to obtain data between 1 and 3 minutes following injection. A slight elevation of arterial carbon dioxide tension (Pa_{CO_2}) was the only change of statistical significance (Table); however, this change was not clinically remarkable. Some patients tended to develop upper airway obstruction indicated by episodes of snoring. No other consistent effects on respiration were noted.

Discussion

Hemodynamic stability was observed following the administration of 0.3 mg/kg of etomidate in patients at risk because of cardiac disease. The slight elevation in heart rate reported in previous studies of noncardiac patients[1, 3, 4] did not occur in our patients. This may be due to the reduced incidence of venous pain on injection with the new, less acidic (pH 5.0) solution[10] that was used in our patients. The stability of cardiovascular function suggests a lack of significant effects on either the peripheral and pulmonary vascular beds or on the myocardium itself. Absence of appreciable changes in arterial blood gases confirms previous findings.[3] Total intrapulmonary shunt was not altered and suggests a role for etomidate in anesthetizing patients with respiratory insufficiency and/or preexisting intrapulmonary shunting.

Cardiovascular stability in our study of high risk cardiac patients corroborates previous findings in noncardiac patients. Alterations in arterial blood gas tensions or total intrapulmonary shunt did not occur. In patients at risk because of heart disease it would appear that etomidate would provide a greater margin of safety during the induction of anesthesia than the ultrashort-acting barbiturates which produce vascular dilation, adversely affect cardiac output, and depress respiration.

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