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Effect of halothane, enflurane and isoflurane on the end-systolic pressure-length relationship

Myocardial contractility was measured using the end-systolic pressure-length (ESPL) relationship in dogs subjected to increasing concentrations of halothane (0.5–2 per cent), enflurane (0.77–2.6 per cent) or isoflurane (0.70–2.13 per cent), combined with an infusion $7 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ of fentanyl, after induction of anaesthesia with $15 \text{ mg}\cdot\text{kg}^{-1}$ thiopentone. The relationship between the concentrations of the different drugs and contractility (ESPL) can best be described by $\text{ESPL} = a + b(\text{MAC fraction})$ where "a" is a constant and "b" is the slope of the curve relating ESPL to MAC. At 1.0 MAC values, the ESPL for halothane ($69.04 \pm 25.83 \text{ mmHg}\cdot\text{mm}^{-1}$) did not differ from that of isoflurane ($63.19 \pm 17.36 \text{ mmHg}\cdot\text{mm}^{-1}$). However, the myocardial contractility during 1.0 MAC halothane and isoflurane anaesthesia was better preserved than that of enflurane ($38.66 \pm 9.73 \text{ mmHg}\cdot\text{mm}^{-1}$; $p < 0.01$, $p < 0.05$ respectively).

Key words

HEART: end-systolic pressure-length relationship, myocardial contractility; ANAESTHETICS, VOLATILE: halothane, enflurane, isoflurane.

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The potent anaesthetic inhalational agents halothane, enflurane and isoflurane cause a dose-related depression of myocardial contractility.^{1–9} However, because of the difficulty in assessing contractility, the differences between the drugs are still debated. We therefore utilized the sensitive and load-independent index of myocardial contractility, the end-systolic pressure length ratio (ESPL or Ees),^{10–18} to determine whether there is a difference in myocardial depression when equipotent concentrations of halothane, enflurane or isoflurane are combined with a constant infusion of fentanyl.

Methods

Thirty mongrel dogs (mean weight 19.20 ± 3.6 (SEM) kg) were used for this study. The animals were cared for according to institutional and national guidelines for laboratory animals.

Animals were premedicated with morphine $1.5 \text{ mg}\cdot\text{kg}^{-1}$ IM and anaesthesia was induced with fentanyl $15 \mu\text{g}\cdot\text{kg}^{-1}$ and thiopentone $15 \text{ mg}\cdot\text{kg}^{-1}$ IV. They were intubated and ventilated with 40 per cent oxygen and 60 per cent nitrogen (fresh gas flow = $3 \text{ L}\cdot\text{min}^{-1}$; CO_2 absorber in circle). The anaesthetic gases were vaporized from calibrated vaporizers (halothane: Drägerwerk, Germany; enflurane and isoflurane: Cyprane, UK) and the end-tidal gas concentrations were monitored with an anaesthetic gas monitor (Normac, Datex, Finland). The tidal volume was adjusted to maintain the PaCO_2 between 38–42 mm Hg.

Normal saline, $5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ was infused into a front leg vein and fentanyl $7 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ was added to the saline. The temperature of the operating table could be controlled and blood temperature was maintained at $36.5\text{--}37.3^\circ\text{C}$ as measured from the thermistor at the tip of a pulmonary artery catheter.

Through an incision in the neck, a 16 g cannula was positioned in the internal jugular artery with the tip of the cannula being within 1 cm from the aortic valve. This was connected to a pressure transducer (Stratham P23,

FIGURE 1 Demonstration of the load independence and linear characteristics of the ESPL relationship. The dark pressure-volume loop indicates the control loop. The preload was reduced and allowed to return to control after which the afterload was immediately increased and this particular photograph taken. The end-systolic pressure-length line is both linear and independent of changes in the pre- and after-load.

Statham, Hato Rey. Natural frequency = 50.3 Hz) for the measurement of arterial blood pressure.

A 7 F pulmonary artery catheter (Edwards Lab, USA) was inserted through the external jugular vein and positioned in the pulmonary artery. The pressure tracing was utilized to guide placement but the position was verified once the chest was opened and the pulmonary artery could be palpated. Cardiac output (CO) was calculated with a cardiac output computer (Mansfield 9530, USA) after manually injecting 5 ml five per cent dextrose (0°C) in less than 4 sec, at the end of expiration. The mean of three measurements is reported. (The coefficient of variation for 400 triplicate measurements in this laboratory is 4.78 per cent.)

Through the femoral artery and vein, occlusion catheters (Edwards Lab, USA), size 8/14 F (model 62-080-8114 F) were placed in the aorta and inferior vena cava. The tip of the aortic catheter was confirmed to be 1 cm distal to the aortic arch in the descending aorta and the vena cava catheter was positioned between the diaphragm and right atrium. The catheters were used to change the pre- and after-load of the heart in order to obtain end-systolic pressure-length points to construct the ESPL.

A left thoracotomy and pericardiectomy were performed and the heart was suspended in a pericardial cradle. A 16 g cannula was sutured into the apex of the left ventricle (LV), and was connected to a pressure transducer (Stratham P23 Db. Natural frequency 50.6 Hz) in order to measure LV pressure. Utilizing a magnified calibration, left ventricle end-diastolic pressure (LVEDP) was also obtained from the LV catheter.

Two piezo-electric crystals (Schuessler and Ass, USA)

were placed 1 cm apart in the minor axis of the LV endocardium. The minor axis was defined as a plane perpendicular to the aortic root-LV apex line. The crystals were inserted 1.5 cm from the left anterior descending coronary artery, midway between the base of the heart and the apex. Endocardial fibre length was measured during the cardiac cycle from the crystals (Ultrasonic Dimension System, Schuessler and Ass), the maximum fibre length (L_{max}) taken at the time the sharp upslope of the LV pressure started and the minimum length (L_{min}) taken at the time of aortic valve closure. Systolic shortening ($dL = L_{max} - L_{min}$) could be determined and was normalized for L_{max} and expressed as a percentage ($dL\%$).

Recordings were made on a six-channel recorder (Beckman Instr, USA). All recordings were made five seconds after the ventilator was disconnected (airway pressure zero). Calibrations were done with a mercury manometer, before and during experiments.

The LV pressure signal and endocardial fibre shortening were combined on a storage oscilloscope (Tektronix Model 5103N, USA) to give a pressure-length loop (Figure 1). Standard calibrations were used throughout. The loops were photographed and were used to give an indication of regional function¹⁹ while the loop area was planimetered to represent regional myocardial work and oxygen consumption.^{20,21}

From the RR interval of the ECG, the heart rate (HR) was recorded and dp/dt_{max} was obtained from the recordings. Blood gases, bicarbonate concentration, pH, haemoglobin concentration and haemoglobin saturation, as well as carboxyhaemoglobin were measured (IL 282 Co-oximeter, IL 613 Bloodgas Analyser, Instrumentation Labs, USA)²² between the experimental steps and correction were applied when necessary.

By increasing the aortic or inferior vena cava balloon volume, the afterload or preload could selectively be increased or decreased. The resultant change in the volume and configuration of the pressure-length loop was photographed and an end-systolic pressure-length line could be drawn (Figure 1).

The blood levels for thiopentone was done with thin layer chromatography in 12 experiments.

Data were stored on disc and the paired student's *t* test was used where applicable. For statistical analysis of the differences between the slopes of the various regressions performed, analysis of covariance was utilized. A value of $p < 0.05$ was considered to be significant.

Experimental protocol

After completion of surgery, calibrations were performed, the blood gas status checked and 20–25 minutes were allowed for equilibration at the initial experimental anaesthetic gas value.

TABLE I Arterial blood gases and pH for 150 samples representing all the experimental steps. Mean $\pm$ SEM

Temp °C	PaO ₂ (mmHg)	PaCO ₂ (mmHg)	pH
36.75 ± 0.09	185.07 ± 14.37	38.14 ± 2.22	7.43 ± 0.004

Each group evaluated consisted of ten dogs, each dog being subjected to a single anaesthetic agent. The anaesthetic concentrations (randomly selected for a particular drug) used were:

Halothane:	0.5	0.7	1.0	1.5	2	%
Enflurane:	0.77	1.2	1.68	2.13	2.6	%
Isoflurane:	0.70	1.13	1.52	1.90	2.13	%

These concentrations are the mean end-tidal concentrations which corresponded to constant settings on the vaporizers. MAC values which will be referred to subsequently are 0.87 per cent for halothane, 2.20 per cent for enflurane and 1.48 per cent for isoflurane.^{23,24} Twenty-five minutes were allowed for equilibration between each step and during that period calibrations and arterial blood gases and pH were rechecked and corrected when necessary.

After the general haemodynamics were recorded and blood collected for blood gas studies, the aortic occlusion balloon was partially inflated in 3–5 seconds. The change in the pressure-length loop was photographed, after which the balloon was deflated. After a 3-min period, the IVC balloon was inflated in 5–7 sec and the resultant change in the pressure-length loop was again photographed. The circulation was allowed to stabilize after deflation of the IVC balloon (5 min) before the anaesthetic concentration was increased.

Results

Data presented are the mean $\pm$ SEM for ten experiments.

The concentration of thiopentone in the blood for the duration of the experiments were:

Before induction	=	0.00 $\mu\text{g}\cdot\text{ml}^{-1}$
5 min after induction	=	84.44 $\pm$ 17.98 $\mu\text{g}\cdot\text{ml}^{-1}$
60 min after induction	=	27.60 $\pm$ 6.71 $\mu\text{g}\cdot\text{ml}^{-1}$
120 min after induction	=	6.06 $\pm$ 1.60 $\mu\text{g}\cdot\text{ml}^{-1}$
180 min after induction	=	4.82 $\pm$ 1.26 $\mu\text{g}\cdot\text{ml}^{-1}$
240 min after induction	=	3.50 $\pm$ 0.97 $\mu\text{g}\cdot\text{ml}^{-1}$
300 min after induction	=	3.00 $\pm$ 1.20 $\mu\text{g}\cdot\text{ml}^{-1}$

Table I summarizes the arterial blood gases and temperatures for all the experimental stages. The blood gases and haemoglobin did not vary significantly during the experiments.

Table II summarizes the basic haemodynamic data.

FIGURE 2 The change in the ESPL relationship as the anaesthetic gas concentration was increased. The regression analysis for the various drugs are:

Halothane: $\text{ESPL} = -3.64 + 72.68/\text{MAC}$

Enflurane: $\text{ESPL} = -16.456 + 55.13/\text{MAC}$

Isoflurane: $\text{ESPL} = -95.37 + 158.56/\text{MAC}$

Statistical analysis indicated differences in the slopes for the regression lines between halothane and isoflurane ($p < 0.01$) and halothane and enflurane ($p < 0.001$). However, no difference could be demonstrated between enflurane and isoflurane.

With each of the drugs there was a decrease in cardiac output as the anaesthetic concentration was increased and this was accompanied by a decrease in arterial pressure. The heart rate and filling pressure of the left ventricle remained constant.

Table III summarizes the end-systolic pressure-length data and indicates statistical differences where applicable. There was a decrease in ESPL as the anaesthetic depth was increased. The line describing the trend (Plot 50, Textronics, USA) for the ESPL against MAC values were $\text{ESPL} = a + b/(\text{MAC fraction})$ (Figure 2). An excellent correlation for the data points around the line was demonstrated (halothane $r = 0.99$, $p < 0.01$, $n = 10$; enflurane $r = 0.99$, $p < 0.01$, $n = 10$; isoflurane $r = 0.98$, $p < 0.01$, $n = 10$).

When the slopes for the various regression analyses were compared, no difference could be demonstrated between isoflurane and enflurane ($F = 0.00$, $df = 1, 6$) but a significant difference could be demonstrated when halothane was compared with isoflurane ($F = 14.51$, $df = 1, 6$, $p < 0.01$) and enflurane ($F = 25.84$, $df = 1, 6$, $p < 0.01$). The data from Table III are replotted as MAC against $1/\text{ESPL}$ values to yield a linear regression (Figure 3).

Discussion

There are a number of limitations in the model we have been using which needs to be put into perspective. This study should not be viewed as a cardio-pharmacological study but should rather be seen as relevant to clinical practise for reasons which are outlined below.

For logistical and ethical reasons, it was elected to

TABLE II Haemodynamic data. Mean $\pm$ SEM (n = 10)

	<i>Halothane MAC</i>					<i>Enflurane MAC</i>				
	0.57	0.80	1.15	1.72	2.30	0.35	0.55	0.76	0.97	1.17
HR (beats·min ⁻¹)	94.70 ± 5.82	95.80 ± 3.99	93.70 ± 4.20	98.50 ± 3.99	97.50 ± 3.80	96.40 ± 9.40	100.00 ± 5.92	99.70 ± 7.52	99.60 ± 7.37	102.50 ± 6.91
MAP (mmHg)	77.73 ± 4.58	75.93 ± 4.40	71.33† ± 3.41	57.13‡ ± 2.69	42.40‡ ± 2.82	69.37 ± 4.64	70.40 ± 2.59	66.20 ± 2.47	58.40 ± 2.40	51.03* ± 2.75
LVEDP (mmHg)	5.70 ± 0.75	6.21 ± 0.65	6.36 ± 0.65	5.60 ± 0.84	5.85 ± 0.86	5.40 ± 0.37	6.03* ± 0.68	6.15 ± 0.54	5.44 ± 0.70	6.00 ± 0.50
CI (L·min ⁻¹ ·m ⁻²)	3.27 ± 0.32	3.13 ± 0.23	2.54* ± 0.12	2.00† ± 0.07	1.42‡ ± 0.07	2.88 ± 0.27	2.47 ± 0.30	2.43 ± 0.19	2.20† ± 0.14	1.71 ± 0.10

	<i>Isoflurane MAC</i>				
	0.52	0.76	1.02	1.28	1.44
HR (beats·min ⁻¹)	114.30 ± 6.22	114.60 ± 6.34	120.50 ± 5.57	126.30 ± 4.32	125.10 ± 4.25
MAP (mmHg)	84.30 ± 7.43	79.40 ± 5.28	75.00 ± 3.65	66.07† ± 4.11	60.73† ± 4.29
LVEDP (mmHg)	6.01 ± 1.00	5.80 ± 1.12	6.20 ± 0.83	5.39 ± 0.96	7.02 ± 1.59
CI (L·min ⁻¹ ·m ⁻²)	2.87 ± 6.24	2.73 ± 0.22	2.68 ± 0.19	2.38* ± 0.29	2.10† ± 0.20

Statistical analysis was performed between the initial value for each agent and the subsequent steps.

*p < 0.05.

†p < 0.01.

‡p < 0.001.

TABLE III Myocardial contractility (Ees) and the volume in the LV when the LV pressure is zero (V₀) are given in this table. Mean $\pm$ SEM (n = 10)

	<i>Halothane (MAC)</i>					<i>Enflurane (MAC)</i>				
	0.57	0.80	1.15	1.72	2.29	0.35	0.55	0.76	0.97	1.17
Ees (mmHg·min ⁻¹)	137.07 ± 16.58	105.73† ± 16.72	62.83‡ ± 7.83	46.27‡ ± 7.26	29.18‡ ± 4.15	142.06 ± 38.55	79.31 ± 15.01	61.65 ± 9.40	41.65* ± 5.32	28.16* ± 2.46
V ₀ (mm)	8.16 ± 0.68	8.14 ± 0.66	7.81 ± 0.59	7.48 ± 0.58	7.42 ± 0.82	9.30 ± 0.72	8.95 ± 0.76	9.01 ± 0.78	8.98 ± 0.78	8.80 ± 0.83

	<i>Isoflurane (MAC)</i>				
	0.52	0.76	1.03	1.28	1.44
Ees (mmHg·min ⁻¹)	225.01 ± 100.50	93.70 ± 17.22	47.69* ± 6.27	37.40* ± 3.67	27.88* ± 2.51
V ₀ (mm)	7.85 ± 0.97	7.80 ± 0.27	7.08 ± 0.34	7.01 ± 0.35	6.75 ± 0.56

For statistical analysis each subsequent step was compared with the lowest value for each agent.

*p < 0.05.

†p < 0.01.

‡p < 0.001.

FIGURE 3 MAC values plotted against the function $1/\text{ESPL}$ in order to show the differences between the slopes for the various regressions. The linear regression for the drugs are:

Halothane: $y = 0.015(\pm 0.001) - 0.002$ $r = 0.997$ $p < 0.001$
 Enflurane: $y = 0.033(\pm 0.004) - 0.006$ $r = 0.948$ $p < 0.001$
 Isoflurane: $y = 0.003(\pm 0.002) - 0.014$ $r = 0.988$ $p < 0.001$

The slope for halothane was significantly different from that of enflurane and isoflurane ($p < 0.001$ and $p < 0.01$) but enflurane and isoflurane did not differ significantly.

induce general anaesthesia with thiopentone. A previous study demonstrated that very little, if any, depression of myocardial contractility ($dp/dt_{\max}$; dQ/dt) occurred at thiopentone concentrations of 6–12 mg·100 ml⁻¹.²⁵ Experiments in this study were conducted in the period 120–300 min at which time the blood concentration was less than 12 mg·100 ml⁻¹. Furthermore, no difference could be demonstrated between the values obtained during the time period 120–300 min. Although the use of thiopentone can be criticized, the low but constant blood levels during the experiment enable the observer to make valid deductions from the results obtained.

Because the initial values of the inhalational agents used for the experiments were less than 1.0 MAC, it was deemed necessary to add fentanyl as a constant infusion to obtain any possible perception of pain by the experimental animals. It is not expected that this dose of fentanyl would cause myocardial depression.²⁷ Even if the fentanyl did cause changes in the preload or afterload, this will not affect the measurement of the end-systolic pressure-length (ESPL) relationship as this index is load independent.

In this study the ESPL was determined for halothane, enflurane and isoflurane by increasing the afterload to LV ejection with an aortic balloon. For each of the drugs there was a decrease in ESPL as the end-tidal anaesthetic gas concentration was increased and the lines which describe this trend are demonstrated in Figure 2. When projec-

tions were made for values at 1.0 MAC for the three agents, the ESPL for halothane was 69.04 ± 25.83 mmHg·mm⁻¹, for enflurane 38.66 ± 9.73 mmHg·mm⁻¹ and for isoflurane 63.19 ± 17.36 mmHg·mm⁻¹. A difference was demonstrated for both halothane and isoflurane when compared to enflurane ($p < 0.01$ and $p < 0.05$), but no difference was shown between values obtained for halothane and isoflurane.

In the only other study in which the end-systolic pressure-volume relationship (ESPV) was utilized as a method to compare the effects of anaesthetic drugs, halothane, enflurane and N₂O were evaluated in dogs (with awake controls) and patients in the post-cardiopulmonary bypass period.²⁷ These authors demonstrated a 20 per cent reduction in ESPV, compared to awake controls, at 0.72 MAC halothane and 0.71 MAC enflurane. In our study, a 20 per cent reduction in contractility (compared to the initial concentration used) was evident at 0.71 MAC halothane. However, we did not have awake controls although the percentage myocardial depression at 0.71 MAC would suggest that the initial values used in this study are comparable to awake controls. Nevertheless, such an extrapolation is not justified.

The data in Figure 2 appear to suggest that it is possible for the ESPL ratio to have an infinite value if the MAC value is zero. We have observed a vertical end-systolic pressure-length line in animals kept under anaesthesia with fentanyl (i.e., no myocardial depression). This indeed suggests that the slope of pressure-length relationship can have a value which tends towards infinity, due to the maximum value for pressure and a constant value for length. The equation also suggests that the ESPL ratio will become zero and may even assume a negative value for certain of the anaesthetic agents tested. However, although it is mathematically correct to suggest that the value for "y" tends towards zero, it is not a physiological possibility. If the ESPL relationship is zero, it will imply that the heart does not generate pressure and hence no circulation is possible. Moreover, as the ESPL ratio is decreased during the depression of the heart, it will approach the diastolic pressure-length relationship (passive pressure-length relationship) and once the line ESPL is equivalent to the diastolic pressure-length relation, no function is possible. This will only apply if the diastolic pressure-length relationship is not altered in a similar direction. There is therefore, a difference in the mathematical possibility and the physiologic reality.

It is also relevant to note that the volume in the ventricle when the pressure was zero (V_0) did not change (Table III and Figure 4). In this study the abbreviation V_0 was used, although it will have units in mm opposed to ml as in the ESPV relationship. This signifies that the ESPL did not undergo a parallel shift (which is not similar to a change in

FIGURE 4 A series of pressure-length loops obtained in this study. The end-systolic pressure and volume points used to calculate the ESPL value, as well as the regional myocardial length of the ventricle at the time the ventricular pressure is zero (V_0), are indicated. In studies conducted for these experiments, regional length and not LV volume was used as an index of volume and hence the units will be in mm.

ESPL per se) and that the trend (decrease) of the ESPL, as registered in this study, does in fact represent a depression in myocardial contractility.

In order to accentuate the differences in the slopes for the relationship ESPL and MAC multiples for the drugs tested, a plot of $1/\text{ESPL}$ was attempted (Figure 3). From this it can clearly be seen that halothane will be the drug associated with the least myocardial depression as the concentration of the anaesthetic agent is increased.

The end-systolic pressure-volume (ESPV) relationship of the heart is regarded as a sensitive and load-independent method of quantifying myocardial contractility (Figure 4). This index is based on the observation that the end-systolic pressure-volume relationship has a linear character which is independent of the method by which it is achieved, i.e., isometric or isotonic contractions.¹⁰⁻¹⁶ Because of this characteristic, the end-systolic pressure-volume relationship sets the margins for myocardial fibre shortening.²⁸ As this index is independent from changes in the myocardial loading conditions, either a reduction in preload or an increase in afterload can be used to obtain the various pressure and volume points to compute the ESPL or ESPV. To date the majority of work has been done with the ESPV relationship. The end-systolic pressure-length (ESPL) relationship was used as an index of contractility in this study. It has been established that the ESPL relationship can serve as an index of contractility,^{17,18} but a distinction should be made between the ESPV relationship as an index of global LV function as opposed to the ESPL ratio which serves as an index of regional contractility. However, if the coronary arteries are normal there is little reason to

believe that there will be a difference between contractility in the rest of the myocardium and the area measured in this study. It should further be noted that the ESPV and ESPL relationships are not equivalent because of the nature of the relationship of volume to circumference of a sphere and that ESPL has been shown to change to a similar extent as the change in ESPV when myocardial contractility is altered.^{17,18}

In Figure 1 the linear and load-independent character of the method used in this study is demonstrated. For the total study the correlation between three end-systolic pressure and volume points were highly significant ($r = 0.99$, $n = 450$, $p < 0.001$).

Contractility and the function of the heart as a pump is not synonymous and the latter may be the more important in clinical practice. Contractility represents a "potential" to deliver a certain flow but this potential can be modified by heart rate, the positive relationship between preload and flow (within certain limits) and the inverse relationship between stroke volume and ventricular ejection pressure. Due to changes in afterload, either because of the characteristic of the anaesthetic agent or manipulations by the anaesthetist, severe depression of contractility as such can usually be accommodated without causing an insufficient cardiac output. This "reserve" in myocardial contractility is less in the patient with heart disease and in these patients, or in experimental work, the correct quantification of contractility is essential and it is suggested that the end-systolic volume or length and pressure relationship is used to that end.

Because of the other drugs involved in the study, results obtained should not be used as an indication what the effect of the three inhalational agents alone would have been on myocardial contractility. However, because of the current trend in anaesthesia with regard to the use of opiates, we believe the results of this study are relevant to everyday practice.

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Résumé

Chez des chiens on a mesuré la relation entre la contractilité myocardique (mesurée à l'aide de la longueur-pression télé-diastolique) (LPTD) et des concentrations croissantes d'halothane (0.5-2 pour cent), d'enflurane (0.77-2.6 pour cent) et d'isoflurane (0.70-2.13 pour cent) combiné à une infusion de $7 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ de fentanyl après avoir fait une induction anesthésique avec $15 \text{ mg} \cdot \text{kg}^{-1}$ de thiopental. La relation entre les différents médicaments et la contractilité (LPDT) peut être mieux décrite par l'équation $\text{LPTD} = a + b(\text{fraction MAC})$ où a est une constante et b est la pente de la courbe qui met en relation la LPTD au MAC. A des valeurs MAC 1, la LPTD pour l'halothane ($69.04 \pm 25.83 \text{ mmHg} \cdot \text{mm}^{-1}$) ne différait pas de celle de l'isoflurane ($63.19 \pm 17.36 \text{ mmHg} \cdot \text{mm}^{-1}$). Cependant, la contractilité myocardique durant une anesthésie 1 MAC à l'halothane et à l'isoflurane était mieux conservée que durant une anesthésie à l'enflurane ($38.66 \pm 9.73 \text{ mmHg} \cdot \text{mm}^{-1}$; $p < 0.01$, $p < 0.05$).