A CLINICAL COMPARISON OF CHLOROFORM AND HALOTHANE BY A BLIND STUDY TECHNIQUE

BETTY J. BAMFORTH, M.D., KARL L. SIEBECKER, M.D., JOHN E. STEINHAUS, PH.D., M.D., O. SIDNEY ORTH, PH.D., M.D.

MANY attempts have been made to find a nonexplosive, potent, nontoxic inhalation anesthetic agent. One compound screened by laboratory investigation, and after extensive clinical investigation made available commercially, is halothane, 2-bromo-2-chloro-1,1,1-trifluoroethane (Fluothane). When the initial reports of this drug were made, we were impressed by the description of a new nonexplosive potent agent. After clinical trial, we were impressed by the apparent similarity to chloroform. The present study was devised in an attempt to discover whether it is possible in the routine administration of these two agents to distinguish between them by purely clinical means.

METHOD OF STUDY

Bottles containing either halothane or chloroform were labeled using a series of code numbers known only to one of us, and were selected at random for use by the anesthesiologists. The code was changed weekly, so that one could not repeatedly use the same agent.

The anesthetics were administered by anesthesiology residents in training, supervised by a staff anesthesiologist, after the residents had been indoctrinated into the clinical use of chloroform and were familiar with its potency and dangers. The individuals using these agents were requested not to attempt identification by smelling the material, but to note carefully the clinical effects.

The agent was to be used as a supplement to nitrous oxide-oxygen anesthesia after preliminary medication with a belladonna alkaloid, and an opiate if desired. Induction of anesthesia was to be accomplished with a minimal dose of thiobarbiturate intravenously, or rectally in children. Succinylcholine could be used as needed to facilitate intubation of the trachea. All patients' tracheas were intubated, and topical spray was used rarely. A measured amount of the volatile agent was then placed in a Foregger "Vinethene" vaporizer and vaporized by blowing a nitrous oxide-oxygen mixture over the surface of the liquid. Administration was by means of a semiopen technique, using a nonrebreathing valve, or with an absorption technique, high flows and a "pop-off" valve using about 30 per cent oxygen in most instances. When needed, nonvolatile supplements were added in the manner in which we might customarily use them for supplementing a nitrous oxide anesthetic. It was urged that the unknowns be used in the careful fashion with which we are accustomed to giving chloroform. The level of anesthesia was maintained in plane 1 or 2 of stage III as nearly as could be judged. Every effort was made to maintain adequate pulmonary ventilation. Thirty-nine arterial blood samples were obtained for determination of oxygen, carbon dioxide and *p*H as a check on the adequacy of ventilation. Twenty of these samples were obtained during chloroform anesthesia and 19 during administration of halothane. The *p*H determinations were made on a Beckman Model G *p*H meter a few minutes after the sample had been drawn. The carbon dioxide and oxygen contents were determined by the Van Slyke manometric method and the P_{CO_2} calculated from the nomogram of Singer and Hastings. In some of the patients the oxygen concentration of the inspired atmosphere was determined and found to be always greater than 25 per cent.

The patients ranged in age from six months to 79 years, the age distribution was similar in both groups and more than half of them were adults between 30 and 59 years. Sex and physical status were distributed evenly.

Received from the Department of Anesthesiology, Medical School, University of Wisconsin, Madison, Wisconsin, and accepted for publication January 19, 1960. Dr. Steinhaus' present address: Emory University, Atlanta, Georgia.

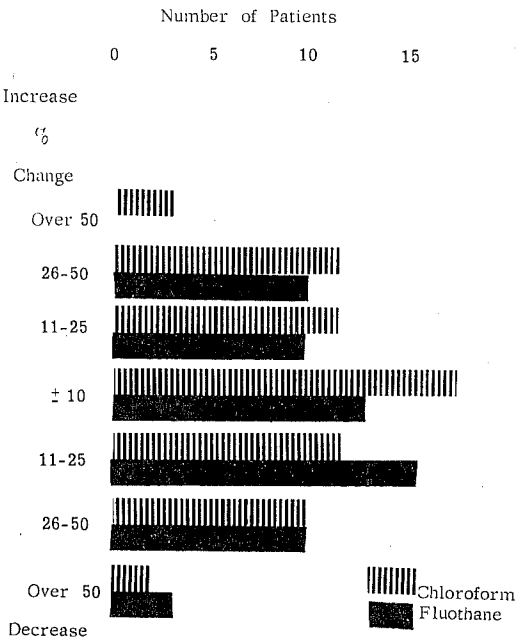

FIG. 1. The percentage change in systolic blood pressure from preoperative readings during anesthesia supplemented with chloroform or halothane (Fluothane).

The majority of operations were neurosurgical procedures. No abdominal or thoracic operations were included.

Intravenous supplementation with opiates was rarely used and there seemed to be no statistical difference in the amounts used with either agent. The average total dosage of thiobarbiturate administered for induction and maintenance of anesthesia was 3.25 mg./pound of body weight, ranging from 1.1 mg./pound to 7.3 mg./pound, with no apparent difference in requirement produced by either of the volatile supplements. This information seems to indicate that the two groups of patients were fairly comparable in several respects. The clinical observations of the effects of the supplementary volatile anesthetic agent were then studied.

RESULTS

Effect on Blood Pressure. The changes in systolic blood pressure observed during the anesthetic administrations are presented in figure 1. These values are reported as a percentage change from the preanesthetic readings for the greatest rise and fall of systolic blood pressure. The percentage changes correspond rather well to the changes as recorded in millimeters of mercury, and show that in this series of patients there was a slightly greater incidence of hypotension in patients who were given halothane. The changes of diastolic pressure were smaller, but similar in direction and number. There were, of course, a great many variables during operation, such as blood loss, postural changes, surgical stimulation, trauma and other drugs used, as well as the effect of the anesthetic agent. In order to eliminate some of these, the changes in blood pressure after the first fifteen minutes of administration of the supplementary volatile agent were analyzed. In most instances the unknown drug was not introduced into the anesthetic system until anesthesia was well stabilized following the barbiturate induction, and the operation had not yet started. We believed that changes occurring at this time might more closely reflect the influence of these drugs on the blood pressure. These changes are summarized in figure 2, in which the change in the systolic blood pressure is recorded in millimeters of mercury, and is also expressed as a percentage of the systolic pressure determined just before the addition of the unknown agent. Here again a slightly greater increase is seen in the incidence of hypotension with the administration of halothane.

Effect on the Pulse Rate. The change in pulse rate at the end of 15 minutes of the administration of the unknown drug are represented in figure 3. Changes in rate of 10 per cent or less were not considered to be measurable with any degree of accuracy under the conditions of this study. It will be noted that 22 patients receiving chloroform and only 12 of the patients who were given halothane showed a slowing of more than 10 per cent at the end of the first fifteen minutes of administration of the volatile drug. The incidence of bradycardia is then significantly higher in the chloroform group. The number of patients exhibiting an increase in pulse rate is small in either group.

Effect on Respiration. In the majority of the patients studied there was but little change in the rate of respiration. There were several

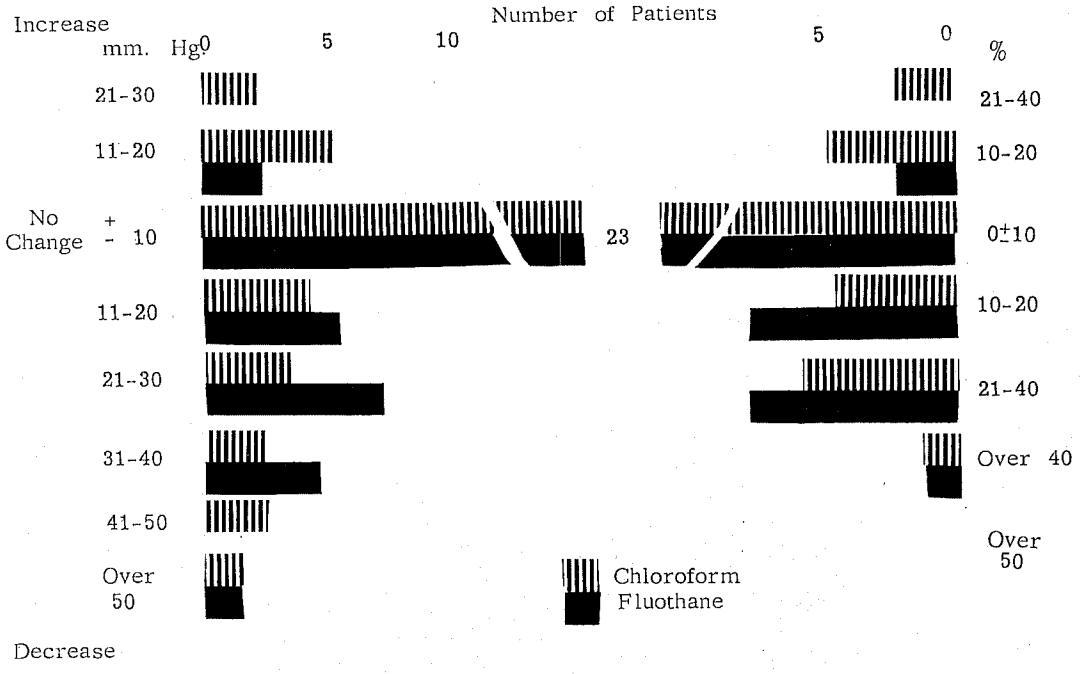

FIG. 2. Change in systolic blood pressure in millimeters of mercury and percentage change after 15 minutes of chloroform or halothane (Fluothane).

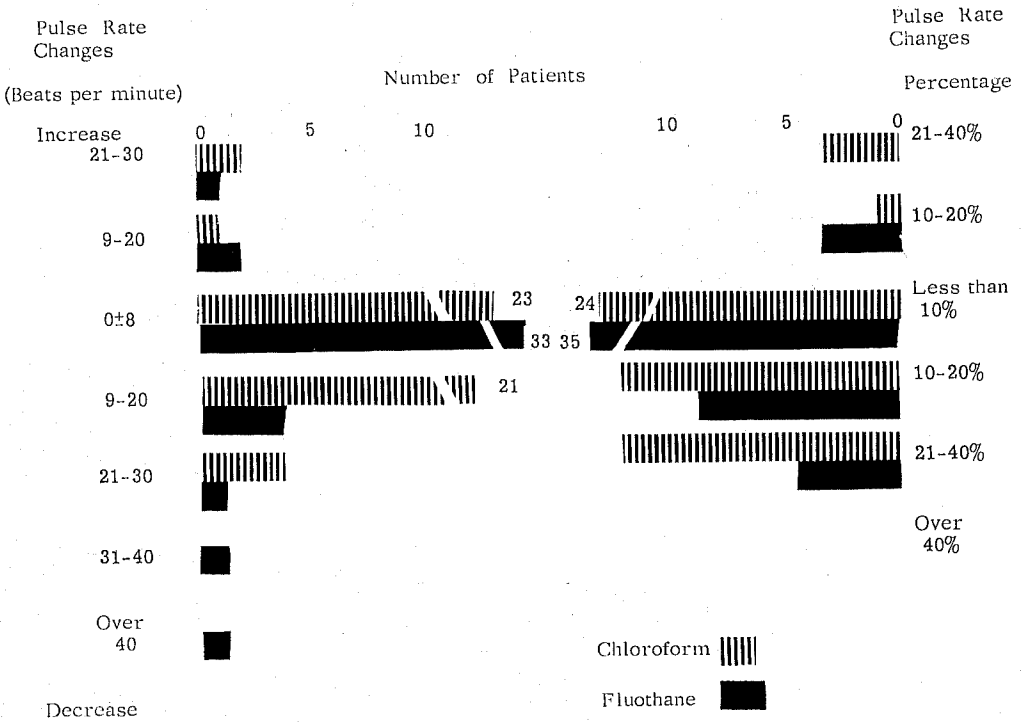

FIG. 3. Change in pulse rate after 15 minutes of chloroform or halothane (Fluothane).

anesthetic records in each group on which the anesthesiologist had noted that there had been apparent volumetric depression of respiration during short periods of greater depth of anesthesia. The arterial blood analyses revealed that in all but four instances the *p*H values ranged from 7.35 to 7.45 and the P_{CO_2} below 47 mm. of mercury with one exception. There were three patients in whom the oxygen saturation of the blood fell below 92 per cent. One patient receiving halothane had a period of severe coughing and breath-holding during positioning for a suboccipital craniotomy. A blood sample taken at this time revealed a *p*H of 7.22, P_{CO_2} of 70 mm. of mercury and oxygen saturation of 87 per cent. A sample taken later showed a return to normal in all respects. He suffered no apparent ill effects and had an uneventful recovery. No blood samples were obtained from any of the patients who died.

In table 1 the complications noted by the anesthesiologist during the administration of the anesthetic are tabulated. Some patients exhibited more than one such cause of anesthetic dissatisfaction. The clinical impression of those administering these anesthetics is that halothane produced slightly greater difficulty in providing depression of cough reflexes, in avoiding occasional movement of the patient in response to surgical stimulation, and in depression of blood pressure. The latter was generally transitory and reversible. The two patients who suffered cardiac arrest will be described in greater detail. Complications during recovery from the anesthetic and the presence of reflexes at that time are summarized in table 2. Recovery was complicated

TABLE 1

COMPLICATIONS DURING ANESTHESIA

	Chloroform	Halothane
None	28	22
Vomiting	1	1
Hypertension	1	0
Hypotension	5	9
Bradycardia	1	0
Other Arrhythmias	4	2
Cardiac Arrest	2	0
Respiratory Depression	0	1
Cough	10	14
Obstruction	2	1
Inadequate Relaxation	0	4
Others	1	2

TABLE 2

COMPLICATIONS DURING RECOVERY

	Chloroform	Halothane
None-Reflexes Present	35	44
Retching	3	0
Emesis	2	0
Obstruction	2	0
Dead	2	0

in some patients by retching and emesis in the chloroform group, although recovery of protective reflexes seemed to be quite rapid after both agents. Most of these patients had protective reflexes and were responsive to verbal stimuli while still in the operating room.

Postoperative Complications. There were a large number of postoperative complications of various types in this series of 100 patients, as might be anticipated from the type of operations included. Especially in the neurosurgical group, where depressed levels of consciousness were not unusual, pulmonary and circulatory problems were frequent. In none of these patients, however, was there respiratory or circulatory disturbances which could be related to the anesthetic administration. Indeed, it seems likely that the prompt recovery of protective pharyngeal reflexes, with the avoidance of long periods of depression sometimes produced by the nonvolatile supplements used with nitrous oxide, may have been advantageous. There was no suggestion of hepatic dysfunction in any of these patients during the postoperative period.

Deaths. Six of the patients in this study died in the hospital, 4 after receiving chloroform and two after receiving halothane. Two of those in the chloroform group died in the operating room. A brief summary of each of these cases is presented. The deaths in the operating room are discussed in greater detail.

Case 1. Chloroform was used as the supplementary agent during operation for excision of a cerebral meningioma. The patient remained comatose during the postoperative period, and died on the ninth postoperative day after having developed an uncontrollable auricular fibrillation on the third postoperative day and exhibiting signs of marked cerebral edema.

Case 2. Chloroform supplement during removal of a pituitary tumor, was followed by recovery of reflexes in the operating room. Death occurred ten hours postoperatively, when she be-

came deeply comatose, cyanotic, developed bradycardia and died rather suddenly.

Case 3. During anesthesia with nitrous oxide and halothane this 52 year old woman had a period of respiratory failure shortly after craniotomy was begun, presumably from increased intracranial pressure, and medullary failure. She remained comatose, with Cheyne-Stokes type respiration until her death three hours postoperatively of circulatory and respiratory failure.

Case 4. Halothane and nitrous oxide were used for craniotomy for the removal of a brain tumor. There was a brief period of bigeminal rhythm during the operation, which responded to the discontinuance of the agent. The patient was responding at the time he left the operating room. He died on the third postoperative day, and autopsy revealed the presence of epidural hematoma, cerebral edema and hemorrhage.

Case 5. A 68 year old female was to undergo craniotomy for a frontoparietal tumor. Anesthesia was begun with thiobarbiturate and succinylcholine, tracheal intubation was accomplished and a craniotomy was started. Anesthesia was being maintained, approximately in plane 1, stage III with nitrous oxide at seven liters per minute and oxygen three liters per minute. Chloroform was being administered as a supplement. Blood pressure was stable at 140/90 and the pulse rate was steady at a rate of 88 per minute.

When the skull was opened, the sagittal sinus was torn and profuse hemorrhage occurred. The anesthetic agents were immediately turned off, and oxygen alone was used for the rest of the procedure. It was impossible to replace the blood rapidly enough to maintain a level of blood pressure compatible with survival. One hour later cardiac arrest occurred and resuscitative efforts were ineffective. The anesthetic agent cannot be completely absolved here as being contributory to the hypotension; however, the massive hemorrhage was thought to be the cause of death.

Case 6. A 67 year old man was admitted to the hospital with the chief complaint of slowly increasing dizziness of about six weeks duration. After complete examination, a tentative diagnosis of brain tumor was made. Immediately after a pneumoencephalogram was performed, a suboccipital craniotomy was deemed necessary. Premedication for the pneumoencephalogram had been 60 mg. of codeine sulfate, given subcutaneously at 7:30 a.m. When the patient arrived in the operating room, at 9:00 a.m., his physical status was judged to be 3. Two tenths of a milligram each of l-hyoscyamine and hyoscine were given intravenously at 9:15 a.m. The blood pressure was recorded as 110/70. The hypopharynx was then sprayed with 4 ml. of hexylcaine (5 per cent). Four hundred milligrams of 2.5 per cent thiopental was given intravenously over a four minute period. At 9:20 a.m., 60 mg. of succinylcholine was given intravenously. The patient was then ventilated with oxygen for sixty seconds. A no. 37 (French) anode tube with cuff was placed in the trachea. Oxygen was again given until spontaneous respirations returned. Nitrous oxide and oxygen in a ratio of about 3:1 was then administered using a Stephen-Slater nonrebreathing valve. At 9:30 a.m. chloroform supplementation was added.

The patient's lower extremities were wrapped in elastic bandages. A no. 15 needle was placed in a left hand vein and 5 per cent dextrose in water was given by slow intravenous drip. Over a 25-minute period the patient was raised from supine to sitting position with cervical hyperflexion maintained by a suboccipital headrest. The patient coughed several times during this procedure. Urea solution (30 per cent in invert sugar) was given intravenously to decrease cerebrospinal fluid and brain volume, starting at 9:55 a.m. The blood pressure increased to 130/110 coincident with this; respiration and pulse rate increased from 20 and 90 respectively to 30 and 120. These vital signs returned to preoperative levels over the next 45 minutes. An electrocardiac monitor was applied with needle electrodes.

Just prior to the surgical incision the blood pressure was 110/85, pulse 80, respirations 20, and minute volume 4 liters. As the incision was made, the patient coughed and the minute volume rose to 8 liters per minute. A tracheobronchial toilet was done, but little material was obtained. The endotracheal tube was withdrawn one inch. The patient coughed again. The patient was estimated to be in the first plane of surgical anesthesia at 10:45 a.m. The blood in the incision was bright red. Nitrous oxide was discontinued shortly and the patient maintained on oxygen and small amounts of chloroform.

As the deeper muscle layers were being divided the blood was noted to be darker in color. Respirations were assisted. At 10:55 a.m. the pulse was palpable and the heart monitor was "beeping" regularly. At 10:57 a.m. the pulse could not be palpated. However, respirations were continuing unchanged, spontaneous, and adequate; and the monitor indicated a continuing regular electrical activity of the heart. The chloroform was discontinued, and 100 per cent oxygen administered. Several minutes were spent trying to auscultate heart sounds over the precordium, complicated by the difficulty presented by the awkward position and surgical draping, and attempting to measure blood pressure by both auscultation and palpation. During this time the monitor continued demonstrating regular electrical activity of the heart, and respirations were rhythmical and adequate. At this point 11.5 ml. of chloroform had been vaporized over an 87 minute period in a 10 liter per minute flow of diluent gas. From the molecular weight and specific gravity this calculates to an average chloroform concentration of 0.37 per cent.

The diagnosis of cardiac arrest was made at 11:01 a.m. when no precordial heart sounds could

be heard. The patient was then undraped and placed in the supine position; the cumbersome headrest caused further delay. Thoracotomy was performed and cardiac massage begun at 11:05. This represents a delay of eight or nine minutes from time of onset of the arrest. At this time the heart was observed to be in asystole, flabby, cyanotic and dilated. The cardiac monitor was still indicating regular rhythmical electrical activity, and spontaneous respiration continued.

Massage was continued by a senior resident in thoracic surgery. A surgically exposed vein was used to administer blood when the previously placed needle became dislodged. Epinephrine and phenylephrine were given by intracardiac injection on three occasions. The resulting heart beat was weak. Massage produced a systolic blood pressure of 60 to 70 mm. of mercury. Electrocardiographic tracings revealed an idioventricular pattern. Levarterenol was given by intravenous drip and later procaine amide was given in increments to a dose of 1 Gm. Adequate cardiac activity never returned. From 11:45 a.m. until 12:20 p.m. respiratory activity became first slow and deeper, then slower and weaker. At 12:21 p.m. ventricular fibrillation was observed grossly, and by electrocardiographic tracing. Further efforts at resuscitation were discontinued.

At the time of thoracotomy an indurated tumor was palpated behind the heart in the hilar structures. Frozen section of a node from this area revealed adenocarcinoma. Permission for an autopsy was obtained from the relatives. The autopsy revealed: Bronchogenic adenocarcinoma, partially obstructing the left main bronchus, with metastases to right lung, kidney, cerebellum, cerebrum, brain stem, regional nodes, and left adrenal; atelectasis, marked, left lower lobe; emphysema, moderate; hydrocephalus, secondary; pressure cone of brain stem, marked; pressure cone of cerebellum, marked; and edema, collateral, left cerebellum.

What role the agent played in the death of the two patients suffering cardiac arrest in the operating room is hard to say. Certainly it is difficult to exonerate the anesthetic in such a circumstance. It seems likely, when the details of these two cases are reviewed, that the management of these cases, in which technical problems of ventilation and blood replacement occupied too great a portion of the attention of the anesthesiologist, and the delay in establishing the diagnosis of cardiac asystole was of greater importance than the actual anesthetic agent used earlier in the operation. We do not believe the agent contributed to the other deaths in this series. Review of the liver sections from the two patients in whom an autopsy was performed showed no abnormalities suggestive of damage produced by the agent.

Discussion

Chloroform has been the cause of considerable controversy during the years, has variously been praised and condemned, and has an extremely bad reputation in some areas. At the time of the centennial of chloroform, the group at Wisconsin studied chloroform as though it were a new agent. The results of this re-evaluation were published in a monograph edited by Waters.[1] It was their belief that chloroform did not deserve to be abandoned as a surgical anesthetic. Its potency and its nonexplosiveness are desirable characteristics. The relatively nonirritant effects upon respiratory reflexes make it unlikely that the patient will "protect himself by refusing to inhale an improper mixture, as is the case with ether."[1] The difficulty in fine control of the percentage concentration of the vapor of chloroform, the lack of reliable physical signs of depth of anesthesia, the respiratory depression at deep levels of anesthesia, and a fall in blood pressure as a result of the action of high concentrations of chloroform on the heart and of decreased vascular tone are all features of chloroform administration which were observed. From the studies at Wisconsin it was also concluded that delayed chloroform poisoning and disturbance of liver function tests were not likely in the presence of adequate oxygenation and elimination of carbon dioxide.

Since the completion of this study, chloroform has continued to be used in our institution in certain types of cases each year. These are primarily cases in which a nonexplosive technique is indicated, and in which difficulty with ventilation is not anticipated. It is avoided whenever reasonable certainty of good ventilation is not always present, as in thoracotomies.[2] The methods of administration are generally similar to those used in this study.

Halothane has received enormous publicity and intensive investigation in the past three years. It has been highly favored by many

because of its potency and noninflammability. It shares with chloroform the advantage of other inhalation agents, in that it may be rapidly removed from the body by vigorous ventilation.[3] Intravenously administered, nonvolatile agents, on the other hand depend upon detoxification or the relatively slow removal by renal activity. Respiratory depression has been reported with the use of this agent.[4, 5, 6] The importance of assisted respiration has been stressed.

Many authors have mentioned the hypotension which is commonly produced with high concentrations of halothane.[6, 7] The recommendations for slower inductions, and that the administration of the drug not be "pushed," [7] are similar to those made for the safe administration of chloroform.[1] Accurate, careful administration, and cautious, continuous observation of the patient is of utmost importance with *both* agents.

Cardiac arrhythmias have long been feared with the use of halogenated hydrocarbons as anesthetic agents. Cardiac arrest, due probably to myocardial depression, is known to occur with chloroform, and myocardial depression has also been demonstrated with halothane.[8] With both drugs, sensitization of the myocardium to epinephrine may result in ventricular tachycardia and ventricular fibrillation.[9, 10] Bradycardia is said to be usual with halothane,[7] but is limited by atropine premedication. The slowing of the pulse rate is often used as a reliable guide to the depth of chloroform anesthesia.

The effect of halothane on liver function is of interest. Many authors have reported that hepatic function is not altered significantly by this drug.[9, 10, 11] However, reports of pathological damage to the liver have begun to appear in the literature.[12, 13, 14] The evidence suggests that liver damage is at least possible after administration of halothane, although the hazard is probably less in improperly managed cases than with chloroform. It is possible that further reports of such damage will occur. Currently at Wisconsin a series of patients are being subjected to a battery of liver function studies preoperatively, and after the administration of halothane as the sole agent in an attempt to see how the results of these more sensitive tests compare with those following chloroform anesthesia.

Summary

In a series of 100 patients anesthetized with chloroform and halothane as a supplement to nitrous oxide-oxygen anesthesia, and with the anesthesiologist not knowing which drug he was using, it was found that it was not possible to identify the agent solely by means of its clinical effect. The changes in blood pressure, in pulse rate, respiration, and complications during anesthesia were similar to the two groups of patients. We believe that halothane bears a strong clinical resemblance to chloroform.

This study was presented at the annual session of the American Society of Anesthesiologists, Inc., Chicago, Illinois, November 20, 1958.

REFERENCES

1. Waters, R. M.: Chloroform: A Study After 100 Years. Madison Wisconsin, The University of Wisconsin Press, 1951.
2. Siebecker, K. L., and Orth, O. S.: Report of seven administrations of chloroform for open thoracic operations, ANESTHESIOLOGY 17: 792, 1956.
3. Griffith, H. R., Robson, J. G., Cullen, W. G., and Gillies, D. M.: Fluothane, modern substitute for chloroform, Anesth. & Analg. 37: 316, 1958.
4. Devine, J. C., Hamilton, W. K., and Pittinger, C. B.: Respiratory studies in man during Fluothane anesthesia, ANESTHESIOLOGY 19: 11, 1958.
5. Stephen, C. R., Lawrence, J. H., Fabian, L. W., Bourgeois-Gavardin, M., Dent, S., and Grosskreutz, D. C.: Clinical experience with Fluothane; 1,400 cases, ANESTHESIOLOGY 19: 197, 1958.
6. Hall, K. D. and Norris, F. H., Jr.: Respiratory and cardiovascular effects of Fluothane in dogs, ANESTHESIOLOGY 19: 339, 1958.
7. Johnstone, M.: Human cardiovascular response to Fluothane anesthesia, Brit. J. Anaesth. 28: 392, 1956.
8. Severinghaus, J. W. and Cullen, S. C.: Depression of myocardium and body oxygen consumption with Fluothane, ANESTHESIOLOGY 19: 165, 1958.
9. Hall, K. D. and Norris, F. H., Jr.: Fluothane sensitization of dog heart to action of epinephrine, ANESTHESIOLOGY 19: 631 (Sept.–Oct.) 1958.

10. Raventos, J.: Action of Fluothane; new volatile anaesthetic, Brit. J. Pharm.: 11: 394, 1956.
11. Virtue, R. W., Payne, K. W., Caranna, L. J., Gordon, G. S., and Rember, R. R.: Observations during experimental and clinical use of Fluothane, ANESTHESIOLOGY 19: 478, 1958.
12. Virtue, R. W. and Payne, K. W.: Postoperative death after Fluothane (Current Comment), ANESTHESIOLOGY 19: 562, 1958.
13. Burnap, T. K., Galla, S. J., and Vandam, L. D.: Anesthetic circulatory and respiratory effects of Fluothane, ANESTHESIOLOGY 19: 307, 1958.
14. Jones, W. M., Margolis, G., and Stephen, C. R.: Hepatotoxicity of inhalation anesthetic drugs, ANESTHESIOLOGY 19: 715, 1958.

NEW ANALGESIC WIN 14098, a new piperidine-derived analgesic, is reported. This was used in a study of 100 patients observed during labor and was found to produce less fetal depression compared to meperidine hydrochloride. (*Groeber, W. R., and Ziserman, A. J.: WIN 14098, A New Analgesic Agent, Obst. & Gynec. 14: 743 (Dec.) 1959.*)

OBSTETRICAL ANESTHESIA The greatest help to the obstetrical anesthetist is the obstetrician who states what conditions he requires and what he intends to do, and then leaves the conduct of the anesthesia to the anesthesiologist. Of all drugs now available, the barbiturates are undoubtedly the most dangerous and least valuable in obstetrical anesthesia. Nitrous oxide, with adequate oxygen, has no ill effect on the baby. Self-administered trichlorethylene has produced an increased incidence of atelectasis in infants, and is no longer used during labor. Cyclopropane administration is limited to three minutes before the infant is born, whenever possible, to avoid undue depression of the baby's respiratory center. Muscle relaxants such as decamethonium and succinylcholine have shown no clinical evidence of passing the placental barrier, and may be used whenever profound relaxation is required. Spinal and epidural block may produce hypotension with resulting hypoxia of the fetus, but this complication need never occur, as the hypotension is easily controlled by vasoconstrictors. Epidural block has found more favor than spinal because of the relatively high incidence of headache following the latter. The epidural space is not easily determinable, and if accidental subarachnoid injection is made, a total spinal block will result. One must always be prepared to maintain respiration and blood pressure when this occurs. The total block will usually wear off in 30 to 60 minutes and labor proceed uneventfully, with no harm resulting. (*Carroll, J.: Anaesthesia for Obstetrical Manoeuvres and Operations, Canad. M. A. J. 82: 184 (Jan. 23) 1960.*)

DEXTROMORAMID Twenty mg. sufficed in 50 per cent of the patients to give pain relief for delivery. Up to 80 mg. were given in a 6 hour period. The patients were drowsy, but oriented and cooperative. There was no increase of forceps deliveries. Because of respiratory depression in babies born during the height of effect, the mother was given 5–10 mg. nalorphine at the end of the second stage of labor. This completely prevented asphyxia neonatorum. About two-thirds of the patients had no, or only insignificant, pain during delivery. (*Drasche, E., and Boch, M.: Report Concerning 100 Cases of Obstetrical Analgesia with Dextromoramid, Der Anaesthesist 8: 324 (Nov.) 1959.*)

NATURAL CHILDBIRTH A psychological explanation is offered for natural childbirth by an academic psychologist, who had experienced natural childbirth herself. Understanding three generally accepted principles of psychology helps to explain the workings of this method of childbirth. (*Senders, V. L.: An Academic Psychologist Looks at Natural Childbirth, Obst. & Gynec. 14: 817 (Dec.) 1959.*)