***Conclusion**: L'anesthésie au propofol est associée à une baisse de $\dot{V}i$ alors que sous anesthésie à l'halothane $\dot{V}i$ ne change pas. Les paramètres de l'effort et du synchronisme ventilatoires du groupe P différaient de ceux du groupe H.*

Reports of apnea during propofol anaesthesia suggest that it may modify ventilatory control mechanisms.[1,2] In this study, an analysis was made of the inspiratory flow waveform during spontaneous ventilation and the pressure waveform during an occluded inspiration, to characterize the effect of propofol on ventilatory control in terms of (1) the Timing of the respiratory cycle, (2) the Amplitude of the respiratory neural drive and (3) the Shape of the intra-breath flow profiles in order to further characterize this apparent effect on Ventilatory Control.[3,4] Similar measurements were made in a second group of subjects during halothane anaesthesia.

Methods

Patient preparation

With Institutional ethics committee approval, informed parental consent was obtained for 20 children presenting for dental restoration under general anaesthesia. All patients were fasting, ASA grade I and were unpremedicated. Patients were randomised to receive either propofol (Group P) or halothane (Group H), for induction and maintenance of anaesthesia.

Preliminary trials showed that propofol alone did not provide the level of anaesthesia required for the dental procedure and the anaesthetic technique required a background of nitrous oxide and ketorolac (900 $\mu g \cdot kg^{-1}$). Anaesthesia was induced in Group P with propofol 2.5 $mg \cdot kg^{-1}$. Anaesthesia was induced in Group H patients with an inspired halothane concentration (F_IH) of 2%. All patients received atropine (20 $\mu g \cdot kg^{-1}$) and vecuronium (100 $\mu g \cdot kg^{-1}$) *iv*. Naso-tracheal intubation was performed with an uncuffed Sheridan endotracheal tube (ETT) with a distal sideport for CO_2 sampling. A throat pack was inserted in all children. The leak around the ETT was detected at 15 cm H_2O. The fresh gas flow of 70% N_2O:O_2 was delivered through a Mapleson D breathing system with a fresh gas flow of 12 $L \cdot min^{-1}$. Muscle paralysis was reversed after 30 min with a neostigmine/atropine mixture (50 $\mu g \cdot kg^{-1}$ and 20 $\mu g \cdot kg^{-1}$ respectively).

Anaesthesia was maintained with propofol or halothane using a washout of anaesthetic agent. Flow ($\dot{V}$), airway pressure (Pao) and end tidal carbon dioxide concentration ($P_{ET}CO_2$) were recorded at three concentrations of halothane (F_IH): 2, 1 and 0%; and five propofol infusion rates (RivP): 18, 15, 12, 9 and 0 $mg \cdot kg^{-1} \cdot hr^{-1}$. Data were collected from spontaneously breathing patients after 30 min at each set F_IH or RivP. Although 30 min may not represent a true pharmacological steady state, we considered it a reasonable compromise between the time constraints imposed by pharmacological and ethical considerations. (Ventilation was assisted between periods of data collection.)

At the end of the procedure, the anaesthetic agents were discontinued and data were recorded continuously until arousal (spontaneous movement) at which point the behavioral response necessitated tracheal extubation. The $F_{ET}H$ corresponding to F_IH 0%, 1%, and 2% were 0.191 ± 0.01%, 0.857 ± 0.03% and 1.38 ± 0.06%. Preemergence occlusion data in Group H were collected at $F_{ET}H$ 0.3%. The F_IH 0% and RivP 0 $mg \cdot kg^{-1} \cdot hr^{-1}$ values were collected in the last 30 sec as the preemergence values in unstimulated patients. Preemergence occlusion data in Group P was collected 10 min after stopping the RivP. Since the children were unresponsive at the time of airway occlusion, a residual effect of propofol was probably present.

In each patient, body temperature (rectal) was maintained within 0.5°C of the post induction value.

Signal measurement

As previously reported[4,5] airway pressure (Pao) was measured through a sideport in the ETT connector using a piezoresistive differential pressure transducer (Microswitch #162PC01D, Honeywell, Scarborough, Ontario) referenced to the atmosphere. Flow ($\dot{V}$) was measured by a heated Fleisch pneumotachograph (#0). The pressure drop across the pneumotachograph was measured with another peizoresistive differential pressure transducer (Microswitch #163PC01036). The dead space of this pneumotachograph with connector was 15 ml. Calibration of this device was performed using the flow rotameters. Volume calibration was performed with a precision 100 ml syringe and the gas mixture used for the study. Pressure was calibrated with a pressure manometer. The stability of the calibration with time was verified for each patient.

Halothane and $P_{ET}CO_2$ were measured with a Nellcor N-2500 multi-function pulse oximeter with agent analyzer (Nellcor Inc., Hayward, CA). The accuracy of the CO_2 analyzer was verified with a known concentration of CO_2. The accuracy of the halothane analyzer was checked periodically with a known concentration of halothane.

A two-way valve with an inflatable balloon in the inspiratory limb (Hans Rudolph #2384B) was positioned between the pneumotachograph and the fresh gas flow inlet to allow interruption of the circuit during expiration, so that the next inspiration could be occlud-

ed. Even with rapid respiratory rates, the airway could be reliably occluded at end-expiration.

Data analysis

A detailed description of the data analysis is provided in previous publications.[4,5] The beginning and end of inspiration and expiration were identified from the zero crossing points of the signal. Volume ($\dot{V}$) was obtained by numerical integration of $\dot{V}$.

From the $\dot{V}$ signal we determined tidal volume (V_T), minute ventilation ($\dot{V}i = 60 \cdot V_T \cdot Ttot^{-1}$) and the parameters of breath Amplitude: mean inspiratory flow (V_T/Ti); breath Timing: total cycle time (Ttot) and inspiratory time (Ti); and breath Shape: the inspiratory flow centroid (Ci/Ti) and the inspiratory duty cycle (Ti/Ttot). The mathematical equation used to calculate Ci/Ti is given in reference 4 and reflects the skewness of the flow waveform. Finally, end tidal CO_2 ($P_{ET}CO_2$) as obtained as the highest value of the smoothed expiratory PCO_2 signal from each breath.

During expiration the airway was occluded and the occluded pressure waveform of the occluded inspiration was recorded. The breath Amplitude parameter $dP/dt_{0.1}$, a correlate of $P_{0.1}$, was obtained from the occluded Pao waveforms as the slope of the line fit (by linear regression) to the first 100 msec of the occluded inspiration.[5] The coefficient of determination for the regressions was >0.97. Values for $dP/dt_{0.1}$ at all FiH and RivP were obtained from at least three occluded pressure waveforms.

Finally, the ratio between the occluded and unoccluded inspiratory times (Ti^{occ}/Ti) was determined, where Ti^{occ} is the occluded inspiratory time defined as the time taken for Pao to decrease to its minimum value from baseline.

Statistical analysis

Group data are presented as box plots. Intra and intergroup sample means were assessed with ANOVA for repeated measures.[6] Mean parameter values obtained at FiH 1% and 2% were compared with the pre-emergence (FiH 0%) values by Dunnett t test for repeated measures.[7] Mean parameter values obtained at RivP 18, 15, 12 and 9 $mg \cdot kg^{-1} \cdot min^{-1}$ were compared with the pre-emergence (RivP 0 $mg \cdot kg^{-1} \cdot min^{-1}$) values by Dunnett t test for repeated measures.[7] Intergroup differences for the preemergence values were assessed with an unpaired t test. A P value of 0.05 was considered statistically significant.

Results

We studied 10 patients in Group P (aged 4.5 ± 0.4 yr, weighing 17.5 ± 1.2 kg and whose body surface area (BSA) was 0.707 ± 0.03 m^2) and 10 patients in Group H (aged 4.0 ± 0.3 yr, weighing 17.1 ± 1.4 kg, whose BSA was 0.686 ± 0.04 m^2). Body temperature in Group P was 35.8 ± 0.2°C and in Group H it was 35.6 ± 0.2°C. Three patients in Group P had nonpurposeful movement to surgical stimulus at RivP 9 $mg \cdot kg^{-1} \cdot r^{-1}$. In one, this necessitated an increase of RivP to 12 $mg \cdot kg^{-1} \cdot hr^{-1}$ and representative data at RivP 9 $mg \cdot kg^{-1} \cdot hr^{-1}$ could not be obtained. Satisfactory occluded inspiratory pressure waveforms were obtained for ten patients in Group P and nine patients in Group H.

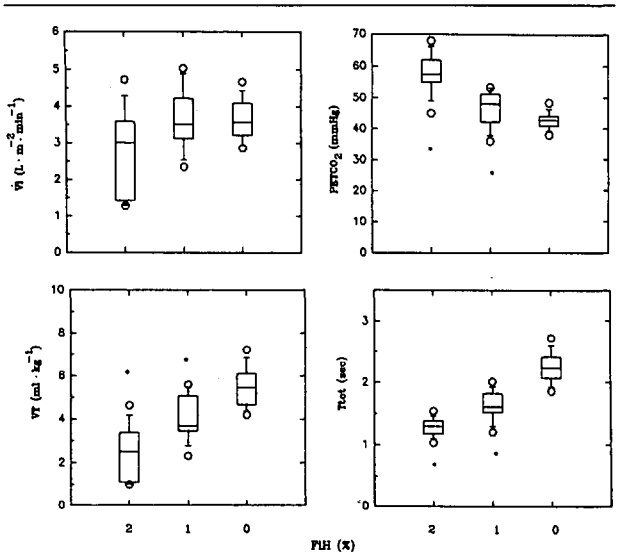

FIGURE 1 Effect of washout of halothane on the ventilatory parameters $\dot{V}i$, $P_{ET}CO_2$, V_T and Ttot. Data are presented as open box plots (showing 10th and 90th percentiles, central mean and the 5th and 95th error bars and outliers.) $\dot{V}i$ was unchanged in Group H. $P_{ET}CO_2$ increased during halothane anaesthesia whereas V_T and Ttot decreased. Intragroup differences from FiH 0% are shown (*P < 0.05).

There were no differences between groups in age, weight, BSA or body temperature. There were no differences between groups in the pre-emergence values of $\dot{V}i$, V_T, V_T/Ti or $P_{ET}CO_2$ (Figures 1, 2 and Table I) nor were there differences in the Shape parameters Ti/Ttot or Ci/Ti (Table II). There were no differences in the pre-emergence values for Respiratory Mechanics [PMAX/V_T and $(V_T/Ti)/dP/dt_{0.1}$].

Figures 1 and 2 summarize the pattern of breathing during a washout of anaesthetic in Group P and Group H. The $\dot{V}i$, V_T, Ttot increased during a washout of propofol and $P_{ET}CO_2$ decreased. In contrast the $\dot{V}i$ did not change in Group H during a washout of halothane. The $P_{ET}CO_2$ however did increase during halothane anaesthesia, in fact since the mean Te in Group H was less (0.71 sec) than in Group P (1.12 sec), the expiratory

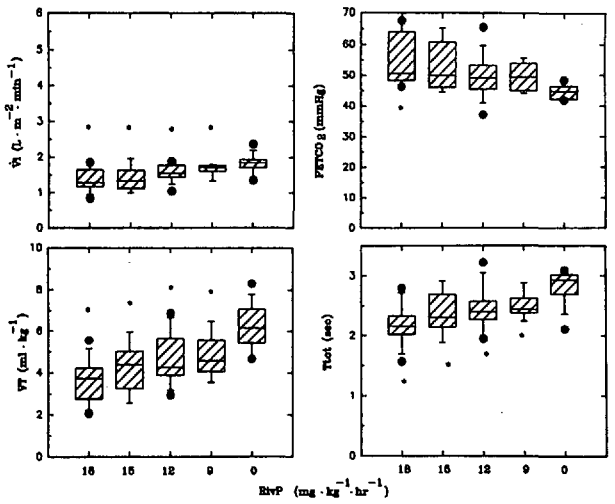

FIGURE 2 Effect of washout of propofol (hatched box plots) on the ventilatory parameters $\dot{V}i$, $P_{ET}CO_2$, V_T and Ttot. The scales of the Y-axis are identical for ease of comparison with Figure 1. The $\dot{V}i$ V_T and Ttot increased in a dose response fashion during the washout of propofol, from a RivP of 18 mg·kg^{-1}·hr^{-1} to 0 mg·kg^{-1}·hr^{-1}, whereas $P_{ET}CO_2$ decreased. Intragroup differences from RivP 0 mg·kg^{-1}·hr^{-1} are shown (*$P < 0.05$).

TABLE I Mean values (X ± SEM) for the parameters presented in Figures 1 and 2. Intragroup differences from FiH 0% or RivP 0 mg·kg^{-1}·hr^{-1} are shown *($P < 0.05$). Intergroup differences between FiH 0% and RivP 0 mg·kg^{-1}·hr^{-1} are shown †($P < 0.05$).

RivP (mg·kg^{-1}·hr^{-1}) (n = 10)	$\dot{V}i$ (L·min^{-1}·m^{-2})	V_T (ml·kg^{-1})	Ttot (s)	Ti (s)	$P_{ET}CO_2$ (mmHg)
18	2.4*	3.6*	2.18*	0.97*	54.9*
	(0.2)	(0.3)	(0.12)	(0.03)	(2.7)
15	2.5*	4.2*	2.39*	1.09	52.6
	(0.2)	(0.4)	(0.13)	(0.06)	(2.8)
12	2.8*	4.7*	2.45*	1.09	49.6
	(0.2)	(0.4)	(0.12)	(0.05)	(2.4)
9	2.8*	4.8*	2.5*	1.13	49.7
	(0.2)	(0.4)	(0.08)	(0.05)	(1.6)
0	3.2	6.2	2.82	1.16	44.8
	(0.2)	(0.4)	(0.09)	(0.03)	(0.7)
FiH (%) (n = 10)					
2	2.8	2.5*	1.28*	0.57*	58.0*
	(0.4)	(0.4)	(0.05)	(0.02)	(2.1)
1	3.7	4.0*	1.63*	0.70*	46.7*
	(0.3)	(0.3)	(0.08)	(0.03)	(1.9)
0	3.6	5.5	2.24†	0.94†	42.7
	(0.2)	(0.3)	(0.08)	(0.04)	(0.9)

TABLE II Mean values (X ± SEM) for V_T/Ti, Ti/Ttot, Ci/Ti, PMAX/V_T and (V_T/Ti)/dP/dt$_{0.1}$. Intragroup differences from FiH 0% or RivP 0 mg·kg^{-1}·hr^{-1} are shown (*$P < 0.05$).

RivP (mg·kg^{-1}·hr^{-1})	V_T/Ti ml·m^{-2}·sec^{-1}	Ti/Ttot	Ci/Ti	P_{MAX}/V_T cm·ml^{-1}	(V_T/Ti)/dP/dt$_{0.1}$ ml·cm^{-1}
18	88.2*	0.463*	0.485	0.174	3.75
	(8.3)	(0.01)	(0.01)	(0.2)	(0.36)
15	92.8	0.456	0.503	0.183	4.83
	(8.7)	(0.01)	(0.01)	(0.02)	(1.02)
12	107	0.445	0.497	0.164	4.52
	(8.9)	(0.01)	(0.02)	(0.02)	(0.68)
9	104	0.451	0.507	0.164	4.64
	(7.4)	(0.01)	(0.02)	(0.02)	(0.51)
0	132	0.413	0.518	0.175	4.73
	(8.7)	(0.01)	(0.01)	(0.02)	(1.01)
FiH (%)					
2	106*	0.448*	0.468*	0.215	1.85*
	(14.8)	(0.01)	(0.01)	(0.03)	(0.25)
1	144	0.432	0.47*	0.189	2.05*
	(13.6)	(0.01)	(0.01)	(0.03)	(0.26)
0	152	0.418	0.519	0.19	2.61
	(11)	(0.02)	(0.02)	(0.03)	(0.36)

plateau on the CO_2 waveform may have underestimated the arterial CO_2 in Group H. The pattern of breathing in Group P was rapid and shallow as both V_T and Ttot decreased in both groups.

At high values of FiH and RivP the Drive parameter V_T/Ti decreased (Table II). However the Drive parameter dP/dt$_{0.1}$ did not change in either group. The pre-emergence dP/dt$_{0.1}$ was lower in Group P, indeed dP/dt$_{0.1}$ in Group H was roughly twice that of Group P at all RivP (Figure 3) ($P < 0.05$). The Shape parameters Ti/Ttot and Ci/Ti showed inconsistent changes during the washout of anaesthetic agents (Table II). There were group differences in the pre-emergence parameters of Timing (Ttot, Ti and Ti^{occ}/Ti) with Group H having lower values. Whereas Ti^{occ}/Ti decreased during halothane anaesthesia, it did not change during propofol anaesthesia (Figure 4). In Group P PMAX/V_T and (V_T/Ti)/dP/dt$_{0.1}$ did not change during propofol administration, whereas in Group H (V_T/Ti)/dP/dt$_{0.1}$ decreased.

Discussion

Although the procedure required the addition of N_2O and ketorolac to the anaesthetic, we made the assumption that the major effect on ventilation during the period of study was due to the anaesthetic agent – propofol or halothane. Minute ventilation did not change during halothane anaesthesia and this is in agreement with previous work[5] which showed that $\dot{V}i$ was maintained at FiH 2% in oxygen in a study of similarly aged children. In contrast, the decrease in $\dot{V}i$ with propofol anaesthesia occurred at an low RivP 9 mg·kg^{-1}·hr^{-1} even though this was below anaesthetic levels in two patients. Therefore at 2 MAC halothane $\dot{V}i$ was maintained,

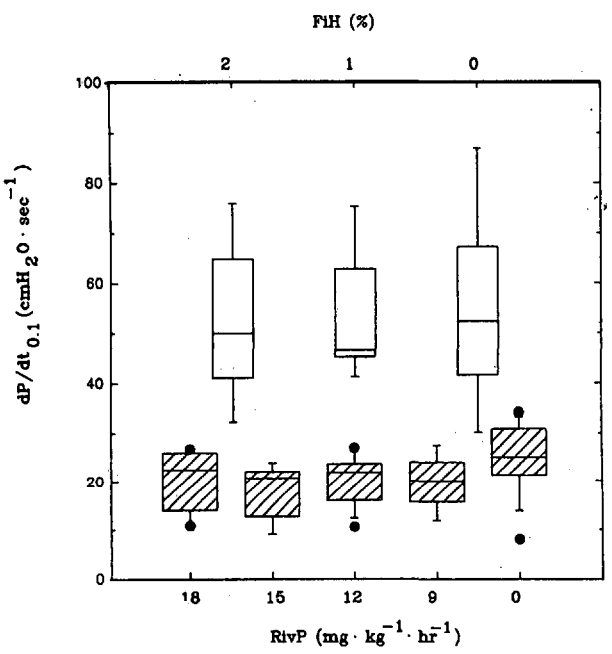

FIGURE 3 Values of $dP/dt_{0.1}$ for Group H (Open box plots) and Group P (Hatched Box plots). The value for $dP/dt_{0.1}$ did not change with either anaesthetic. In Group H the value was higher than in Group P ($P < 0.05$).

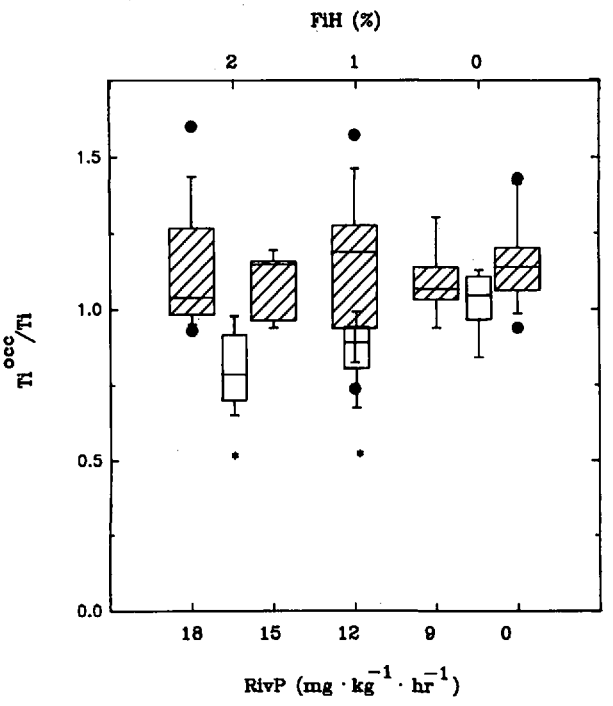

FIGURE 4 Ratios Ti^{occ}/Ti for Group H (Open box plots) and Group P (Hatched box plots). The pre-emergence value for Group H was less than Group P. During halothane anaesthesia it decreased whereas in Group P it did not change ($P < 0.05$). See text for discussion.

whereas even at light levels of propofol anaesthesia depression in $\dot{V}i$ occurred.

The $P_{ET}CO_2$ increased in both groups supporting the notion of respiratory depression during both halothane and propofol anaesthesia. Both agents have been shown to depress the ventilatory response to carbon dioxide.[8,9] In Group H the rapid and shallow pattern of breathing probably contributed to an increase in dead space ventilation predisposing to the increase in $P_{ET}CO_2$ even though $\dot{V}i$ did not change. The pattern of ventilation in Group P seemed less rapid and shallow although comparison at equivalent anaesthetic depth was problematic. Unlike halothane anaesthesia, propofol was associated with a dose response decrease in $\dot{V}i$ which may also have contributed to the dose response increase in $P_{ET}CO_2$. As a parameter of breath Drive, $dP/dt_{0.1}$ has an advantage over V_T/Ti because measured during airway occlusion, it is minimally affected by the mechanics of the respiratory system.[10] Although the V_T/Ti decreased during anaesthesia in both groups, the $dP/dt_{0.1}$ did not change. For Group H this finding agrees with a previous investigation in children of a similar age.[5] A striking finding was the intergroup difference in the value of $dP/dt_{0.1}$, with values in Group H being roughly twice that of Group P for all RivP and FiH. Does this represent a difference in the effect of halothane and propofol on ventilatory drive?

An inherent flaw in the study design which makes interpretation of these results difficult is that control values for $dP/dt_{0.1}$ were not available. In Group H, preemergence values were recorded at $F_{ET}H$ 0.3% and in Group P there was an unknown concentration of propofol. Therefore preemergence values for $dP/dt_{0.1}$ in both groups did not represent unanaesthetized values. The expected value for $dP/dt_{0.1}$ may well be intermediate between the preemergence value of Group H, with halothane tending to increase $dP/dt_{0.1}$, and propofol tending to depress it. Certainly Canet suggested that halothane increased $P_{0.1}$, a $dP/dt_{0.1}$ correlate, from awake values.[11]

Interpretation of $dP/dt_{0.1}$ as a drive parameter is complicated by the potential effect of lung volume on the shape of the diaphragm and its force-length characteristics.[10] A decrease in functional residual capacity (FRC), as occurs during anaesthesia,[12] might enhance the force generating characteristics of the diaphragm. If FRC decreased to a greater extent during halothane anaesthesia then this might account for the difference in $dP/dt_{0.1}$. Although tracheal intubation was facilitated with nondepolarizing muscle relaxants which were subsequently antagonized, no FRC restoring measures were taken. Therefore, the decrease in FRC should have been similar in both groups, indeed inductive plethysmography

has been used in adults to demonstrate a fall in FRC with induction of propofol anaesthesia.[2] Therefore, *a priori*, a difference in lung volume should not account for the difference in $dP/dt_{0.1}$.

The ratio $(V_T/Ti)/dP/dt$ has been used as an index of the efficiency of the respiratory pump in generating flow. It has been shown to decrease during methoxyflurane[13] and halothane anaesthesia[6] and may indicate a mechanical impairment of the respiratory pump. The fact that it did not change in Group P suggests that respiratory efficiency may be maintained during propofol anaesthesia.

The ratio Ti^{occ}/Ti is an index of the Hering Breuer reflex (HBR) assessing the influence of lung inflation on inspiratory duration.[14,15] The ratio Ti^{occ}/Ti decreased during halothane anaesthesia in Group H which is consistent with studies in halothane anaesthetized adults.[12,16,17] The decrease in Ti^{occ}/Ti suggests a central mechanism for the tachypnea associated with halothane anaesthesia, a notion which is consistent with experimental data.[18,19] In Group P the fact that the ratio Ti^{occ}/Ti did not change suggests that during propofol anaesthesia pulmonary input remained an important determinant of the duration of inspiration.

The Shape parameters in Group H changed in that Ti/Ttot decreased during a washout of halothane and Ci/Ti increased. In a previous report of similarly aged children anaesthetized with halothane in air:oxygen we showed no change in the Shape parameters Ci/Ti and Ti/Ttot.[5] Similarly Jonson[20] showed that the Shape of the flow waveform was not changed with halothane administration. It may be that N_2O can produce an effect on the Shape parameters since the Ti/Ttot also decreased during the washout of propofol and Ci/Ti tended to increase although this trend did not reach statistical significance.

In summary, $P_{ET}CO_2$ increased during anaesthesia in both groups. We speculate that the increase in Group H was due to the more rapid shallow pattern of breathing, whereas in Group P it was associated with the decrease in $\dot{V}i$. There was a twofold difference in the breath drive parameter $dP/dt_{0.1}$ between Group H and Group P which suggests that ventilatory drive was lower in the children anaesthetized with propofol compared with those anaesthetized with halothane, although the potential effect of lung volume on $dP/dt_{0.1}$ cannot be excluded. The decrease in the Timing parameter Ti^{occ}/Ti during halothane anaesthesia and the lack of change in Ti^{occ}/Ti suggests that the duration of inspiration during halothane anaesthesia was primarily determined by central events whereas during propofol anaesthesia pulmonary input remained an important determinant of inspiratory duration.

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