Haemodynamic effects of atropine during halothane or isoflurane anaesthesia in infants and small children

David J. Murray MD, Robert B. Forbes MD, Judith B. Dillman MD, Larry T. Mahoney MD, David L. Dull MD

In this study, two-dimensional and pulsed Doppler echocardiography were used to measure cardiovascular changes before and after IV atropine in 31 infants and small children during halothane (n = 15) or isoflurane (n = 16) anaesthesia. Prior to induction of anaesthesia heart rate (HR), mean blood pressure (MBP), and two-dimensional echocardiographic dimensions of the left ventricle and pulmonary artery blood flow velocity were measured by pulsed Doppler echocardiography. Cardiovascular measurements were repeated while anaesthesia was maintained at 1.5 MAC halothane (n = 15) or isoflurane (n = 16). Atropine 0.02 mg·kg^{-1} IV was then administered and two minutes later, a third set of cardiovascular data was obtained. Heart rate decreased during halothane anaesthesia but did not change significantly during isoflurane anaesthesia. Mean blood pressure, cardiac output (CO) and stroke volume (SV) decreased similarly during 1.5 MAC halothane or isoflurane anaesthesia. Ejection fraction (EF) decreased and left ventricular end-diastolic volume (LVEDV) increased significantly in both groups, but decreases in EF (32 ± 5 per cent vs 18 ± 5 per cent) and increases in LVEDV (18 ± 7 per cent vs 7 ± 5 per cent) were significantly greater during halothane than during isoflurane anaesthesia. Following atropine, HR increased more in the patients maintained with halothane (31 ± 6 per cent), than during isoflurane anaesthesia (18 ± 5 per cent). Atropine increased CO in both groups of patients, but SV and EF remained unchanged. When compared with awake values, HR increased similarly and significantly (18 ± 4 per cent) following atropine in both groups, and CO returned to control levels. Halothane decreased EF and increased LVEDV more than isoflurane at 1.5 MAC end-expired anaesthetic levels. Atropine did not diminish the myocardial depression produced by halothane or isoflurane. The increase in CO following atropine during halothane and isoflurane anaesthesia in infants and small children is the result of increases in HR alone.

Key words

ANAESTHESIA: paediatric; ANAETHETICS, VOLATILE: halothane, isoflurane; HEART: echocardiography, myocardial function; MEASUREMENT TECHNIQUE: pulsed Doppler echocardiography, two-dimentional echocardiography.

From the Departments of Anesthesia, Pediatrics and Preventive Medicine, University of Iowa College of Medicine, Iowa City, IA.

Address correspondence to: Dr. D. Murray, Department of Anesthesia, University of Iowa Hospitals and Clinics, Iowa City, IA 52242.

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In children (mean age 6 yrs) IV atropine administered during halothane and nitrous oxide anaesthesia increased cardiac output (CO).[1] In previous studies of infants who received PO or IM atropine premedication, heart rate (HR) and mean blood pressure (MBP) have been reported to be greater than during halothane and nitrous oxide anaesthesia alone.[2,3] This suggests that atropine may attenuate the cardiovascular depression produced by halothane and nitrous oxide anaesthesia.[2,3] Whether these haemodynamic changes following atropine are solely the result of an increase in HR or are the result of changes in other myocardial determinants, as well as increases in HR, is unclear.[1,3]

Halothane and isoflurane produce dose-related direct myocardial depression that appears to be more profound in the young compared with the adult animal.[4,5] In an *in vitro* study using feline papillary muscle, the degree of myocardial depression produced by halothane and isoflurane appear similar, if compared at equianaesthetic levels.[6] In a previous clinical study of infants and small children that compared equianaesthetic concentrations of halothane and isoflurane, the haemodynamic differences between halothane and isoflurane in infants and children were mainly the result of differing effects on HR, as decreases in ejection fraction (EF), stroke volume index (SVI) and cardiac index (CI) were of similar

magnitude.[7] In older children, halothane decreased heart rate but also depressed myocardial determinants more than isoflurane.[8]

The purpose of this study was to determine whether atropine modified the myocardial depression produced by halothane and isoflurane by an effect other than HR and to establish whether the haemodynamic differences between halothane and isoflurane anaesthesia in infants and children are altered by the use of atropine.

Methods

After the protocol was approved by the hospital's Human Studies Committee and informed written consent was obtained from the parent(s), 31 ASA physical status I infants and small children who required elective surgery were studied.

Parents accompanied the unpremedicated infants and small children to a pre-surgical care unit where pre-induction HR, BP by automated oscillotonometry (Omega 1600®), and two-dimensional echocardiographic measuresements of left ventricular area and length in diastole, as well as pulsed Doppler measurements of pulmonary artery blood flow velocity, were determined for each infant prior to induction of anaesthesia.

The patients were alternately assigned to receive mask inhalation induction of anaesthesia with halothane (n = 15) or isoflurane (n = 16). In the operating room, anaesthesia was induced with halothane or isoflurane in N_2O:O_2 (3:2 $L \cdot min^{-1}$) using a semi-closed paediatric circle system. Monitoring included precordial stethoscope, mean blood pressure (MBP) (Omega 1600®), and heart rate (HR) by ECG. Inspired and expired gas concentrations were measured and recorded using a Perkin-Elmer® mass spectrometer.

Approximately 15 minutes following induction of anaesthesia, N_2O was discontinued and the O_2 flow increased to 5 $L \cdot min^{-1}$. Inspired halothane or isoflurane concentrations were adjusted to maintain end-expired concentrations of 1.5 MAC halothane or isoflurane (corrected for age)[9,10] as measured under a tight-fitting Rendell-Baker mask. With anaesthesia maintained at 1.5 MAC halothane or isoflurane, ten minutes after the discontinuation of N_2O a second set of cardiovascular data was recorded over a two-minute period. Atropine 0.02 $mg \cdot kg^{-1}$ IV was then administered and approximately two minutes later, a third set of cardiovascular data was collected with end–expired levels of halothane or isoflurane maintained at 1.5 MAC. All cardiovascular measurements were completed before tracheal intubation and the start of elective surgery. Ventilation was controlled throughout the study period (approximately 30 minutes) and IV fluids were withheld.

The echocardiography technician who recorded and measured all echocardiographic data was unaware of the volatile agent used to induce or maintain anaesthesia. Ultrasound studies were performed with patients in the supine position using an Ultra Imager 260D® (Biosound, Inc., Indianapolis) mechanical sector scanner with a 4 MHz single element transducer combined with a 3.5 MHz Doppler interrogation frequency. Short axis views at the high papillary muscle level and the great vessel level, and apical four-chamber views were obtained in each subject. Left ventricular cross-sectional area was measured at the level of the papillary muscles. Left ventricular cavity length was measured in the apical four-chamber view. Pulmonary artery diameter was measured immediately above the level of the semilunar valve.

After the heart and great arteries were imaged a Doppler sample volume was positioned within a 75° sector sampling arch.[11] A line on the sample volume cursor documented the flow angle estimated by the ultrasonographer and the velocity was then corrected (velocity/cos θ). The sample volume axial dimension was kept to 3 mm and the lateral width was constant at 1.5 mm. The sample volume was placed in the pulmonary artery immediately above the pulmonary valve and positioning for maximal flow velocity was confirmed by both the intensity of the audio signal and the spectral display of the Doppler shift frequency obtained from fast Fourier transformed spectral analysis. Peak velocity was measured to the top with the most dense signal on the velocity curve and 3 beats $\cdot min^{-1}$ averaged.[11–13] Continuously updated two-dimensional images, Doppler profiles and simultaneous electrocardiographic tracings were displayed on a monitor and recorded on video tape. Selective frozen images were recorded on a strip chart recorder for measurement.

Left ventricular endocardial enclosed volume was determined at end–diastole from two orthogonal planes: parasternal short axis and apical four-chamber views. Using a microsonic CAD-886 image processing and video quantifications system (Microsonics, Inc., Indianapolis) images were traced along the endocardium utilizing the leading edge method.[10,12–14]

The two-dimensional recordings of left ventricular area at the papillary muscle level and left ventricular length at end–diastole were used to calculate left ventricular end–diastolic volumes (LVEDV) at each study level.[13,14] Volume was calculated from:

$$\text{Volume} = 5/6 \text{ area} \times \text{length}$$[13–16]

This formula assumes the ventricular configuration to be a hemispheric cylinder. This measurement correlates with a more sophisticated algorithm calculation (Simpson's Rule) used to measure two-dimensional left ventricular volumes and also with angiographically determined left

ventricular volumes in various clinical situations including left ventricular overload.[16,17]

The Doppler determined mean velocity of pulmonary artery blood flow and echocardiographically determined pulmonary artery diameter were used to calculate CO[11,13] using the formula:

$$CO = \text{mean pulmonary blood flow velocity} \times \text{pulmonary artery area} \cdot \cos \theta^{-1}$$

Data were analyzed using a t-test to compare the ages and weights of infants and small children and a multiple factor repeated measures design to assess differences among the cardiovascular measures recorded prior to induction of anaesthesia at 1.5 MAC, and following IV atropine in each patient. Comparisons were also made between the two groups who received either halothane or isoflurane anaesthesia. Analysis of variance was performed to determine statistical significance and define interaction. When interaction was present, follow-up comparisons were made. Bonferroni adjustment was used to protect the overall error rates.[17] Statistical significance was accepted at $P < 0.05$. All values are expressed as mean ± SEM.

TABLE I Inspired and end-expired levels of halothane and isoflurane recorded at the time cardiovascular data were collected

Halothane	*End-Expired* (Vol%)*	*Insp% (Vol%)*
Before atropine	1.57 ± 0.06	1.84 ± 0.07
Following atropine	1.55 ± 0.05	1.82 ± 0.07
Isoflurane		
Before atropine	2.60 ± 0.06	3.10 ± 0.07
Following atropine	2.56 ± 0.05	3.02 ± 0.07

*1 MAC halothane 1–6 mos = 1.2 vol%; 6–24 mos = 1.0 vol%; 1 MAC isoflurane 1–6 mos = 1.8 vol%; 6–24 mos = 1.6 vol%.

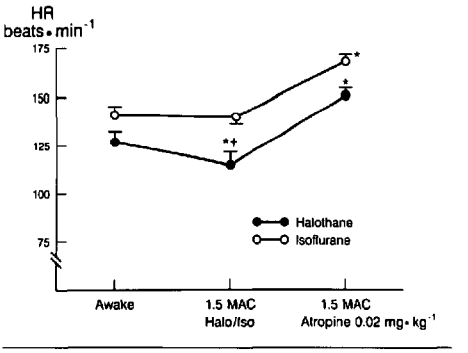

Figure 1 Heart rate (HR). Values are expressed as mean ± SEM. $P < 0.05$ from awake, $^{+}P < 0.05$ from isoflurane.

Results

The infants and small children in the two groups were of similar age (halothane 11.9 ± 2.3 months (3 mos–29 mos), isoflurane 8.9 ± 1.7 (2 mos–24 mos)) and weight (halothane 8.8 ± 0.6 kg, isoflurane 7.3 ± 0.6 g).

Inspired and expired anaesthetic gas concentrations are reported in Table I. The mean end-expired CO_2 recorded during the study period was 31.1 ± 1.7 mmHg.

Heart rate and MBP decreased from awake values during 1.5 MAC halothane anaesthesia (Figure 1, Table II). In the isoflurane group, MBP decreased but HR was not significantly different from awake levels. Following atropine, both HR (31 ± 6 per cent) and MBP (11 ± 4 per cent) increased in the infants and children maintained with halothane (Figure 1, Table II). In the group maintained with isoflurane, HR increased (18 ± 6 per cent) but MBP was unchanged (Figure 1, Table II). When compared with

TABLE II Changes in heart rate, mean blood pressure and left ventricular end-diastolic volume (LVEDV)

	Awake	*1.5 MAC*	*1.5 MAC + atropine*
Heart rate (beats·min^{-1})			
Halothane	127 ± 5.5	115 ± 6.0*†	151 ± 4.0†
Isoflurane	141 ± 5.0	140 ± 1.5	168 ± 3.0†
Mean blood pressure (mmHg)			
Halothane	74.3 ± 1.9	55.4 ± 1.5*	62.5 ± 2.7*†‡
Isoflurane	73.5 ± 2.4	57.8 ± 2.6*	59.4 ± 3.1*
LVEDV (ml)			
Halothane	11.9 ± 1.2	14.0 ± 1.1*†	14.4 ± 1.3*†
Isoflurane	9.5 ± 0.6	10.2 ± 0.8*	9.8 ± 0.8‡

Results are expressed as mean ± SEM.
*$P < 0.05$ from awake measurement.
†$P > 0.05$ from isoflurane.
‡$P < 0.05$ from prior to administration of atropine.

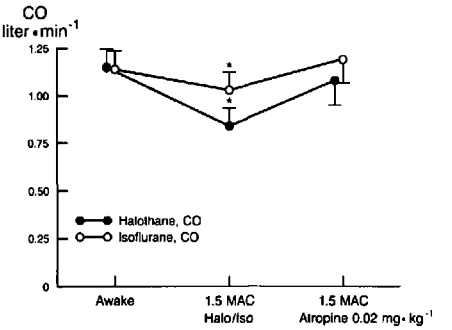

Figure 2 Cardiac output (CO). Values are expressed as mean ± SEM. *P < 0.05 from awake.

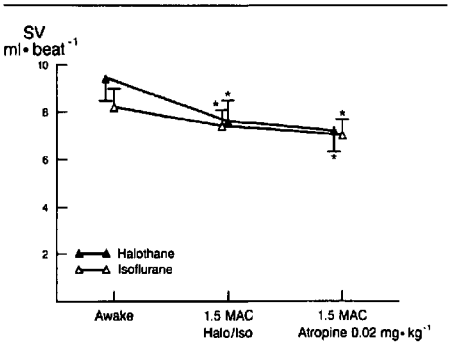

Figure 3 Stroke volume (SV). Values are expressed as mean ± SEM. *P < 0.05 from awake.

awake levels, atropine increased HR (18 ± 4 per cent) similarly in both patient groups (Figure 1, Table II).

Cardiac output and SV decreased significantly during halothane and isoflurane anaesthesia (Figure 2 and Figure 3, Table II). Following 0.02 mg·kg^{-1} atropine, CO increased significantly in both groups and was similar to awake measurements. While HR and CO increased, SV did not change significantly in either group of infants following atropine (Figure 2). Ejection fraction also decreased from awake values during both halothane and isoflurane anaesthesia, 32 ± 5 per cent and 18 ± 7 per cent respectively (Figure 4). Following atropine, EF did not change significantly in either the halothane or isoflurane group (Figure 4). Left ventricular end–diastolic volume increased significantly from awake values following induction of anaesthesia with both halothane and

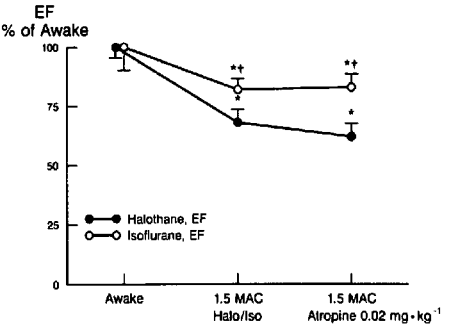

Figure 4 Per cent of awake ejection fraction (EF). Values are expressed as mean ± SEM. *P < 0.05 from awake, †P < 0.05 from halothane.

isoflurane. The increase in LVEDV was greater during halothane anaesthesia. Following administration of atropine, LVEDV remained unchanged in the halothane group, but returned to awake levels in the isoflurane group.

Discussion

The equianaesthetic end-expired levels of halothane or isoflurane[9,10] were measured from under a tight fitting mask during controlled ventilation. Inspired anaesthetic gas can be entrained from apparatus dead space (anaesthesia mask and connector) during the measurement. This problem is magnified in infants and small children with small tidal volumes and rapid respiratory rate.[18,19] Mixing of inspired gas could lead to an overestimation of the actual end–tidal levels. For this reason, we controlled ventilation with respiratory rates less than 30 breaths per minute and tidal volumes of 10–15 ml·kg^{-1}. Capnograms and measured anaesthetic concentrations were recorded when an alveolar plateau on the capnogram confirmed an adequate end–tidal sample had been obtained. For this reason, moderate hyperventilation and hypocarbia (31 ± 4 mm) occurred during the study period. This helped provide more reliable estimates of actual end–expired anaesthetic levels.[18,19] The estimates of end–tidal halothane and isoflurane concentrations made in this manner may not be accurate even when an alveolar plateau on the capnogram reflects an accurate alveolar sample has been recorded, particularly with a long dwell line connection to a multiplexed mass spectrometer.[20]

We used two-dimensional echocardiography to measure end–diastolic volumes. These volumes correlate

with, but tend to underestimate, invasive angiographic volumes.[13–16] The end-diastolic volume and end–systolic volume measured by two-dimensional echocardiography can be used to determine SV and CO. However, the underestimation of left ventricular volumes can lead to large errors when SV is measured in this manner. For this reason, pulsed Doppler estimates of the mean pulmonary artery velocity were used to measure CO and SV.[12,13,21,22] A pulsed Doppler technique used to measure the frequency shift of reflected sound waves from blood flow in the pulmonary artery can be used to quantitate the velocity of blood flow.[21,22] With trained personnel using video quantification systems CO and SV determined in this manner correlate with invasive angiographic methods in infants and children (> 0.92).[21,22]

In this study HR was similar to awake levels at 1.5 MAC isoflurane anaesthesia. In previous clinical studies of infants and children during isoflurane anaesthesia HR has been reported to be increased or similar to awake levels.[8,9] Many factors alter the heart rate during inhalational anaesthesia: a decrease occurs when nitrous oxide is substituted for either halothane or isoflurane in infants and small children.[23] The haemodynamic effects of halothane and isoflurane were measured ten minutes following the discontinuation of N_2O which could have altered the chronotropic effects of halothane and isoflurane which were recorded later.

We found that halothane increased preload (LVEDV) and decreased EF more than isoflurane at equianaesthetic levels but both drugs had similar effects on SV and CO. Whether these haemodynamic differences indicate that halothane decreases contractility more than isoflurane or whether this represents a difference between halothane and isoflurane on afterload could not be determined by the pulsed Doppler and two-dimensional echocardiographic technique used in this study. In a previous study of infants and small children the only significant differences between equianaesthetic halothane and isoflurane were in HR.[7] Ejection fraction, cardiac index (CI) and stroke volume index (SVI) decreased similarly and LVEDV increased a similar amount during halothane and isoflurane anaesthesia.[7] In this study, we compared the haemodynamic effects of greater end–expired concentrations (1.5 MAC vs 1.25 MAC) and also maintained a longer period of stable end–expired levels than in the earlier study.[7] Because of the greater solubility of halothane than isoflurane, a shorter period of stable end–tidal levels could result in lower myocardial (vessel rich) concentrations of halothane than isoflurane. This may explain why in this study, unlike the previous study, we found greater decreases in EF and increases in LVEDV with halothane than isoflurane. Despite these differences between halothane and isoflurane in EF and LVEDV, the decreases in CO and SV during halothane and isoflurane anaesthesia were of similar magnitude.

The addition of atropine attenuated the differential effect of 1.5 MAC halothane or isoflurane on HR. Heart rate increased similarly from awake values in both groups (18 ± 4 per cent) but the increase from the 1.5 MAC anaesthesia level was significantly greater in the halothane group (31 ± 6 per cent) than in the isoflurane group (18 ± 6 per cent). Despite the effect of atropine in balancing HR, halothane still produced greater increases in preload (LVEDV) and decreases in EF than isoflurane. This suggests the differences in cardiovascular effect of equianaesthetic concentrations of halothane and isoflurane in children are not related to an effect on HR alone.

In a previous study of older premedicated children (mean age six years), when atropine was administered during N_2O and halothane anaesthesia, CO increased to levels greater than control values measured prior to induction of anaesthesia.[1] In this study of unpremedicated infants and small children, mean age = 10 months, atropine increased HR resulting in a return of CO to awake values. Similar to previous studies of PO or IM atropine premedication, in the halothane group, HR and MBP were greater following IV atropine;[2,3] but this was not associated with an improvement in EF or SV.

Atropine is recommended to prevent the vagal responses that frequently occur in infants and children during airway manipulation. Also, it may be useful to prevent or treat the decrease in HR during inhalational induction of anaesthesia which may occur more frequently in children when narcotic premedication or nitrous oxide are used in combination with volatile anaesthetics or when higher concentrations of volatile anaesthetic are administered.[1–3,23–25]

In summary the use of atropine diminishes the differences in HR that exist during halothane and isoflurane anaesthesia in paediatric patients. In addition, it augments CO by its effect on HR. Atropine does not influence other differences in cardiovascular function that occur during inhalational anaesthesia and does not attenuate the myocardial depression produced by either halothane or isoflurane in infants and small children.

References

1 *Barash PG, Glanz S, Katz JD, Taunt K, Talner NS.* Ventricular function in children during halothane anesthesia. Anesthesiology 1978; 49: 79–85.

2 *Miller RB, Friesen RH.* Oral atropine premedication infants attenuates cardiovascular depression during halothane anesthesia. Anesth Analg 1988; 67: 180–5.

3 *Friesen RH, Lichtor JL.* Cardiovascular depression during halothane anesthesia in infants: a study of three induction techniques. Anesth Analg 1982; 61: 42–5.

4 *Boudreaux JP, Schieber RA, Cook DR.* Hemodynamic effects of halothane in the newborn piglet. Anesth Analg 1984; 63: 731–7.

5 *Schieber RA, Namnoun A, Sugden A, Shin EK, Orr RA, Cook DR.* Hemodynamic effects of isoflurane in the newborn piglet: comparison with halothane. Anesth Analg 1986; 65: 633–8.

6 *Wolf WJ, Neal MB, Matthew BP, Bee DE.* Comparison of the in vitro myocardial depressant effects of isoflurane and halothane anaesthesia. Anesthesiology 1988; 69: 660–6.

7 *Murray DJ, Vandewalker G, Matherne GP, Mahoney LT.* Pulsed doppler and two-dimensional echocardiography: comparison of halothane and isoflurane on cardiac function in infants and small children. Anesthesiology 1987; 67: 211–7.

8 *Wolf WJ, Neal NB, Peterson MD.* The hemodynamic and cardiovascular effect of isoflurane and halothane in children. Anesthesiology 1986; 64: 328–33.

9 *Gregory GA, Eger EI, Munson ES.* The relationship between age and halothane requirement in man. Anesthesiology 1969; 30: 488–91.

10 *Cameron CB, Robinson S, Gregory GA.* The minimum anesthetic concentrations of isoflurane in children. Anesth Analg 1984; 63: 418–20.

11 *Goldberg SJ, Sahn DJ, Allen HD, Valdes-Cruz LM, Hoenecke H, Carnahan Y.* Evaluation of pulmonary and systemic blood flow by 2-dimensional echocardiography using fast Fourier transform spectral analysis. Am J Cardiol 1982; 50: 1392–400.

12 *Mahoney LT, Coryell KG, Lauer RM.* The newborn transitional circulation: a two-dimension Doppler echocardiographic study. J Am Coll Cardiol 1985; 6: 623–29.

13 *Disessa TG, Friedman WF.* Echocardiographic evaluation of cardiac performance. Cardiol Clin 1983; 1: 487–99.

14 *Schnittger I, Gordon EP, Kritz-Gerald PJ, Pop RL.* Standardized intracardiac measurements of two dimensional echocardiography. J Am Coll Cardiol 1983; 2: 934–8.

15 *Mercier JC, Disessa TG, Jarmakani JM.* Two dimensional echocardiographic assessment of left ventricular volumes and ejection fraction in children. Circulation 1982; 65: 962–9.

16 *Tortoledo FA, Quinones MA, Fernandez GC, Waggoner AD, Winters WL.* Quantification of left ventricular volumes by two dimensional echocardiography: a simplified and accurate approach. Circulation 1983; 67: 579–84.

17 *Winer BJ.* Stastistical Principle Design. 2nd Edition. New York, McGraw-Hill, 1971, pp. 185–96, 301–5.

18 *Sasse FJ.* Can we trust end-tidal carbon dioxide measurements in infants? J Clin Monit 1985; 1: 147–8.

19 *Schieber RA, Namnouma A, Sugden A, Saville AL, Orr RA.* Accuracy of expiratory carbon dioxide measurements using the coaxial and circle breathing circuits in small subjects. J Clin Monit 1985; 1: 149–55.

20 *Scamman FL.* High frequency response of a central mass spectrometer. Anesthesiology 1987; 67: A176.

21 *Vieli A.* Doppler flow determination. Br J Anaesth 1988; 60: 1075–84.

22 *Cahalan MK, Litt L, Botvinick EH, Schiller NB.* Advances in noninvasive cardiovascular imagining: implication for the anesthesiologist. Anesthesiology 1987; 66: 356–74.

23 *Murray DJ, Forbes RB, Murphy K, Mahoney LT.* Nitrous oxide: cardiovascular effects in infants and small children during halothane and isoflurane anesthesia. Anesth Analg 1988; 67: 1059–64.

24 *Hickey PR, Hansen DD, Strafford M, Thompson JE, Jonas RE, Mayer JE.* Pulmonary and systemic hemodynamic effects of nitrous oxide in infants with normal and elevated pulmonary vascular resistance. Anesthesiology 1986; 65: 374–8.

25 *Friesen RH, Lichtor JL.* Cardiovascular effects of inhalation induction with isoflurane in infants. Anesth Analg 1983; 62: 411–4.

Résumé

Nous avons utilisé un appareil à échocardiographie bi-dimensionnelle couplé à un Doppler pulsé chez des bébés et de jeunes enfants pour évaluer l'impact hémodynamique de l'halothane (n = 15) et de l'isoflurane (n = 16) et la modification possible de ces effets par l'atropine. Nous avons mesuré la fréquence cardiaque (FC), la pression artérielle moyenne (PAM), la dimension de la cavité ventriculaire gauche (par écho bi-dimensionnelle) et la vélocité du flot sanguin pulmonaire (par Doppler) et ce, en trois occasions soit avant l'induction, après l'instauration de 1.5 MAC d'halothane ou d'isoflurane et finalement, deux minutes après l'injection IV de 0.02 mg·kg^{-1} d'atropine. On ne nota une baisse de la fréquence cardiaque qu'avec l'halothane tandis que la PAM, le débit cardiaque (DC) et le volume d'éjection (VE) diminuaient autant avec l'un ou l'autre anesthésique. La diminution de la fraction d'éjection (FE) et l'augmentation du volume télédiastolique du ventricule gauche (VTDVG) significatives pour les deux groupes, étaient plus marqué avec l'halothane qu'avec l'isoflurane: FE 32 ± 5 pour cent vs 18 ± 5 pour cent; VTDVG 18 ± 7 pour cent vs 7 ± 5 pour cent. Avec l'atropine, la FC monta plus dans le groupe halothane (31 ± 6 pour cent) que dans le groupe isoflurane (18 ± 5 pour cent), le DC augmentant dans les deux groupes, alors que le VE et la FE demeuraient inchangés. Comparée aux mesures pré-induction, l'atropine amenait une hausse significative de la FC, semblable dans les deux groupes (18 ± 4 pour cent) et restaurait le DC. Donc, chez les bébés et les jeunes enfants, à 1.5 MAC, l'halothane diminue la FE et augmente le VTDVG plus que ne le fait l'isoflurane. L'atropine ne modifie pas la dépression myocardique et elle ne restaure le DC que par une hausse de la FC.