# Drive and timing components of respiration in young children following induction of anaesthesia with halothane or ketamine

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*Timing and drive components of respiration were studied in 18 young children following induction of anaesthesia with ketamine and were compared with results from ten children following induction of anaesthesia with halothane. During one minute of quiet breathing, signals from a pneumotachograph attached to the anaesthetic mask were analysed for tidal volume (Vt), respiratory frequency (f), minute volume (V̇e), inspiratory and expiratory times (Ti, Te) and flow patterns. Following induction of anaesthesia with ketamine, children breathed more slowly and deeply than children receiving halothane, but there was no significant difference in V̇e or in Vt/Ti, suggesting that respiratory drive was similar in the two groups of children. In the children receiving ketamine, Ti was more than twice as long, and thus the ratio Ti/Te was significantly increased, in comparison with the group receiving halothane. In addition to the prolonged Ti in the children induced with ketamine, there was a more rapid increase in volume in early inspiration than in late inspiration, which is an apneustic breathing pattern. There was a slower decrease in volume in early expiration, with occasional early expiratory breath holding lasting up to three seconds, in the ketamine-induced children. The unique breathing pattern demonstrated with ketamine, consisting of large Vt, increased Ti/Te ratio, apneustic inspiratory pattern, and expiratory braking, contributed to an increased mean lung volume above functional residual capacity, of 2.40 ml·kg$^{-1}$ body weight, in comparison to 1.27 ml·kg$^{-1}$ in the children receiving halothane.*

**Key words**

ANAESTHETICS, INTRAVENOUS: ketamine; ANAESTHETICS, INHALATION: halothane; ANAESTHESIA: paediatric.

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The breathing cycle is controlled largely by two mechanisms, the first drives inspiration, and the second controls respiratory timing, the inspiratory on-off switch.[1] Inspiratory drive may be measured by deriving mean inspiratory flow which is tidal volume (Vt) divided by inspiratory time (Ti), Vt/Ti. The timing mechanism is represented by the ratio of Ti to expiratory time (Te), Ti/Te, or to total cycle time (Ttot), Ti/Ttot. Minute ventilation (V̇e) can be expressed as the product of the drive and timing components:

$$\dot{V}e = Vt \times f = (Vt/Ti) \times (Ti/Ttot)$$

where f is the respiratory frequency.

In adults Ti/Ttot is increased during the initial 15 min following the discontinuation of a ketamine infusion.[2] Ketamine anaesthesia in cats causes an "apneustic" pattern of breathing in which there is an initial rapid rise in volume during early inspiration, a slower rise or a pause in late inspiration and a marked increase of the Ti/Ttot ratio.[3] This results in a larger mean lung volume above functional residual capacity (FRC) during inspiration. We questioned whether the effects of ketamine on respiratory timing in the above animal and human studies could be demonstrated in pre-school children.

There are conflicting reports on the effect of ketamine on respiratory drive, with some authors reporting that ketamine is a respiratory stimulant[2,4] and others demonstrating respiratory depression[3] with hypoxaemia[5] during ketamine anaesthesia. Analysis of the drive component of respiration during ketamine anaesthesia, in doses that have been shown to provide surgical anaesthesia, may provide information contributory to this question.

In this study we examined the drive and timing components of respiration and mean lung volume during tidal breathing in pre-school children following induction of anaesthesia with ketamine and with halothane.

## Methods

The study subjects were 28 children, 17 boys and 11 girls,

TABLE I Anthropometric variables. Mean (range)

| *n* | *Ketamine group* *18* | *Halothane group* *10* |
|---|---|---|
| Height (cm) | 105 (66–124) | 106 (80–122) |
| Weight (kg) | 18.0 (6.8–24.5) | 18.6 (12.0–25.5) |
| Age (months) | 56 (9–83) | 53 (17–80) |

whose anthropometric data are summarized in Table I. They were scheduled to undergo elective surgery not associated with the cardiorespiratory system. All were ASA physical status class I, and none had cardiorespiratory problems at birth. The protocol received approval from the Medical Ethics Committee of the hospital, and informed consent was obtained from the parents of each child.

The anaesthetic techniques were similar to those used in a previous study of the effect of ketamine anaesthesia on the end expiratory lung volume in young children.[6] Following premedication with triclofos 70 mg·kg$^{-1}$ body weight, 18 subjects received intravenous ketamine 1–2 mg·kg$^{-1}$ over 15 sec followed by a continuous infusion of 0.1 per cent ketamine. The total ketamine dose administered during the study period was 0.05–0.2 mg·kg$^{-1}$·min$^{-1}$, which was sufficient to prevent reaction to the test procedure and to the subsequent surgical stimulus, and is consistent with the dosage required to establish surgical anaesthesia in previous reports.[7–9] These subjects spontaneously breathed room air during the test procedure.

In the remaining ten children, anaesthesia was induced by spontaneous breathing of halothane in oxygen via a Jackson-Rees modification of the Ayre's T-piece. Sufficient anaesthetic depth was obtained to prevent reaction to the insertion of an intravenous cannula, and to the test procedure and subsequent surgery. Care was taken to prevent distention of the anaesthetic bag in order to avoid inadvertent application of continuous positive airway pressure.

For the measurement of respiratory volumes and timing, a Rendall-Baker face mask was sealed to the child's face with soft silicon putty. A screen penumotachograph (Mercury Electronics, Scotland, type F2, 19 mm), which had resistance of 0.9 cm $H_2O \cdot L^{-1} \cdot sec^{-1}$ and linear flow-pressure characteristics up to a flow of 1.8 $L \cdot sec^{-1}$, was attached to the face mask in the group receiving ketamine, and was inserted between the face mask and the anaesthetic circuit in the subjects receiving halothane. The two pressure outputs from the penumotachograph were connected to a differential pressure transducer (Validyne MP45) and the signal was amplified (Hewlett Packard Amplifier 8805C) and integrated (Hewlett Packard Integrator 8815A). The resulting flow and volume signals were displayed on an oscilloscope (Tektronix T935A) and recorded on magnetic tape (Hewlett Packard Instrumentation Recorder 3964A) during at least one minute of quiet breathing. Zero flow was recorded at the end of this period by briefly occluding the exit port of the pneumotachograph. In the group receiving halothane, this entailed briefly removing the anaesthetic circuit from the penumotachograph in order to ensure that there was no gas flow.

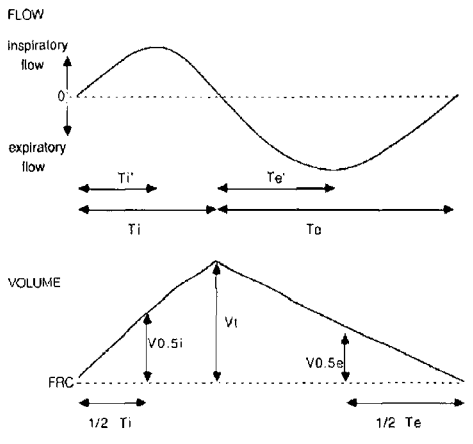


FIGURE 1 Schematic diagram of the flow and volume signals showing the method of measurement of the following parameters: Ti – inspiratory time, Te – expiratory time, Ti' – time to peak inspiratory flow, Te' – time to peak expiratory flow, Vt – tidal volume, V0.5i – volume after one-half Ti has elapsed, V0.5e – volume after one-half Te has elapsed.

The signals were subsequently played on to a paper recorder and analysed manually. Every second breath during one minute of quiet breathing was analysed. Mean Vt and f were measured from the volume recording (Figure 1), which was the integral of the flow signals. The volumes at one-half Ti (V0.5i), and one-half Te (V0.5e) were measured (Figure 1) and expressed as their ratio with Vt. Ti and Te were the time intervals between instants of zero flow during inspiration and expiration respectively. The time interval from the beginning of inspiratory flow to peak inspiratory flow (Ti') was measured (Figure 1) and expressed as its ratio with Ti (Ti'/Ti). The comparable parameters in expiration, Te' and Te'/Te, were also determined (Figure 1).

The volume measurements for the subjects in the halothane group were corrected for the difference produced in the pneumotachograph signal by the greater viscosity of the halothane-oxygen mixture than that of room air in order to compare the results from the two groups of children. The correction factor was obtained by the method described by Hobbes.[10]

TABLE II Respiratory volumes and timing

| *Subject* | $\frac{Vt}{(ml \cdot kg^{-1})}$ | $\frac{f}{(min)}$ | $\frac{Ti}{(s)}$ | $\frac{Te}{(s)}$ | $\frac{Ti'}{Ti}$ | $\frac{Te'}{Te}$ | $\frac{V0.5i}{Vt}$ | $\frac{V0.5e}{Vt}$ |
|---|---|---|---|---|---|---|---|---|
| *Ketamine group* | | | | | | | | |
| 1 | 8.24 | 31 | 0.87 | 1.04 | 0.44 | 0.44 | 0.51 | 0.53 |
| 2 | 8.79 | 26 | 0.92 | 1.41 | 0.44 | 0.19 | 0.61 | 0.42 |
| 3 | 7.30 | 30 | 1.12 | 1.12 | 0.39 | 0.45 | 0.50 | 0.44 |
| 4 | 8.58 | 29 | 0.90 | 1.15 | 0.31 | 0.50 | 0.60 | 0.59 |
| 5 | 7.13 | 31 | 1.00 | 0.93 | 0.45 | 0.58 | 0.54 | 0.48 |
| 6 | 10.61 | 22 | 1.14 | 1.58 | 0.32 | 0.52 | 0.64 | 0.61 |
| 7 | 6.82 | 34 | 0.82 | 0.94 | 0.43 | 0.72 | 0.54 | 0.60 |
| 8 | 6.35 | 22 | 1.65 | 1.10 | 0.40 | 0.35 | 0.56 | 0.50 |
| 9 | 6.48 | 29 | 0.84 | 1.26 | 0.37 | 0.24 | 0.57 | 0.46 |
| 10 | 10.67 | 41 | 1.39 | 1.67 | 0.55 | 0.33 | 0.47 | 0.45 |
| 11 | 6.05 | 21 | 1.54 | 1.26 | 0.37 | 0.69 | 0.51 | 0.57 |
| 12 | 8.77 | 27 | 1.13 | 1.11 | 0.40 | 0.40 | 0.55 | 0.52 |
| 13 | 8.57 | 21 | 1.24 | 1.64 | 0.51 | 0.36 | 0.47 | 0.54 |
| 14 | 10.63 | 23 | 1.31 | 1.33 | 0.27 | 0.23 | 0.58 | 0.41 |
| 15 | 8.98 | 21 | 1.39 | 1.44 | 0.24 | 0.51 | 0.68 | 0.53 |
| 16 | 8.49 | 20 | 1.42 | 1.58 | 0.40 | 0.60 | 0.55 | 0.52 |
| 17 | 6.14 | 31 | 0.83 | 1.10 | 0.55 | 0.60 | 0.49 | 0.50 |
| 18 | 7.06 | 21 | 1.57 | 1.35 | 0.31 | 0.42 | 0.54 | 0.61 |
| Mean | 8.09 | 26 | 1.17 | 1.28 | 0.40 | 0.45 | 0.55 | 0.52 |
| SD | 1.53 | 5 | 0.28 | 0.24 | 0.09 | 0.15 | 0.06 | 0.06 |
| *Halothane group* | | | | | | | | |
| 1 | 5.23 | 45 | 0.59 | 0.74 | 0.54 | 0.32 | 0.54 | 0.42 |
| 2 | 3.31 | 39 | 0.54 | 1.00 | 0.43 | 0.14 | 0.51 | 0.42 |
| 3 | 5.42 | 48 | 0.48 | 0.78 | 0.44 | 0.18 | 0.53 | 0.43 |
| 4 | 3.84 | 38 | 0.57 | 0.99 | 0.60 | 0.15 | 0.36 | 0.26 |
| 5 | 5.66 | 40 | 0.53 | 0.96 | 0.46 | 0.34 | 0.54 | 0.47 |
| 6 | 5.44 | 31 | 0.79 | 1.15 | 0.58 | 0.22 | 0.43 | 0.27 |
| 7 | 3.45 | 43 | 0.57 | 0.84 | 0.48 | 0.22 | 0.48 | 0.31 |
| 8 | 5.14 | 34 | 0.74 | 1.04 | 0.36 | 0.25 | 0.54 | 0.33 |
| 9 | 6.63 | 31 | 0.67 | 1.24 | 0.45 | 0.22 | 0.49 | 0.29 |
| 10 | 4.28 | 34 | 0.75 | 1.03 | 0.63 | 0.23 | 0.48 | 0.24 |
| Mean | 4.84* | 38* | 0.62* | 0.98* | 0.50* | 0.23* | 0.49* | 0.34* |
| SD | 1.08 | 6 | 0.11 | 0.16 | 0.09 | 0.06 | 0.06 | 0.08 |

*Significantly different from the ketamine group ($\alpha < 0.05$).

The Mann-Whitney U-test was used for comparison of the measurements between the two groups. A level of $\alpha < 0.05$ was considered to indicate a significant difference. The breath by breath variability of the measurements within each subject was assessed by calculating the coefficient of variation (CV, standard deviation divided by the mean).

## Results

Respiratory volumes and timing are presented in Table II for the children anaesthetized with ketamine and halothane. The ketamine-induced children breathed more slowly and more deeply than those in the group anaesthetized with halothane (Figures 2 and 3, Table II). Mean weight-corrected Vt was significantly greater in the children anaesthetized with ketamine. However, f was

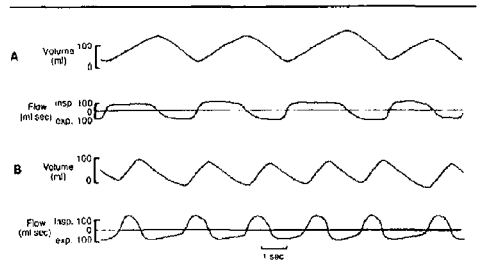


FIGURE 2 Pattern of breathing during anaesthesia showing the volume and flow signals from, A: subject 11 from the ketamine group, and B: subject 3 from the halothane group. Note the greater Vt and prolonged Ti and Te' in A compared with B.

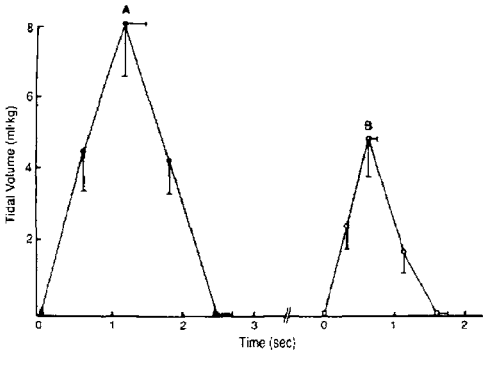


FIGURE 3 Schematic diagram showing the mean values of Vt, Ti, Te, V0.5i and V0.5e for the respiratory cycle in the ketamine and halothane groups. The bars indicate one SD.

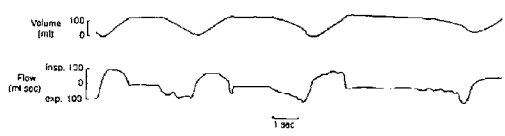


FIGURE 4 Volume and flow signals from a subject anaesthetized with ketamine demonstrating inspiratory pauses. This subject was not included in the study group.

increased in the subjects receiving halothane, and thus mean weight-corrected $\dot{V}e$ was not significantly different between the two groups ($207 \pm 42$ ml · min$^{-1}$ · kg$^{-1}$ for the children receiving ketamine and $184 \pm 45$ ml · min$^{-1}$ · kg$^{-1}$ for the children receiving halothane). Mean Ti and Te were both significantly greater in the children receiving ketamine (Table II, Figure 3) but Ti was almost two times greater in this group and thus mean Ti/Te was significantly greater in the group receiving ketamine ($0.93 \pm 0.22$) than in the group receiving halothane ($0.64 \pm 0.09$). Ti′/Ti was significantly less and V0.5i/Vt was significantly greater in the group receiving ketamine than in the children receiving halothane (Table II). End inspiratory pauses were observed in children receiving ketamine, but not in those receiving halothane. The longest end inspiratory pauses were observed in a child who received ketamine but who was not included in the

TABLE III Mean intra-individual coefficients of variation (%)

| | *Vt* | *Ti* | *Te* | *Ti′/Ti* | *Te′/Te* | *V0.5i/Vt* | *V0.5e/Vt* |
|---|---|---|---|---|---|---|---|
| Ketamine | 11.2 | 12.1 | 11.9 | 26.9 | 36.7 | 10.8 | 11.1 |
| Halothane | 8.3 | 6.1 | 8.0 | 11.4 | 16.9 | 9.3 | 10.4 |

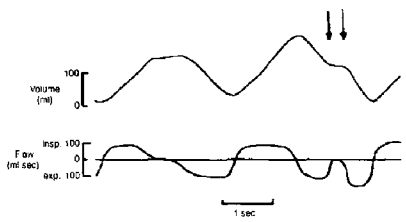


FIGURE 5 Volume and flow signals from subject 12 from the ketamine group, demonstrating a brief interruption of flow, marked by arrows, early in expiration.

study group when the attending anaesthetist stopped the ketamine infusion prematurely (Figure 4). Although breath holding of this type was of concern, no change in the clinical status of any patient occurred as a result of this breathing pattern.

Expiratory flow pattern was notably different between the two groups (Figure 2). The expiratory flow during halothane anaesthesia was characterized by an early peak flow, while in the ketamine group expiratory flow was uneven and peak flow often occurred late in expiration. There were occasional interruptions of expiratory flow in early expiration with a "step" noted on the volume trace in the children anaesthetized with ketamine (Figure 5). Mean Te′/Te and V0.5e/Vt were significantly greater in the children anaesthetized with ketamine than in the group receiving halothane (Table II) indicating "braking" of expiratory flow during ketamine anaesthesia.

Mean intra-individual CV for all measurements was greater in the group receiving ketamine than in those receiving halothane (Table III). Especially variable were the times to peak inspiratory and expiratory flows (Ti′/Ti and Te′/Te).

Mean lung volume above FRC, calculated from the area under the curve of Figure 3, for the ketamine-induced subjects (2.40 ml · kg$^{-1}$) was almost two times greater than that for the children receiving halothane (1.27 ml · kg$^{-1}$).

There was no significant difference in Vt/Ti between the group receiving ketamine ($123.7 \pm 40$ ml · sec$^{-1}$) and the group receiving halothane ($141.8 \pm 30.7$ ml · sec$^{-1}$) nor in the weight-corrected Vt/Ti ($6.87 \pm 1.73$ ml · sec$^{-1}$ · kg$^{-1}$ vs $7.92 \pm 2.08$ ml · sec$^{-1}$ · kg$^{-1}$ respectively).

## Discussion

The respiratory pattern we have found in children following induction of anaesthesia with ketamine consists of slow deep breathing with maintenance of a higher mean lung volume during the respiratory cycle and a high Ti/Te ratio in comparison to similar children anaesthetized with

halothane. The changes in respiratory rate and tidal volume induced by anaesthesia with ketamine and halothane have been previously documented. During halothane anaesthesia, a decrease in Vt, and increase in f, and a net decrease in Ve have been demonstrated.[11] Our results (Table II) are in agreement with this study. Ketamine causes increased Vt[8] with variable effects on respiratory rate with both tachypnoea[8] and bradypnoea with initial apnoea[5] having been reported. We noted end inspiratory breath holding which may appear to be apnoea, though it is less detrimental to gas exchange than true end expiratory apnoea.

An important methodological problem we faced in this study was the difficulty in comparing equivalency of the doses of an inhaled anaesthetic agent with an intravenously administered agent. The signs of anaesthetic depth are different with halothane and ketamine, and do not assist in determining dosage equivalency. We were able to compare the reaction to stimulation in the children from the two groups and noted that, at the doses used, there was no response to the test procedure, and that the anaesthesia was appropriate for the subsequent surgical stimulation. The rate of infusion of ketamine was consistent with previous studies of the dosage of ketamine required for surgical anaesthesia.[7,9] However, this does not establish precise dosage equivalency.

In spite of this limitation, the differences noted in respiratory volumes and timing between the groups were sufficiently marked to indicate that they were agent-specific effects. This was especially true of the differences in the Ti/Te ratio which was almost 50 per cent greater in children following induction of anaesthesia with ketamine than in children of similar age receiving halothane. Awake school-age children studied with a bellows pneumograph placed around the chest have a Ti/Te of 0.59.[12] This is similar to Ti/Te for the group receiving halothane anaesthesia in the present study and considerably smaller than the Ti/Te ratio from the group receiving ketamine. Therefore, it would appear that ketamine caused an increase in Ti/Te in these children. These results are consistent with the animal studies of Jaspar *et al.*[3] Morel *et al.*[2] studied the effect of ketamine on respiratory timing in adult volunteers and found that Ti/Te significantly increased during the first 15 minutes after 1 $mg \cdot kg^{-1}$ of ketamine was infused over five minutes. It is not clear whether these time-related changes were due to changing drug levels of ketamine with redistribution or to end-organ effects. The present study was completed during a constant infusion of ketamine in doses that have been found to give surgical levels of anaesthesia.[7,9] Since the children in this study were awaiting elective surgical procedures, which could influence the breathing pattern, the measurements were not repeated after these post-induction studies. It is possible that there are time-related changes in respiratory volumes or timing subsequent to induction with a constant infusion of ketamine or with surgical stimulation, and this awaits further study.

Ketamine is the only anaesthetic agent associated with increased Ti/Te. Intravenous anaesthetic agents either have no effect on Ti/Te, such as thiopental,[13] or decrease the ratio of Ti/Te, such as althesin and gamma hydroxybutyric acid,[13] droperidol and dextromoramide,[14] fentanyl and phenoperidone.[15] $N_2O$ and halothane anaesthesia in children causes a reduction in the Ti/Te ratio from the preanaesthetic value.[16]

The clinical significance of the unique respiratory pattern seen during ketamine anaesthesia lies in the short Te relative to Ti, which may result in inadequate time for complete lung emptying during expiration. Figure 2 shows a rapid decrease in expiratory flow at FRC in the child receiving ketamine, suggesting that inspiration interrupted the expiratory flow of the previous breath before expiration was completed. Braking of early expiratory flow in combination with the relatively shortened Te may result in gas remaining in the lungs at end expiration, effectively elevating FRC. A similar expiratory flow pattern and elevation of FRC is seen in ill infants and children who breathe rapidly, thus reducing Te, or in those who show expiratory flow braking from increased airway resistance.[17,18] This mechanism for elevation of FRC above the resting end expiratory lung volume may be the explanation for the absence of a deleterious effect of ketamine on FRC,[6] whereas all other commonly used anaesthetic agents are associated with an immediate and marked decrease in FRC.

Additionally, braking and occasional interruption of expiratory flow in early expiration seen with ketamine (Figures 2 and 5) and represented by prolonged Te'/Te and increased V0.5e/Vt in comparison to the group receiving halothane, represents maintenance of lung volume above FRC for a greater proportion of expiration. The mechanisms responsible for decreasing expiratory flow include glottic narrowing and maintenance of inspiratory muscle activity during early expiration. In contrast, children in the halothane group showed rapid early expiratory flow and slowing of expiratory flow in late expiration. The mechanism for this is probably prolongation of the expiratory time constant as the lung volume decreases to relaxed end expiratory lung volume during halothane anaesthesia.[19]

Inspiratory flow is more rapid during early inspiration in the children anaesthetized with ketamine as shown by the short Ti'/Ti and the increased V0.5i/Vt in comparison with those anaesthetized with halothane. This difference in breathing pattern is statistically significant and is

sufficient to maintain mean lung volume above FRC for a greater period of inspiration. End inspiratory pauses (Figure 4) also increase the proportion of the breathing cycle wherein lung volume is maintained well above FRC.

The combination of prolonged Ti/Te, increased early inspiratory flow, braking of expiratory flow, and end inspiratory or early expiratory pauses all contribute to elevate mean lung volume above FRC to a volume almost twice that found during halothane anaesthesia. These mechanisms may defend against the tendency of dependent lung units to collapse at lower lung volumes, and thus help maintain normal (A-a)$DO_2$ during spontaneous air breathing with ketamine anaesthesia.[8,20] This may be especially important in children in whom closing capacity is closer to FRC than in adults, and in whom a decrease in mean lung volume may predispose the child to collapse of dependent lung units and increased ventilation-perfusion mismatch with hypoxaemia.[21]

Vt/Ti was less in the children receiving ketamine anaesthesia than in those receiving halothane, though the difference did not reach statistical significance. Thus in spite of increased Ti/Te ratio with ketamine, Ve was not statistically different between the groups. These factors may be difficult to interpret in view of the difficulty in establishing precisely whether the agents were given in equipotent dosages. However, it is apparent that the major difference between ketamine and halothane anaesthesia in pre-school children is not in the drive component of respiration but in these agents differing effects on respiratory timing.

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Résumé

*Les facteurs temps et force motrice de la respiration ont été étudiés chez 18 jeunes enfants après l'induction de l'anesthésie avec la kétamine et furent comparés avec les résultats obtenus chez 10 enfants après induction de l'anesthésie avel l'halothane. Après une minute de respiration calme, les signaux obtenus dans un pneumotachographe attaché au masque anesthésique furent analysés pour le volume courant (Vt), fréquence respiratoire (f), volume minute (Ve), les temps inspiratoire et expiratoire (Ti, Te) et les tendances des flots. Après l'induction de l'anesthésie avec la kétamine les enfants ont respiré plus lentement et plus profondément que les enfants ayant reçu l'halothane mais il n'y avait pas de différence significative dans le Ve ou le Vt/Ti suggérant que la force motrice respiratoire était similaire dans les deux groupes d'enfants. Chez les enfants ayant reçu la kétamine la Ti était deux fois plus longue et ainsi le rapport Ti/Te était significativement augmenté en comparaison avec le groupe de patient ayant reçu l'halothane. En plus de la prolongation du Ti chez les enfants induits à la kétamine, on nota une plus grande augmentation du volume au début de l'inspiration qu'à la fin de l'inspiration ce qui dénote une respiration apneustique. On nota une plus lente diminution du volume au début de l'expiration avec occasionnellement tôt dans l'expiration un arrêt respiratoire pouvant durer trois secondes chez les patients induits à la kétamine. Ce schéma unique de l'inspiration après kétamine consistait en une Vt large, une augmentation de Ti/Te, une inspiration apneustique et un frein expiratoire contribué en une augmentation du volume pulmonaire moyen supérieur à la capacité résiduelle fonctionnelle 2.40 ml · kg$^{-1}$ du poids corporel en comparaison à 1.27 ml · kg$^{-1}$ chez les enfants ayant reçu de l'halothane.*