

TITLE : PERIPHERAL VASCULAR EFFECTS OF ETOMIDATE

AUTHORS : P. Léger, M.D., J.J. Rouby, M.D., A. Andreev, M.D., M. Arthaud, Ph, S. Cheour M.D., F. Durand, M.D., C. Cabrol, M.D. and P. Viars M.D.

AFFILIATION : Département d'Anesthésie et Service de Chirurgie Cardiovasculaire de la Pitié-Salpêtrière, Université Paris VI, 83 Boulevard de l'Hôpital, 75651 PARIS Cedex 13 - FRANCE - and Department of Anesthesiology of Tunis, TUNISIA

INTRODUCTION

Etomidate is a short-acting non-barbiturate anesthetic agent which has been said to induce less cardiovascular depression than other induction agents¹. It has been proposed as the first choice for patients with cardiovascular instability. In this study, we measured etomidate induced peripheral vascular effects in mechanically ventilated critically ill patients with a Jarvik-7 total artificial heart.

METHOD

Patients : Six patients (mean age 45 ± 7 years) who had a Jarvik-7 artificial heart implantation whilst waiting for cardiac transplantation², were included in the study, according to the following criteria : 1) presence of respiratory failure requiring mechanical ventilation 2) presence of agitation or discoordination from the ventilator, requiring intravenous sedation 3) absence of the administration of any sedative or vasoactive drug in the 12 preceding hours. In each case, informed consent was obtained from the nearest relative and authorization given by the Ethical Committee of our Institution.

Hemodynamic measurements : for cardiovascular monitoring, each patient already had an arterial line and a right atrial catheter percutaneously inserted, and left atrial and pulmonary artery catheters surgically implanted. Two patients had a Jarvik-7 artificial heart model 70 ml and 4 had a Jarvik-7 artificial heart model 100 ml. Right and left cardiac output, and right and left stroke volume were continuously monitored using the cardiac diagnostic unit of the Utah heart-drive console (COMDU TM). Arterial and mixed venous blood samples were simultaneously withdrawn for blood gas analysis and SaO₂ and SvO₂ were measured using a Co-oximeter OSM3. Arteriovenous O₂ difference, oxygen consumption and venous admixture were calculated using standard formula. Arterial pressure, cardiac filling pressures and right and left cardiac output were simultaneously recorded on a Gould ES 1000 multichannel recorder.

Procedure : Initially, the Jarvik-7 artificial heart was set to provide adequate circulatory conditions without the ventricular chambers being filled at the end of diastole. For the period of the study, the Jarvik-7 settings were modified, in order to maintain cardiac output constant in presence of a moderate decrease in venous return : the diastolic time was lengthened by decreasing frequency and/or increasing percentage of systole, so that each ventricular chamber was completely filled well before the end of the time allotted to diastole. With such settings, despite a decrease in venous return, each cardiac chamber remains filled at the end of diastole and cardiac output remains

constant despite the decrease in right atrial pressure. Consequently, the Jarvik-7 artificial heart ensures a constant preload independent cardiac output and represents a unique model for studying the peripheral vascular effects of any drug, independent of its action on cardiac output.

Five minutes after changing Jarvik-7 settings, hemodynamic parameters were recorded and blood samples withdrawn (control values). Etomidate 0.3 mg/kg was then intravenously injected over a 10 second period and hemodynamic parameters were continuously recorded during the following 30 minutes. Blood samples were withdrawn 5, 10, 15 and 30 minutes after etomidate injection. All data are expressed as mean \pm SD and were analysed by a one-way analysis of variance.

RESULTS

1) The Jarvik-7 settings used were : frequency 78 ± 11 bpm, percentage of systole 44 ± 3 %, drive pressure of the left ventricle 193 ± 11 mmHg, drive pressure of the right ventricle 58 ± 12 mmHg.

2) PaO₂ (186 ± 104 mmHg at control using a FIO₂ of 70 ± 15), PaCO₂ (36 ± 2 mmHg at control), temperature ($36 \pm 0.5^\circ\text{C}$ at control) remained unchanged during the study.

3) As shown in the table, all hemodynamic parameters remained unchanged throughout the study period.

Table mean \pm SD	Control	5 min	10 min	15 min	30 min
Mean arterial pressure (mmHg)	72 ± 10	71 ± 11	74 ± 18	76 ± 17	82 ± 19
Mean pulmonary arterial pressure (mmHg)	18 ± 7	20 ± 7	21 ± 7	20 ± 6	21 ± 8
Left atrial pressure (mmHg)	12 ± 6	12 ± 4	12 ± 4	11 ± 4	12 ± 5
Right atrial pressure (mmHg)	14 ± 5	15 ± 4	15 ± 5	15 ± 5	15 ± 4
Venous admixture (%)	27 ± 6	27 ± 9	28 ± 12	27 ± 11	24 ± 10
Left cardiac output (l/min/m ²)	3.0 ± 0.5	3.0 ± 0.3	2.9 ± 0.5	2.9 ± 0.4	2.9 ± 0.5

DISCUSSION

This study confirms that etomidate does not reduce vascular tone, either at the venous level or at the pulmonary and arterial levels. Because etomidate provides remarkable cardiovascular stability, its beneficial effect in patients with decreased cardiac reserve should be weighed against its potential detrimental effect on steroid synthesis³.

REFERENCES

- 1 - Prakash et al. Br. J. Anaesth, 53 : 591, 1981
- 2 - De Wries et al. N.E.J.M., 310 : 273, 1984
- 3 - Ledingham et al. Lancet, ii : 1270, 1983