the couplant water. Despite these changes, it should be remembered that ultrasonic energy has great potential for localized heating and a fire hazard exists when such heating occurs in an oxygen-enriched atmosphere. Users of ultrasonic equipment should be apprised of this hazard, be instructed in the early recognition of malfunction, and be supported by technical specialists who can perform routine checks of the efficiency and safety of equipment.[4]

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References

1. Gauthier WD: Characteristics of therapeutic mist and humidification, Humidification and Mist Therapy. Edited by D Allan. Int Anesthesiol Clin 8:383–595, 1970
2. Stevens HR, Albrect HB: Assessment of ultrasonic nebulization. Anesthesiology 27:648–653, 1966
3. Egan DF: Fundamentals of Inhalation Therapy. St. Louis, C. V. Mosby, 1969, pp 224
4. Green HL, Hieb GE, Schatz IN: Electronic equipment in critical care areas: Studies of devices currently in use. Circulation 43, A 101:22, 1971

Respiratory Excretion of Halothane after Clinical and Occupational Exposure

Thomas H. Corbett, M.D.,* and Gwendolyn L. Ball, M.S.†

Corbett and Ball[1] reported levels of methoxyflurane between 2 and 10 ppm in the inhalation zone of the anesthesiologist under usual working conditions, and measurable levels of methoxyflurane in end-expired air of anesthesiologists for as long as 30 hours following exposure. Patients had detectable levels of methoxyflurane in end-expired air for as long as 18 days after anesthesia. These findings prompted us to perform similar studies with halothane.

Methods

The rates of excretion of halothane were determined by measuring concentrations in the end-expired air of the subjects at random intervals following exposure. Samples were collected in bags impermeable to diffusion of halothane. Subjects were instructed to inhale, exhale about two thirds, and then breathe into the bag.

Halothane concentrations were measured in parts per million (ppm). Patient breath levels were determined using a Beckman GC-2A gas chromatograph equipped with a column of 10 per cent diisodecyl phthalate on 50/60 mesh Chromosorb P and a flame ionization detector. The limit of detection was 40 parts per billion (ppb). Anesthesiologist breath levels and environmental concentrations were determined using a Beckman GC 72-5 gas chromatograph with a similar column and a flame ionization detector. The limit of detection was 4 ppb. At low levels care was taken to avoid contamination of the syringe by previous samples injected into the chromatograph.

Under clinical conditions it was not possible to determine separately the contributions of concentration and length of exposure to the decay curves, as is done in experiments on human volunteers.[2] Although differing exposure times were chosen, patients' inspired concentrations changed during clinical anesthesia. To facilitate comparison of exposures, halothane

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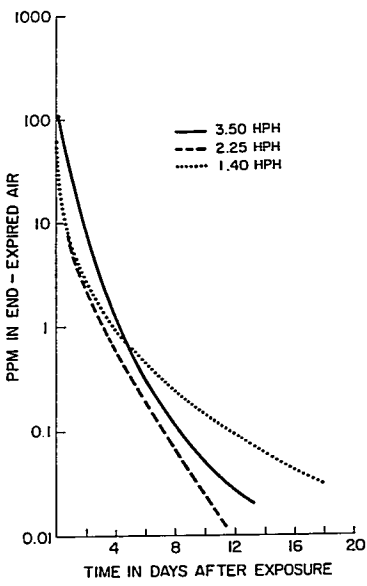

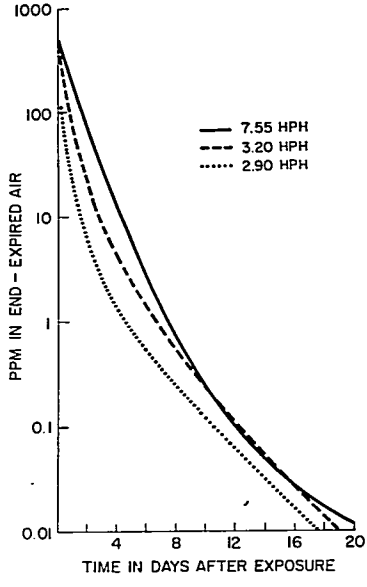

FIG. 1 (*left*). Respiratory excretion of halothane by patients after exposures of 1.40, 2.25, and 3.50 HPH. The numbers of samples taken to determine the curves were 17, 16, and 12, respectively.

FIG. 2 (*right*). Respiratory excretion of halothane by patients after exposures of 2.90, 3.20, and 7.55 HPH. The numbers of samples taken to determine the curves were 15, 18, and 19, respectively.

per cent hours (HPH) was calculated by the following formula:

$$\text{HPH} = \sum \left(\begin{matrix} \text{per cent halothane} \\ \text{in inspired air} \end{matrix} \times \frac{\text{time administered}}{60 \text{ (min)}} \right)$$

RESULTS

Six patients with no history of cardiovascular, pulmonary, hepatic or renal disease were studied. Preanesthetic samples of end-expired air were collected as controls.

Patient exposures to the halothane ranged from 1.40 to 7.55 HPH. Halothane was detectable in end-expired air for 11–20 days following exposure. The numbers of postanesthesia determinations per patient ranged from 12 to 19 (figs. 1 and 2).

Five-breath decay curves were determined after various lengths of exposure of one anesthesiologist to halothane under usual working conditions. Control samples before each exposure showed no measurable levels of halothane. Exposure times ranged from 70 to 390 minutes. Halothane was detected in end-expired air for 26 hours (70-minute exposure) to 64 hours (390-minute exposure) (fig. 3).

The effect of repeated exposure to halothane in the same anesthesiologist described above is shown in figure 4. The first exposure was for

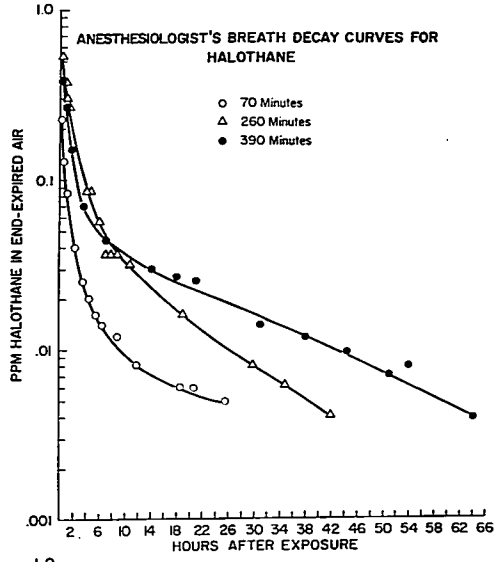

FIG. 3. Respiratory excretion of halothane by the same anesthesiologist following exposures of 70, 260, and 390 minutes.

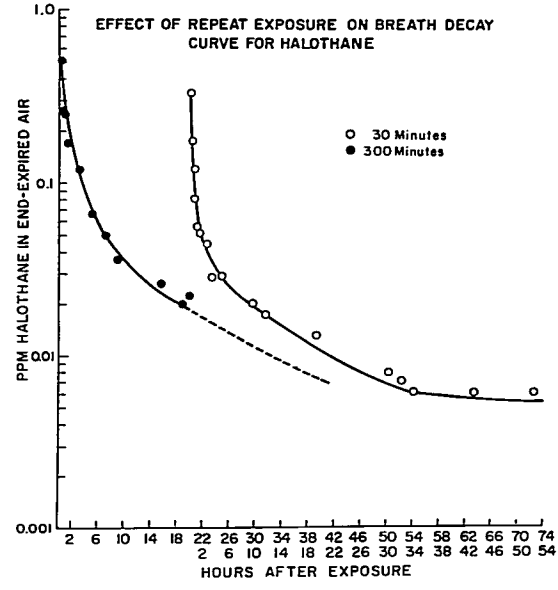

FIG. 4. Respiratory excretion of halothane by the same anesthesiologist following an initial exposure of 300 minutes and a second exposure of 30 minutes 24 hours later.

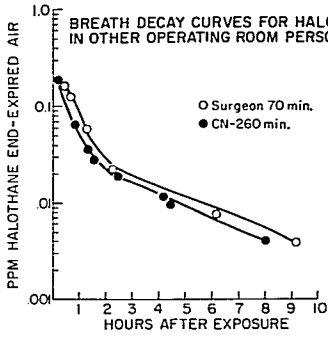

FIG. 5. Respiratory excretion of halothane by a surgeon following a 70-minute exposure and a circulating nurse following a 260-minute exposure.

300 minutes. Twenty-four hours later, the subject was again exposed for 30 minutes. Halothane was detectable for 74 hours following the second brief exposure.

Respiratory excretion of halothane was determined for two surgeons, each with a 70-minute exposure, and for a circulating nurse with a 260-minute exposure (fig. 5). The surgeons had almost identical rates of excretion, each with detectable levels of halothane for nine hours following exposure. The circulating nurse had detectable levels for eight hours following exposure.

Fifteen determinations of halothane levels in operating room air at either 0.5 per cent or 1.0 per cent concentration delivered from the same Fluotec vaporizer were made during administration of anesthesia using a semiclosed system and a 5-l/min total gas flow rate. Specimens collected from the inhalation zone of the anesthesiologist ranged from 1–2 ppm (seven samples) at 0.5 per cent to 4–10 ppm (eight samples) at the 1.0 per cent setting.

Ten determinations of halothane concentrations were made in each of three areas adjacent to the operating rooms. Concentrations of 0.14–0.25 ppm were found in the main hallway leading to the operating rooms. In the anesthesia workroom 0.02–0.04 ppm was found, and 0.10–0.22 ppm was found in the recovery room. These levels, although measurable, are considerably lower than those found in the operating rooms.

DISCUSSION

Our studies have demonstrated measurable levels of halothane in patients for as long as 20 days following anesthesia. We have also demonstrated significant accumulations of halothane in operating room personnel following occupational exposure. Toxicity studies of chronic exposure to low concentrations of anesthetic gases are lacking. Recent reports [3-4] suggest a possible relationship between health problems and chronic exposure to low concentrations of anesthetic gases. Although no relationship has as yet been established, exhaustion of waste anesthetic gases from the operating room through the use of effective gas-scavenging devices on anesthesia machines has been suggested.

REFERENCES

1. Corbett TH, Ball GW: Chronic exposure to methoxyflurane: A possible occupational hazard to anesthesiologists. ANESTHESIOLOGY 34: 532–537, 1971
2. Stewart RD, Dodd AB, Gay HH, et al: Experimental human exposure to trichloroethylene. Arch Environ Health 20:64–71, 1970
3. Cohen EN, Belville JW, Brown BW: Anesthesia, pregnancy and miscarriage: A study of operating room nurses and anesthetists. ANESTHESIOLOGY 35:343–347, 1971
4. Corbett TH, Cornell RG, Lieding K, et al: Incidence of cancer among Michigan nurse-anesthetists. ANESTHESIOLOGY 38:260–263, 1973