

- man during halothane anaesthesia and surgery. *Anaesth Analg* 49:363-366, 1970
8. Jakobsen H, Hertz JB, Johansen JR, Hansen A, Kolliber K: Pre-medication before day surgery. *Br J Anaesth* 57:300-305, 1985
  9. Kanto J: Benzodiazepines as oral premedicants. *Br J Anaesth* 53: 1179-1187, 1981
  10. L'Armand J, Vredevoe LA, Connor JT, Hen GP, Schehl D: Lorazepam and Morphine for surgical premedication. *Br J Anaesth* 52:1259-1263, 1980
  11. Stoltz RR: Psychological preparation and preoperative medications, *Anesthesia*. Edited by Miller RD. New York, Churchill Livingstone, 1986, pp 381-397
  12. Abrams LM, Chamber DA: Preoperative management, cardiac anesthesia. Edited by Kaplan JA. New York, Grune and Stratton, 1979, pp 169-195
  13. Yalow RS, Berson SA: Radioimmunoassay of ACTH in plasma. *J Clin Invest* 47:2725-2729, 1968
  14. Guillemin R, Ling N, Vargo TM: Radioimmunoassay for alpha endorphin and beta endorphin. *Biochem Biophys Res Comm* 77:361-366, 1977
  15. Norris W, Baird WLM: Preoperative anxiety—A study of the incidence and aetiology. *Br J Anaesth* 39:503-509, 1967
  16. White PF: Pharmacological and clinical aspects of preoperative medication. *Anesth Analg* 65:963-974, 1986
  17. Leigh JM, Walker J, Janaganathan P: Effect of preoperative anaesthetic visit on anxiety. *Br Med J* 2:987-989, 1977
  18. Hough LB, Glick SD, Su K: A role for histamine and histamine-H2 receptors in non-opiate footshock-induced analgesia. *Life Sci* 36:859-866, 1985
  19. Wuster W, Duka T, Herz A: Diazepam-induced release of opioid activity in the rat brain. *Neurosci Lett* 16:335-337, 1980
  20. Dubois M, Pickar D, Cohen MR, Roth YF, MacNamara T, Bunnoy WE: Surgical stress in humans is accompanied by an increase in plasma beta endorphin immunoreactivity. *Life Sci* 29:1249-1254, 1981
  21. Krieger DT, Liotta AS, Hauser H, Brownstein MJ: Effect of stress, adreno-corticotropin or corticosteroid treatment, adrenalectomy of hypophysectomy on hypothalamic immunoreactive ACTH concentration. *Endocrinology* 105:737-742, 1979

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## End-tidal $P_{CO_2}$ Monitoring in Infants and Children Ventilated with Either a Partial Rebreathing or a Non-rebreathing Circuit

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End-tidal  $P_{CO_2}$  measurements are frequently less accurate in neonates, infants, and small children than in adults.\*\*<sup>1</sup> The difficulty in obtaining accurate measure-

ments in pediatric patients may be attributed to the low ratio of tidal volume to equipment deadspace, rapid ventilatory rates and high fresh gas flows, and high sampling rates required by  $CO_2$  analyzers. We believe that these problems are maximal when pediatric patients (particularly neonates and infants) are ventilated with partial rebreathing circuits, which allow fresh gas to flow past the endotracheal tube during expiration, and are minimal when these patients are ventilated with circuits which have non-rebreathing valves that separate the inspiratory and expiratory phases of respiration. To verify this clinical impression, we compared the arterial  $P_{CO_2}$  ( $Pa_{CO_2}$ ) with the end-tidal  $P_{CO_2}$  ( $P_{etCO_2}$ ) during ventilation of neonates, infants, and children using a partial rebreathing circuit (Air-Shields Ventimeter® [ASV] and a Mapleson D breathing circuit) or a non-rebreathing circuit (Siemens-Elma "Servo" 900-C® [SES]).

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\*\* Sasse FJ: Can we trust end-tidal carbon dioxide measurement in infants. *J Clin Mon* 1:147-148, 1985.

## METHODS AND MATERIALS

Fifty children with no known cardiopulmonary disease who were scheduled for general surgery or neurosurgical procedures were studied. The patients varied in age from premature newborn to 9 yr, and in weight from 1.65-23.5 kg. The study was approved by the Institutional Re-

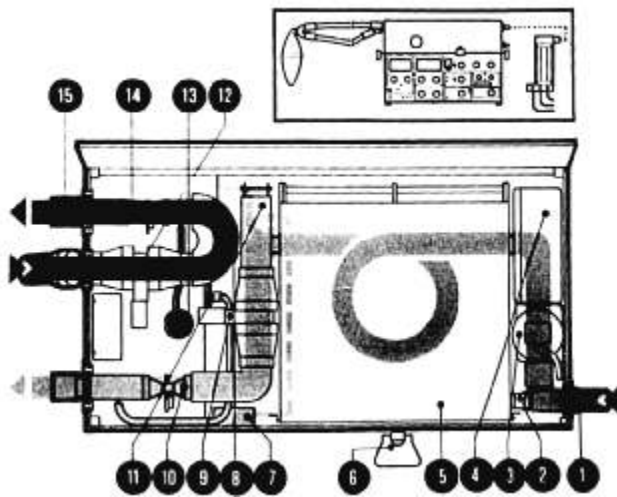


FIG. 1. A diagram of the Siemens-Elema "Servo" 900-C (SES). 1 - gas inlet; 2 = gas supply valve; 3 = O<sub>2</sub> cell; 4 = bacteria filter; 5 = bellows; 6 = working pressure adjustment screw; 7 = working pressure manometer; 8 = safety valve; 9 = inspiratory flow transducer; 10 = inspiratory valve; 11 = airway pressure transducer; 12 = expiratory flow transducer; 13 = expiratory pressure transducer; 14 = expiration valve; 15 = flap valve. Inset: The fresh gas flow line from the anesthetic machine is connected to the low pressure gas inlet of the SES. Fresh gas will flow from the anesthetic machine to the patient if it is delivered from the flowmeters at a rate which exceeds the inspired minute ventilation setting on the SES.

view Board, and informed consent was obtained from the parents.

After induction of general anesthesia with either an intravenous or inhalation induction and endotracheal intubation, ventilation was controlled with: 1) a partial re-breathing circuit (an ASV and Mapleson D circuit [Bain or Jackson-Rees modification of the Ayre's t-piece]), or 2) a non-rebreathing circuit (a SES), or 3) both ASV and SES used in random sequence. When the ASV was used, the initial fresh gas flows were 1000 cc plus 100 cc · kg<sup>-1</sup> for infants and children less than 30 kg in body weight and 2000 cc plus 50 cc · kg<sup>-1</sup> for children greater than 30 kg.<sup>2</sup> Thereafter, fresh gas flows were adjusted to maintain PaCO<sub>2</sub> or PetCO<sub>2</sub> within an acceptable clinical range for general surgery (34–38 mmHg) and neurosurgery (28–34 mmHg). The respiratory rate was between 30 and 40 breaths per minute (BPM) for newborns and infants, and between 20 and 30 BPM for older infants and children. When the ASV was used, the tidal volume (V<sub>t</sub>) was approximately 10–15 cc · kg<sup>-1</sup> with corresponding peak inspiratory pressures of 20–30 mmHg. When the SES ventilator was used, the expired minute ventilation ( $\bar{V}_E$ ) was adjusted by varying the V<sub>t</sub> and respiratory rate (as indicated above) to maintain PaCO<sub>2</sub> or PetCO<sub>2</sub> within the acceptable clinical ranges. In the SES, a flow

transducer in the expiratory channel (fig. 1, No. 12) measures the  $\bar{V}_E$  and calculates the V<sub>T</sub> (V<sub>T</sub> =  $\bar{V}_E$  ÷ respiratory rate). Fresh gas flows from the anesthetic machine were connected to the SES at the low pressure inlet (fig. 1, No. 1) and delivered at a flow rate greater than the  $\bar{V}_E$ . Positive end-expiratory pressures were avoided when either ventilator was used. All data were obtained after the induction of anesthesia and before incision. All patients were supine and horizontal throughout the study. Rectal temperatures were recorded throughout the study and were maintained between 36 and 37° C.

Respiratory gases were sampled through a side stream connector placed between the endotracheal tube and the breathing circuit, and were measured with a mass spectrometer calibrated each day with dry gas. The spectrometer sample flow rate was 240 ml · min<sup>-1</sup>, for periods of 20 s, and at intervals of 1 or more min. The sampling line was 40 m in length from the patient to the mass spectrometer. Exhaled gas measurements were corrected for the presence of water vapor, and reflected saturated alveolar levels at ambient barometric pressures.

Arterial blood samples for PaCO<sub>2</sub> were obtained from an indwelling arterial catheter (n = 5), *via* percutaneous puncture (n = 41), or *via* arterialized heel stick (n = 3). The PaCO<sub>2</sub> samples were measured at 37° C with a CO<sub>2</sub> electrode calibrated prior to each sample and uncorrected for body temperatures.<sup>3</sup> The PaCO<sub>2</sub> was compared to peak-expiratory or end-tidal P<sub>CO<sub>2</sub></sub> values sampled simultaneously. For downsloping plateaus, the peak end-expiratory P<sub>CO<sub>2</sub></sub> value was compared to PaCO<sub>2</sub>, whereas, for flat plateaus, end-tidal P<sub>CO<sub>2</sub></sub> was compared to PaCO<sub>2</sub>. The inspired concentrations of CO<sub>2</sub> (PI<sub>CO<sub>2</sub></sub>) and inspiratory:expiratory time ratios (I:E) were measured and recorded in each patient.

The gradients between PaCO<sub>2</sub> and PetCO<sub>2</sub> values ( $\Delta P_{CO_2(a-et)}$ ) were compared to body weight using non-linear exponential regression analysis and the coefficient of determination (r<sup>2</sup>). After the study was completed, the subjects were divided into four groups according to their weight and the ventilator(s) used during the study (table 1). Differences in the slopes of the regression lines among the groups were compared using analysis of covariance. Differences in weight,  $\Delta P_{CO_2(a-et)}$  values, and PI<sub>CO<sub>2</sub></sub> values among the groups were compared using one-way ANOVA and the Student-Newman-Keuls test. Statistical significance of  $P \leq 0.05$  was accepted.

## RESULTS

Ventilation was controlled with an ASV and Mapleson D circuit alone in 32 patients (21 with a Bain circuit and 11 with an Ayre's t-piece), with an SES alone in 10 patients, and with both ventilators in sequence (denoted by \* in fig. 1) in 8 patients. When the ASV was used, only

TABLE 1. Patients Ventilated with the Air-shields Ventimeter (ASV) and the Siemens-Elcoma "Servo" 900-C (SES)

	ASV		SES		Both Ventilators*	
	<8 kg	≥8 kg	<8 kg	≥8 kg	<8 kg	≥8 kg
Number of patients	17	23	10	8	3	5
Weight (kg)	4.4 ± 1.8‡	12.9 ± 5.1	4.3 ± 1.8	13.8 ± 6.5	5.6 ± 1.8‡	12.6 ± 6.2
ΔP <sub>CO<sub>2</sub></sub> (a-et)† (mmHg)	12.0 ± 7.8§	1.5 ± 2.6	3.4 ± 1.5	2.8 ± 2.3	ASV 12.4 ± 8.0§ SES 2.6 ± 1.0	0.6 ± 2.4 2.2 ± 2.5

Data are mean ± SD.

\* Patients ventilated in random sequence with both ventilators; the data for these 8 patients are also included in the data from the other 42 patients summarized in the first 4 columns.

† ΔP<sub>CO<sub>2</sub></sub> (a-et) = the difference between Pa<sub>CO<sub>2</sub></sub> and Pet<sub>CO<sub>2</sub></sub>.

‡ P < 0.05 compared to ≥8 kg groups.

§ P < 0.01 compared to the other three groups.

minimal adjustments in fresh gas flow (± 10%) were required to maintain the Pa<sub>CO<sub>2</sub></sub> or Pet<sub>CO<sub>2</sub></sub> within the clinical ranges. The mean weight of the <8 kg ASV group did not differ significantly from that of the <8 kg SES group, and, similarly, the mean weight of the ≥8 kg ASV group did not differ significantly from that of the ≥8 kg SES group (table 1).

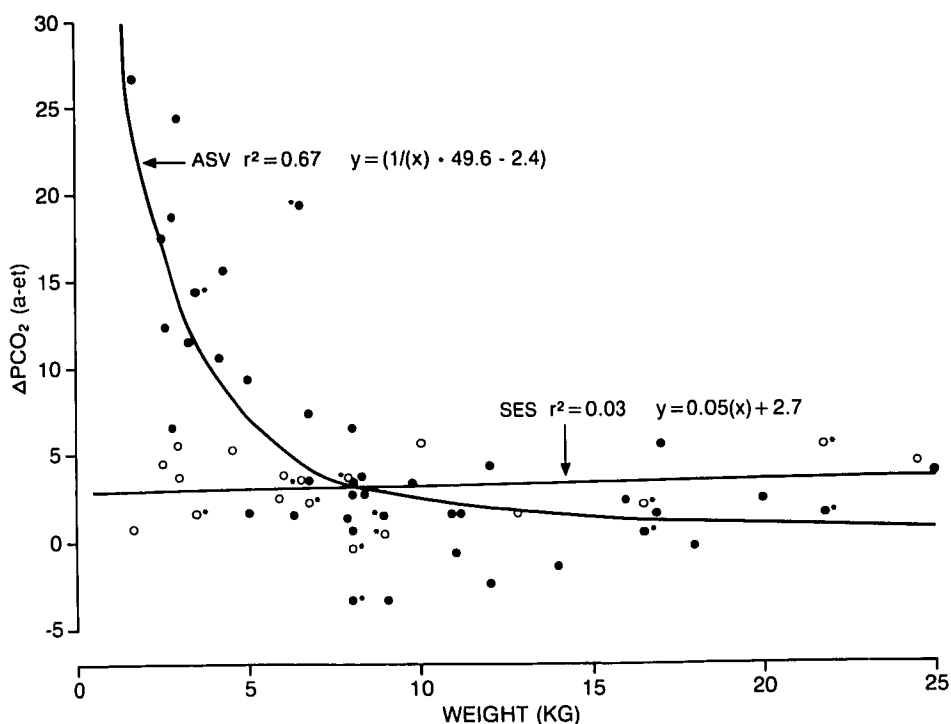
A hyperbolic relationship existed between body weight and ΔP<sub>CO<sub>2</sub></sub> (a-et) values in the patients ventilated with an ASV:  $y = 1 \div 49.6(x) - 2.4$ , ( $r^2 = 0.67$ ) where y is ΔP<sub>CO<sub>2</sub></sub> (a-et) and x is the patient weight (fig. 2). Since the hyperbolic curve for the partial rebreathing circuit crossed the line for the SES group at approximately 8 kg on the abscissa, the patients in the ASV group were divided into two groups according to weight: <8 kg and ≥8 kg. The

relationship between weight and ΔP<sub>CO<sub>2</sub></sub> (a-et) values in the patients ventilated with the SES did not depend significantly on weight:  $y = 0.05(x) + 2.73$  ( $r^2 = 0.03$ ).

The Pet<sub>CO<sub>2</sub></sub> measurements did not approximate Pa<sub>CO<sub>2</sub></sub> measurements in the <8 kg ASV group (fig. 3). However, Pet<sub>CO<sub>2</sub></sub> measurements did approximate Pa<sub>CO<sub>2</sub></sub> measurements in the ≥8 kg ASV group and in all patients (< and ≥8 kg) in the SES groups (fig. 4). The mean ΔP<sub>CO<sub>2</sub></sub> (a-et) value was significantly greater in the <8 kg ASV group compared to both the ≥8 kg ASV group and all patients in the SES group (table 1).

Weight and ΔP<sub>CO<sub>2</sub></sub> (a-et) values obtained from the eight patients ventilated in random sequence with both ventilators were similar to those from patients ventilated with only one of the ventilators (table 1).

FIG. 2. Arterial to end-tidal P<sub>CO<sub>2</sub></sub> gradients compared to body weight when the Air-Shields Ventimeter and Siemens-Elcoma "Servo" 900-C were used to ventilate infants and children (\* = ventilators used in sequence in same patient).



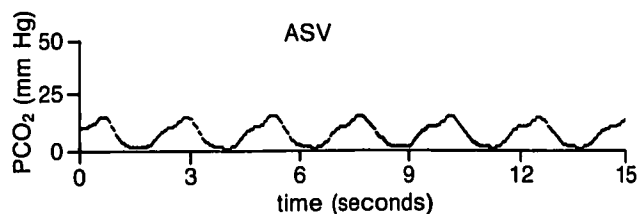


FIG. 3. The capnographic waveform for a 2.5 kg infant ventilated with an Air-Shields Ventimeter and t-piece circuit with a fresh gas flow of  $1.25 \text{ l} \cdot \text{min}^{-1}$  (resp. rate = 33 and I:E ratio = 1:1.1).  $\text{Pa}_{\text{CO}_2}$  = 32.3 mmHg;  $\text{Pet}_{\text{CO}_2}$  = 14.8 mmHg;  $\Delta\text{P}_{\text{CO}_2}$  (a-et) = 11.5 mmHg;  $\text{PI}_{\text{CO}_2}$  = 0.8 mmHg.

There was a trend for the  $\text{PI}_{\text{CO}_2}$  values in the ASV groups with I:E > 1:3.5 (e.g., 1:1, 1:2, and 1:3) to be greater than the  $\text{PI}_{\text{CO}_2}$  values in the SES groups with I:E > 1:3.5 (table 2). The mean  $\text{PI}_{\text{CO}_2}$  value in the >8 kg ASV group with I:E ≤ 1:3.5 was greater than the mean  $\text{PI}_{\text{CO}_2}$  values in all groups with I:E > 1:3.5. In the SES group,  $\text{PI}_{\text{CO}_2}$  was greater in the I:E ≤ 1:3.5 (e.g., 1:4, 1:5, 1:6, and 1:7) groups than in the groups with an I:E ≥ 1:3.5.

Seven of the 50 patients had a  $\text{Pet}_{\text{CO}_2}$  value greater than the  $\text{Pa}_{\text{CO}_2}$  value (negative  $\Delta\text{P}_{\text{CO}_2}$ [a-et]). Of these patients, six were in the ≥8 kg ASV group (mean weight ± SD =  $11.4 \pm 3.6$  kg; mean  $\Delta\text{P}_{\text{CO}_2}$ [a-et] ± SD =  $-1.8 \pm 1.3$  mmHg), and one was in the ≥8 kg SES group (8.1 kg,  $\Delta\text{P}_{\text{CO}_2}$ [a-et] =  $-0.43$  mmHg).

The capnographic waveforms in 13 of 17 patients in the <8 kg ASV group demonstrated either a flat plateau phase, despite a large  $\Delta\text{P}_{\text{CO}_2}$  (a-et) value, or failed to achieve a plateau phase on the capnographic waveform (table 3, fig. 5). Furthermore, the capnographic waveforms in 8 of 23 patients in the ≥8 kg ASV group demonstrated decay of the plateau phase (table 2, fig. 6A). In these eight patients, the  $\text{Pet}_{\text{CO}_2}$  measurements were less accurate than the  $\text{Pet}_{\text{CO}_2}$  measurements in the 15 patients who did not demonstrate decay. When the SES was used, decay of the plateau phase was not observed in any of the patients (fig. 6B).

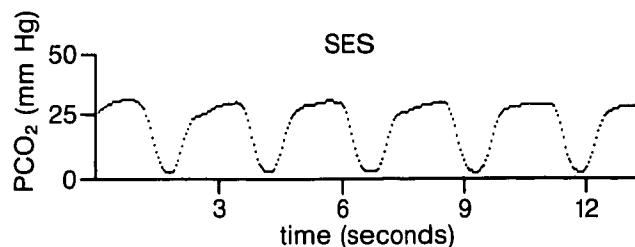


FIG. 4. The capnographic waveform for a 3 kg infant ventilated with a Siemens-Eléma "Servo" 900-C Ventilator with an expired minute ventilation of 1.35 l (resp. rate = 28 and I:E ratio = 1:2.50).  $\text{Pa}_{\text{CO}_2}$  = 28.8 mmHg;  $\text{Pet}_{\text{CO}_2}$  = 26.9 mmHg;  $\Delta\text{P}_{\text{CO}_2}$  (a-et) = 1.9 mmHg;  $\text{PI}_{\text{CO}_2}$  = 1.4 mmHg.

TABLE 2. The Effect of Inspiratory:Expiratory Time Ratios (I:E) on the Inspired Concentration of  $\text{CO}_2$  ( $\text{PI}_{\text{CO}_2}$ ) in Infants and Children Ventilated with the Air-shields Ventimeter (ASV) and the Siemens-Eléma "Servo" 900-C (SES)

	I:E > 1:3.5		I:E ≤ 1:3.5	
	<8 kg	>8 kg	<8 kg	>8 kg
ASV	$4.3 \pm 4.8$ (n = 11)	$5.2 \pm 5.0$ (n = 13)	$8.4 \pm 5.6$ (n = 6)	$10.5 \pm 4.0^*$ (n = 10)
SES	$2.2 \pm 2.0^\dagger$ (n = 3)	$1.8 \pm 0.9^\dagger$ (n = 4)	$8.3 \pm 3.2$ (n = 6)	$7.5 \pm 4.3$ (n = 5)

Data are mean ( $\text{PI}_{\text{CO}_2}$ ) values in mmHg ± SD.

\*  $P < 0.05$  compared to all four groups with I:E > 1:3.5.

†  $P < 0.05$  compared to SES groups with I:E ≤ 1:3.5.

## DISCUSSION

The large  $\Delta\text{P}_{\text{CO}_2}$  (a-et) values in patients <8 kg who are ventilated with the ASV (a continuous flow, time-cycled ventilator) may be attributed, in part, to the dilution of end-tidal gas by the continuous flow of fresh gas past the sampling site at the top of the endotracheal tube.†† Furthermore, the diluted end-tidal gas samples result in capnographic waveforms, which either fail to achieve a flat plateau phase, or reach a flat plateau which underestimates the  $\text{Pa}_{\text{CO}_2}$ . However, in the SES, an inspiratory valve (fig. 1, No. 10) automatically interrupts the flow of fresh gas at the completion of inspiration thereby allowing undiluted alveolar gas to be sampled during the expiratory phase. Consequently,  $\text{Pet}_{\text{CO}_2}$  measurements in patients ventilated with the SES accurately predict  $\text{Pa}_{\text{CO}_2}$  and produce flat plateaus on the capnographic waveform even in very small infants.

It has been suggested that the presence of a "flat alveolar phase" on the capnogram ensures that the  $\text{Pet}_{\text{CO}_2}$  closely approximates the alveolar  $\text{P}_{\text{CO}_2}$  ( $\text{PA}_{\text{CO}_2}$ ) or  $\text{Pa}_{\text{CO}_2}$  in infants.‡‡ However, in the present study, the presence of large  $\Delta\text{P}_{\text{CO}_2}$  (a-et) values in patients with flat plateau phases are consistent with a mathematical model which suggests that, when  $\text{Pet}_{\text{CO}_2}$  is sampled at the proximal end of the endotracheal tube, the "determinants of distortion" (expiratory flow rate and concentration profiles, the sample flow rate, sample tube dimensions, and sample cell volume) may lead to artificially flat plateau phases and  $\text{Pet}_{\text{CO}_2}$  values which underestimate the  $\text{Pa}_{\text{CO}_2}$  values.<sup>4</sup> Thus, a flat plateau phase may not always represent the alveolar phase, particularly when sampled proximally.

†† Gravenstein N, Lampotang S, Beneken JEW: Factors influencing capnography in the Bain circuit. *J Clin Mon* 1:6-10, 1985

‡‡ Schieber RA, Namnoum 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 Mon* 1:149-155, 1985

TABLE 3. Capnographic Waveforms in Patients Ventilated with the Air-shields Ventilometer

	<8 kg			≥8 kg	
	Patients with a Flat Plateau on Capnogram Waveform		Patients without a Flat Plateau on Capnogram Waveform	Decay of the Capnographic Waveform	
	Small $\Delta P_{CO_2}$ (a-et)	Large $\Delta P_{CO_2}$ (a-et)		Present	Absent
Number of patients	4	7	6	8	15
Weight (kg)	6.6 ± 1.0*	3.5 ± 1.5	3.8 ± 1.3	11.4 ± 3.5	13.8 ± 5.8
$\Delta P_{CO_2}$ (a-et)† (mmHg)	2.0 ± 1.0*	14.5 ± 6.6	15.4 ± 6.3	3.2 ± 2.3‡	0.6 ± 2.4

Data are mean ± SD.

\*  $P < 0.01$  compared to the other two <8 kg groups.

†  $\Delta P_{CO_2}$  (a-et) = the difference between  $P_{aCO_2}$  and  $P_{etCO_2}$ .

‡  $P < 0.01$  compared to ≥8 kg patients with absence of capnographic waveform decay.

The negative  $\Delta P_{CO_2}$  (a-et) values in our study are consistent with existing data in adults,<sup>5,6</sup> and may be explained by either calibration errors in the CO<sub>2</sub> electrode or mass spectrometer, the charged membrane hypothesis, or the delayed equilibration theory.<sup>5,6</sup> Since it is currently accepted that  $P_{aCO_2}$  is equal to  $P_{ACO_2}$  in the lung at equilibrium,<sup>5</sup> the small negative or positive  $\Delta P_{CO_2}$  (a-et) values in our data probably reflect small calibration errors or small ventilation to perfusion mismatches in some patients.

It is not known from these data whether  $P_{etCO_2}$  can be measured accurately in small infants when they are ventilated with a continuous-flow, time-cycled ventilator and a conventional circle system. However, accurate  $P_{etCO_2}$  measurements are obtained in newborn pigs only when additional one-way valves are added to the conventional circle system and when measurements are sampled from the distal end of the endotracheal tube.<sup>‡‡</sup> It is also not known from these data whether  $P_{etCO_2}$  can be measured accurately in the ASV or SES using a flow-through type sampling cell. However, since mixing of end-tidal and fresh gases may still occur, one might speculate that  $P_{etCO_2}$  measurements in patients < 8 kg would be accurate with the SES and inaccurate with the ASV.

These data suggest that when either the ASV or the

SES is used, the optimal I:E ratio to prevent rebreathing is > 1:3.5. When the I:E ratios ≤ 1:3.5, the inspiratory time is insufficient to allow adequate removal of exhaled gases and rebreathing occurs even in the SES, a "non-rebreathing" system (fig. 6B).

In a previous study using a lung model, two different sample flow rates (200 and 500 ml · min<sup>-1</sup>) did not significantly effect the accuracy of  $P_{etCO_2}$  measurements.<sup>††</sup> In the present study, we used similar sample flow rates (240 ml · min<sup>-1</sup>) and obtained accurate  $P_{etCO_2}$  measurements in all patients ventilated with the SES and patients ≥ 8 kg ventilated with the ASV. Since the same sample flow rate was used in both the <8 kg SES and <8 kg ASV patients, perhaps the sample flow rate is not an important

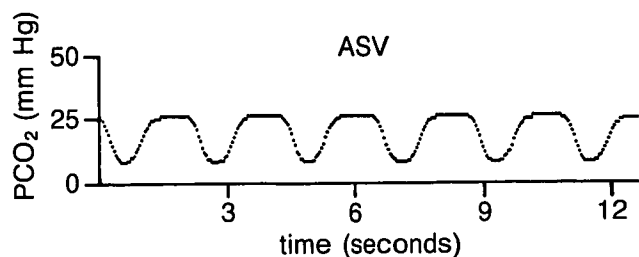


FIG. 5. The capnographic waveform for a 4.1 kg infant ventilated with an Air-Shields Ventilometer and Bain circuit with a fresh gas flow of 1.4 l · min<sup>-1</sup> (resp. rate = 32 and I:E ratio = 1:2.33)  $P_{aCO_2}$  = 40.3 mmHg;  $P_{etCO_2}$  = 26.4 mmHg;  $\Delta P_{CO_2}$  (a-et) = 13.9 mmHg;  $P_{ICO_2}$  = 7.5 mmHg.

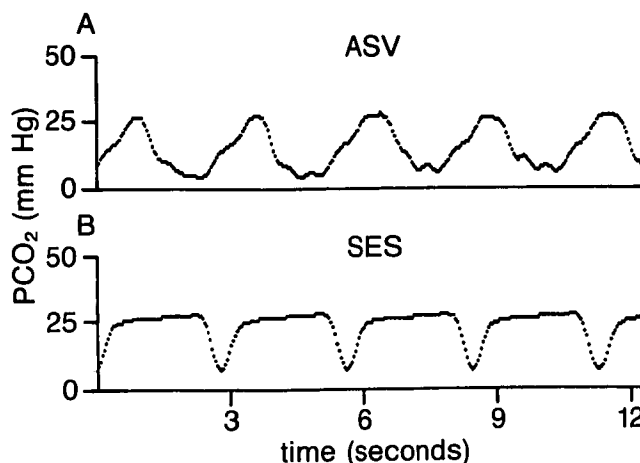


FIG. 6. A. The capnographic waveform for an 8 kg child ventilated with an Air-Shields Ventilometer and t-piece circuit with a fresh gas flow of 2 l · min<sup>-1</sup> (resp. rate = 30 and I:E ratio = 1:1.15).  $P_{aCO_2}$  = 33.9 mmHg;  $P_{etCO_2}$  = 31.0 mmHg;  $\Delta P_{CO_2}$  (a-et) = 2.9 mmHg;  $P_{ICO_2}$  = 8.1 mmHg. B. Same child (as in A) ventilated with a Siemens-Elema "Servo" 900-C Ventilator with an expired minute ventilation of 2 l (resp. rate = 25 and I:E ratio = 1:6.6).  $P_{aCO_2}$  = 30.6 mmHg;  $P_{etCO_2}$  = 31.0 mmHg;  $\Delta P_{CO_2}$  (a-et) = -0.4 mmHg;  $P_{ICO_2}$  = 7.3 mmHg.

factor in preventing accurate  $P_{\text{etCO}_2}$  measurements in patients <8 kg ventilated with an ASV.

In summary, we have shown that  $P_{\text{etCO}_2}$  measurements sampled from the proximal end of the endotracheal tube do not accurately predict  $P_{\text{aCO}_2}$  measurements in patients weighing less than 8 kg who are ventilated with a continuous-flow, time-cycled ventilator and a Mapleson D partial rebreathing circuit. By contrast,  $P_{\text{etCO}_2}$  measurements sampled from proximal sites accurately predict the  $P_{\text{aCO}_2}$  in patients more than 8 kg in weight who are ventilated with this circuit and in all patients (<8 and  $\geq$ 8 kg) ventilated with the Siemens-Elema "Servo" 900-C® Ventilator. This study indicates the need to develop an accurate technique to sample  $P_{\text{etCO}_2}$  when continuous flow ventilators and Mapleson D circuits are used in small infants. Meanwhile, the Siemens-Elema "Servo" 900-C remains a very useful ventilator when accurate end-tidal  $P_{\text{CO}_2}$  monitoring is important in small infants.

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#### REFERENCES

1. May WS, Heavner JE, McWhorter D, Racz GB: Capnography in the Operating Room—An Introductory Directory. New York, Raven Press, 1985, pp 12–13
2. Rose DK, Froese AB: The regulation of  $P_{\text{aCO}_2}$  during control ventilation of children with a t-piece. *Can Anaesth Soc J* 26:104–113, 1979
3. Ream AK, Reitz BA, Silverberg G: Temperature correction of  $P_{\text{CO}_2}$  and  $p\text{H}$  in estimating acid-base status: An example of the emperor's new clothes? *ANESTHESIOLOGY* 56:41–44, 1982
4. Epstein RA, Reznik AM, Epstein MAF: Determinants of distortions in  $\text{CO}_2$  catheter sampling systems: A mathematical model. *Respir Physiol* 41:127–136, 1980
5. Piiper J: Blood-gas equilibrium of carbon dioxide in lungs: A continuing controversy. *J Appl Physiol*. 60:1–8, 1986
6. Hlastala MP, Robertson HT: Evidence for active elimination of carbon dioxide from the lung, *Pulmonary Gas Exchange, Volume II*. Edited by West JB. Orlando, Academic Press, 1980, pp 241, 273

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### Anesthetic Management for Cesarean Section of a Patient with Charcot-Marie-Tooth Disease

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Charcot-Marie-Tooth disease, a rare degenerative disease of the peripheral nervous system has been recognized as a clinical entity since 1886.¶\*\* Described separately by Charcot and Marie in France and Tooth in England, the disease usually follows an autosomal dominant mode of inheritance. The hallmark of the disease process is per-

oneal muscle atrophy, reflecting the tendency for involvement of distal limb musculature. High pedal arches or club feet are common; mildly affected patients may demonstrate only foot deformities. Nerve conduction velocities and sural nerve biopsies permit differentiation into two subtypes. Type I usually has an onset in the first or second decade of life with foot drop and steppage gait. Sensory impairment occurs in a stocking and glove distribution. Later in life, atrophy of intrinsic hand muscles occurs. Tendon reflexes are diminished in affected areas, and foot deformities are common. Type II usually appears in adulthood, with symptoms similar to type I. Either subtype may present at any age, however. Foot deformities may be evident for many years prior to the appearance of muscular atrophy. Progression of type I is slow, and type II, very slow. Incapacitation is very rare, and death usually occurs from other causes.<sup>1</sup>

We recently encountered a patient with Charcot-Marie-Tooth disease who had experienced a severe exacerbation of her disease process during pregnancy. Such occurrences have been rarely reported.<sup>2,3</sup> Anesthesia management of such a patient has never been described, although anes-

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¶ Charcot JM, Marie P: Sur une torme particulière d'atrophie musculature progressive souvet familiale débutant par les pieds et les jambes et atteignant plus tard les mains. *Rev Mèd Pairs* 6:97–138, 1886

\*\* Tooth HH: The peroneal type of progressive muscular atrophy (M.D. thesis). London: Cambridge University, 1886