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Skin and Rectal Temperatures during Ether and Halothane Anesthesia in Infants and Children

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Skin, rectal, and room temperatures and blood gases were monitored in 70 patients 5 months to 6 years old undergoing repair of harelip or cleft palate under ether or halothane anesthesia. During anesthesia, rectal temperatures rose as a function of time with both anesthetics, though with halothane there was an initial drop in temperature. Rectal temperatures rose more frequently, more rapidly, and to higher levels with ether than with halothane. Skin temperatures decreased progressively during both types of anesthesia, but the decreases were greater with ether than with halothane. The differences between rectal and peripheral skin temperatures (internal gradient) increased at both 60 minutes and 120 minutes with both ether and halothane, more so with ether. A decrease of Pa_{CO_2} together with a moderate decrease of base excess was noted with ether but not with halothane. CO_2 accumulation is, therefore, not an etiologic factor in elevation of body temperature during ether or halothane anesthesia in children. (Key words: Temperature, body: diethyl ether; Temperature, body: halothane; Anesthetics, volatile: body temperature; Anesthesia, pediatric: body temperature.)

THERE IS a widespread though undocumented clinical impression that high body temperatures are frequently observed in children during ether anesthesia, whereas during halothane anesthesia body temperatures seldom rise. It has also been suggested, again without documentation, that carbon dioxide retention not only contributes to elevation of body temperature during ether anesthesia but also may play a role in the convulsions that may accompany such rises in temperature.[1-3] The following study was designed 1) to determine whether body temperatures rise more with ether than with halothane when all other factors are kept constant, and 2) to determine whether elevation of temperature during anesthesia is related to carbon dioxide retention.

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Materials and Methods

Each of 110 infants and children between 5 months and 6 years of age undergoing repair of harelip or cleft palate during a 12-month period was assigned to receive ether or halothane on a randomized basis. We excluded from the study patients who had histories of metabolic, respiratory, circulatory or central nervous system disorders, those who had local infection, and those who had had abnormal body temperatures during the previous week, or had rectal temperatures above 37.5 C on the morning of the study. Infants less than 2 years of age were given clear liquids by mouth until 2 hours (others 4 hours) before induction of anesthesia. Secobarbital, 7 mg/kg for patients less than 1 year of age, 6 mg/kg for those less than 2 years of age, and 5 mg/kg for others, was given intramuscularly with 0.015 mg/kg atropine 1 hour prior to induction of anesthesia.

Anesthesia was induced either by open drop or by mask. The trachea was intubated without muscle relaxants and anesthesia was maintained with either ether–oxygen or halothane–oxygen with a Keats nonrebreathing system.[4] Respiration was assisted manually. Concentrations of nitrous oxide were the same with ether and with halothane. Anesthetic gases were not humidified. An intravenous catheter was inserted after induction; through this, 5 per cent dextrose in water (10 ml/kg/h) was infused during anesthesia. Patients who showed excitement before or during induction of anesthesia or coughed or "bucked" on the endotracheal tube during operation were excluded from the study. Also excluded were patients who

TABLE 1. Distribution of the Study Population by Age, Sex, and Anesthetic Agent Used

Age	Number of Patients	Sex		Ether	Halothane
		M	F		
0–6 months	2	1	1	1	1
6–12 months	20	7	13	9	11
12–18 months	12	8	4	4	8
18–24 months	21	14	7	10	11
2–6 years	15	9	6	6	9
TOTAL	70	39	31	30	40

had blood losses of more than 5 ml/kg (as determined by weighing of sponges), were given drugs other than 10 ml physiologic saline solution with 1:200,000 epinephrine for local vasoconstriction during 2–4 hours of operation, or needed suctioning of the respiratory tract or re-draping during the operation. After exclusion of the above-mentioned patients, 30 patients of the ether group and 40 patients of the halothane group were analyzed.

All operations were performed in the morning in the same air-conditioned operating room. Sterilized surgical drapes with the same number of sheets folded in the same fashion were placed on each patient in such a manner that the lower body distal to the inguinal region was exposed to room air. Whenever the rectal temperature exceeded 38 C, sponges soaked with alcohol or icebags were placed on the lower portion of the body. A cooling fan was also occasionally used.

Rectal temperature was measured by a probe inserted 6 cm and skin temperature by a probe on the ventral aspect of the distal phalanx of the left big toe,[5] held in place with adhesive tape. Temperatures were recorded immediately before induction of anesthesia and every 15 minutes thereafter with a Shibaden meter.

A dry–wet thermometer was placed on the operating table 1 meter from the patient's toe and temperatures were recorded every 15 minutes.

Arterial blood samples were drawn anaerobically from the dorsalis pedis, posterior tibial, or femoral artery of the right leg immediately after induction of anesthesia and hourly therafter. Pa_{O_2} and *p*H were measured using an Astrup apparatus within 15 minutes of sampling; Pa_{CO_2} and base excess were calculated using the Siggaard-Andersen nomogram.‡

The patients were divided into two groups according to the anesthetic agents used (ether group and halothane group). Student's t test was used for statistical analyses, with the assumption that the two groups had the same population variance. Results are given as means ± SE; *P* equal to or less than 0.05 was considered significant unless otherwise stated. Also, with the assumption that two groups had different variances, Welch's test was used. The results were the same as those obtained by t test.§

Results

Distribution of the patients by age, sex, and agents used is shown in table 1. Body weights of the children in each age group were within normal limits. The average operating times for repair of harelip (n = 38) and repair of cleft palate (n = 32) were 131 and 129 minutes, respectively. Anesthetic times ranged from 110 to 270 minutes, with a mean of 166.1 minutes. Average blood losses, 2.2 ± 0.1 ml/kg/h (n = 60), were the same with both types of operations. Anesthetic times and blood losses were the same in the ether and halothane groups.

None of the patients had any "unexplained sudden rise" in temperature. None had convulsions.

Room temperature of the dry bulb was 24.7 ± 0.2 C (n = 65) and that of the wet bulb was 19.9 ± 0.3 C (n = 49) during induction of anesthesia; these temperatures were 25.3 ± 0.2 and 20.4 ± 0.3 C, respectively, 120 minutes after induction. Relative humidity was 63–65 per cent. Room temperatures were the same for the ether and halothane groups. Rectal and skin temperatures were the same in both groups before induction.

Rectal temperature exceeded 38 C in 17 of 30 patients in the ether group (highest temperature 39.5 C) and in 12 of 40 patients in the halothane group (highest temperature

‡ Technical assistance was provided by Miss Mitsuko Ohi.

§ Mr. Takashi Miyawaki, Department of Statistics, Yale University, assisted with the statistical analyses. Calculations were done using programs ANOVA and T-TEST in the DATATEXT at the Yale Computer Center.

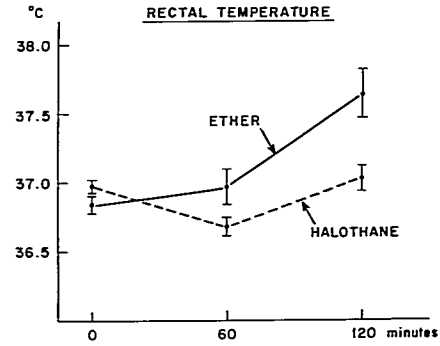

FIG. 1. Changes of rectal temperatures with time during ether and halothane anesthesia. For explanation, see Results.

38.5 C). Temperature elevations were significantly more frequent with ether than with halothane ($P < 0.02$, Chi square test).

Figure 1 shows the average rectal temperatures during anesthesia. In the ether group, rectal temperatures 60 minutes after induction of anesthesia showed no significant change (37.0 ± 0.1 C), but they rose significantly (37.6 ± 0.2 C, $P < 0.05$) 120 minutes after induction. In the halothane group, rectal temperatures 60 minutes after induction decreased significantly (36.7 ± 0.1 C, $P < 0.05$), then returned to preinduction levels (37.0 ± 0.1 C) 120 minutes after induction. The temperature differences between the ether and halothane groups were significant (at 60 minutes, $P < 0.05$; 120 minutes, $P < 0.001$).

The intervals between induction and the times when rectal temperatures reached 38 C were 99.8 ± 7.5 minutes (n = 17) in the ether group and 150.4 ± 13.4 minutes (n = 12) in the halothane group, a statistically significant difference.

Table 2 shows average skin temperatures 0, 60, and 120 minutes after induction of anesthesia in the two groups of patients. Skin temperatures decreased continuously and significantly with both agents. The decrease was more prominent with ether than with halothane. The difference between the two agents was statistically significant at 120 minutes.

The differences between rectal and skin temperatures (internal gradient) increased significantly at both 60 and 120 minutes with both agents. This increase in internal gradient was significantly greater with ether than with halothane (table 3).

Pa_{O_2}, Pa_{CO_2}, pH, and base excess were the same in both groups immediately following induction of anesthesia. There were slight but significant increases in Pa_{O_2} at 60 and 120 minutes with both anesthetics. Pa_{CO_2} was significantly decreased ($P < 0.01$) during ether anesthesia both 60 and 120 minutes after induction. During halothane anesthesia, Pa_{CO_2} decreased significantly only 120 minutes after induction. The differences between the Pa_{CO_2}'s in the two groups at 60 and 120 minutes were highly significant ($P < 0.01$, table 4).

There were significant decreases in base excess in the ether group at both 60 minutes

TABLE 2. Skin Temperatures

Min	Ether (n = 30)	Halothane (n = 37)
0	31.6 ± 0.4	32.5 ± 0.3
60	29.6 ± 0.5*	30.8 ± 0.4†
120	28.4 ± 0.7†‡ (n = 28)	30.3 ± 0.9†‡

TABLE 3. Internal Gradients

Min	Ether (n = 30)	Halothane (n = 36)
0	5.2 ± 0.4	4.5 ± 0.4
60	7.4 ± 0.5†‡	5.8 ± 0.4*‡
120	8.8 ± 0.7†‡ (n = 27)	7.0 ± 0.5†‡

* Significant change from control, $P < 0.05$.
† Significant change from control, $P < 0.01$.
‡ Significant change between agents, $P < 0.05$.

TABLE 4. Blood–Gas Values in the Ether and Halothane Groups

	Ether			Halothane		
	0	60 Min	120 Min	0	60 Min	120 Min
Pa_{O_2} (torr)	103.4 ± 5.7 (n = 22)	135.8 ± 6.7† (n = 23)	132.1 ± 5.9† (n = 22)	115.0 ± 9.0 (n = 28)	125.8 ± 6.2* (n = 28)	127.0 ± 6.5* (n = 29)
Pa_{CO_2} (torr)	39.4 ± 1.6 (n = 24)	34.5 ± 1.2†‡ (n = 20)	30.7 ± 1.0†‡ (n = 19)	41.7 ± 1.2 (n = 28)	40.8 ± 1.2‡ (n = 26)	37.8 ± 1.3*‡ (n = 26)
*p*H	7.32 ± 0.01 (n = 25)	7.32 ± 0.12 (n = 20)	7.31 ± 0.01 (n = 19)	7.30 ± 0.01 (n = 35)	7.28 ± 0.02 (n = 33)	7.31 ± 0.01 (n = 32)
Base excess (mEq/l)	−5.7 ± 0.5 (n = 24)	−7.4 ± 0.5* (n = 20)	−7.6 ± 0.5* (n = 19)	−5.8 ± 0.4 (n = 28)	−6.5 ± 0.4 (n = 26)	−7.0 ± 0.5 (n = 26)

* Significant change from control, $P < 0.05$.
† Significant change from control, $P < 0.01$.
‡ Significant change between agents, $P < 0.001$.

and 120 minutes. In the halothane group, the decreases in base excess were not significant (table 4). There was no change in *p*H in either group.

There was no correlation between rectal temperature and Pa_{CO_2} or base excess. Mean Pa_{CO_2} and base excess 120 minutes after induction in those patients whose rectal temperatures rose above 38 C were 34.0 ± 1.3 torr (n = 22) and −7.6 ± 0.4 mEq/l (n = 21), respectively. These values are not significantly different than those obtained in patients whose temperatures did not rise. Pa_{CO_2} and base excess in the latter at 120 minutes were 32.7 ± 2.5 torr and −7.0 ± 0.5 mEq/l, respectively.

During ether anesthesia, rectal temperature was significantly higher and skin temperature significantly lower in the 7–12-month age group than in the 19–24-month and the 2–6-year age groups. Changes in temperatures during halothane anesthesia were not related to age (fig. 2).

Discussion and Conclusion

The lower Pa_{CO_2} in the ether group in this study probably did not result from increased ventilation secondary to increased body temperature, because 60 minutes after induction, at a time when there was a significant decrease of Pa_{CO_2}, rectal temperature remained unchanged. Although Pa_{CO_2} was lower with ether than with halothane, increased respiratory work secondary to the respiration stimulating effect of ether[6] cannot be considered a cause of increased body temperature since the Pa_{CO_2} of the patients whose rectal temperatures rose above 38 C was the same as that of patients whose temperatures did not become elevated.

Ether, in contrast to halothane, increases circulating catecholamine levels in man.[7] Norepinephrine is increased in adults, whereas epinephrine is more elevated in children.[8,9] Epinephrine, which increases oxygen consumption and elevates body temperature, has a calorigenic effect which norepinephrine does not have. The calorigenic effect of epinephrine is also more pronounced in younger age groups.[10] It is, therefore, possible that ether anesthesia in infants and children elevated the blood epinephrine level, which resulted in increased metabolism reflected in the decreased base excess seen in this study, thereby producing excess heat and so causing elevation of body temperature. It is unlikely that the metabolic acidosis observed in the study was secondary to increased body temperature, since it appeared when rectal temperature was still normal.

Skin temperatures in the ether group decreased progressively and remained lower than during halothane anesthesia. The peripheral vasoconstriction reflected in the decreased peripheral skin temperature may, therefore, have contributed to the disturbance of heat loss associated with ether.

Eger and associates found[11,12] increased cutaneous blood flow and temperature during light planes of ether anesthesia without a subsequent decrease in temperature during maintenance of prolonged anesthesia. The reasons for the discrepancy between our findings and those of Eger *et al.* probably

include: 1) in Eger's study, body temperature was maintained at control level by changing room temperature, whereas in our study room temperature was kept constant; 2) Eger's subjects were adults, and ours were between 5 months and 6 years old; 3) in Eger's study a skin-temperature thermistor was taped onto the forearm or dorsum of the hand, whereas in the present study the big toe was used to measure skin temperature.[5]

It is interesting that in the present study peripheral skin temperatures decreased progressively during anesthesia, a decrease more marked with ether than with halothane. Rectal temperatures, however, tended to rise as anesthesia progressed, the change with ether again being more pronounced than that with halothane. The difference between rectal and peripheral skin temperatures (internal gradient)[13] increased at both 60 and 120 minutes with both ether and halothane, increasing more with ether.

From the present study it is concluded that in infants and children, rectal temperatures rise more frequently, more rapidly, and to higher levels with ether anesthesia than with halothane anesthesia.

Neither CO_2 accumulation nor abnormal metabolism was observed in patients whose rectal temperatures rose above 38 C.

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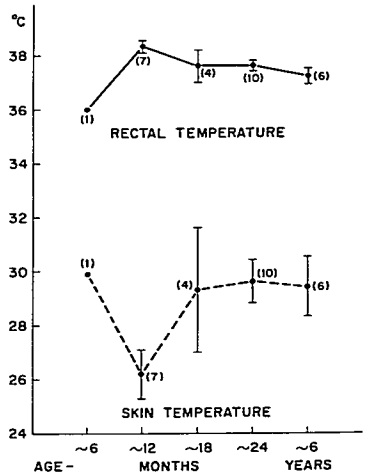

FIG. 2. Rectal and skin temperatures of children of different ages 2 hours after starting ether anesthesia. ( ) indicates number of patients. For explanation, see Results.

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