ELECTROCARDIOGRAPHIC STUDIES DURING INDUCTION OF ANESTHESIA USING ETHYL CHLORIDE, ETHER, VINETHENE AND CYCLOPROPANE *

ROBERT W. VIRTUE, M.D., PH.D., AND ALSON F. PIERCE, M.D.

Denver, Colorado

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IN discussing ethyl chloride as an anesthetic agent Goodman and Gilman (1) stated that "although ethyl chloride is less toxic than chloroform, ventricular fibrillation and sudden cardiac arrest may occur." In perusing their reference in support of this statement it was found that a case was cited by Guedel and Knoefel (2) in which no electrocardiogram was made and no proof was obtained that ventricular fibrillation had occurred. Wachsmuth and Eismeyer (3), in 1928, recorded electrocardiograms during 3 of 5 inductions with ethyl chloride followed by ether and observed loss of the P wave, lowering of the P wave, or its inversion. After several animal experiments they decided that the anesthesia, not trauma, was responsible. Hill (4), in 1932, reported 4 cases of ethyl chloride induction, 2 of which were recorded electrocardiographically, and in none of the 4 was cardiac irregularity found. Henderson and Kennedy (5) published the circumstances surrounding 22 deaths using ethyl chloride. The deaths were attributed to either respiratory or circulatory failure or both. Valuable precautions concerning the use of this drug were presented. Electrocardiograms were not recorded. It seems, then, that electrocardiographic evidence of ventricular fibrillation during ethyl chloride anesthesia with human subjects is lacking.

A few series of electrocardiograms made using gas-oxygen-ether or gas-oxygen have been reported. Lennox et al. (6), in 1922, found that in 15 of 50 patients there was transient displacement of the pacemaker when ether or nitrous oxide-oxygen was used. Marvin et al. (7), in 1925, reported that 7 of 30 patients given gas-oxygen-ether lost a P wave. In another series of 30 patients anesthetized with gas-oxygen, 3 showed loss of the P wave. Ward and Wright (8) obtained electrocardiograms on 16 normal subjects who were given pure nitrous oxide for induction. In 5 of the 16 the P wave diminished in height after air was given. They report "variable changes in the P wave" for the series of 16.

An extensive study on arrhythmias during anesthesia was made by Kurtz et al. (9) in 1936. They studied 41 cases using cyclopropane, 20

* From the University of Colorado Medical Center, Denver, Colorado.

using ether, 13 with procaine used locally, 11 with ethylene, 10 with nitrous oxide, 7 with vinethene, 6 with chloroform and 5 with avertin. With each agent used, except procaine, some arrhythmia developed in over 70 per cent of the cases. When procaine was employed, 46 per cent had an arrhythmia. The commonest types of arrhythmia were sinus arrhythmia, extra systoles and a diminishing displaced pacemaker. The irregularities did not appear to be related to depth of anesthesia. They reported no cases using ethyl chloride.

Two recent articles have indicated some change in the electrocardiogram during anesthesia. Lindgren and Ohnell (10), in 1949, stated that the pacemaker wandered during at least 12 of 54 cases of narcotal-nitrous oxide-ether anesthesia, and that this occurred usually when the patients were in fairly deep anesthesia. Hunter (11), in 1950, reported that while using methyl-N-propyl ether one of 36 patients had a displaced pacemaker and that another had a flat P wave.

Volpitto and Marangoni (12) and Storm (13) found no electrocardiographic irregularities of significance using pentothal and evipal.

EXPERIMENTAL DATA

To obtain evidence as to whether ethyl chloride may be safe for induction, anesthesia was induced in 106 consecutive cases by open drop on a face mask, using electrocardiographic control. Half the patients were given ethyl chloride-air and the other half ethyl chloride-oxygen. As soon as the patients lost consciousness the ethyl chloride was discontinued and ether was started. Electrocardiograms were made continuously until the patient had passed into third stage anesthesia, after which time they were taken intermittently. Following this series with ethyl chloride inductions, anesthesia was induced in 10 cases with open drop ether, in 11 with vinethene alone and in 11 with cyclopropane. In a few instances an oximeter was available, so anesthesia was induced using this instrument with ethyl chloride, vinethene and cyclopropane while electrocardiograms were recorded.

RESULTS

None of the 106 electrocardiograms taken during ethyl chloride inductions shower irregularities which could be attributed specifically to the agent. The usual results of muscle action were present, and sinus arrhythmia and sinus tachycardia were frequently seen. The P wave either disappeared completely or became so low as nearly to disappear in 47 of the 106 cases, but only after ethyl chloride had been stopped, ether had been given and third stage anesthesia had been reached. To find whether this effect was due to ethyl chloride or to ether, some inductions were carried out with ether only. The same phenomena occurred in 5 of 10 cases. Hunter's report concerning the loss of the P wave during anesthesia with methyl-N-propyl ether ap-

peared about this time, so it was decided to obtain a series using vinethene and one using cyclopropane. Eight of the 11 patients given vinethene and 8 of the 11 given cyclopropane showed the wandering pacemaker. Typical electrocardiograms are illustrated (fig. 1). When the oxime ter was used at the time the pacemaker was wandering, it registered 97 to 98 per cent saturation of the hemoglobin, the same as before induction.

DISCUSSION

Ethyl chloride was used in this series only to the point of loss of consciousness. It cannot be stated, therefore, whether or not it would have produced the same loss of P wave which occurred when the other inhalation agents were employed. It is possible that the incidence of arrhythmias in the 106 cases in which ethyl chloride-ether was given might have been greater had electrocardiograms been made throughout the third stage of ether anesthesia. Some arrhythmias may have been missed because electrocardiographic records were taken only intermittently after induction. No evidence was obtained that arrhythmia developed during induction with ethyl chloride because of the agent itself. Reports of deaths when ethyl chloride was used have not been proved to be the result of ventricular fibrillation and while they can frequently be explained on some other basis, such a possibility still remains. The timely warning by Henderson and Kennedy (5) with regard to the conduct of ethyl chloride anesthesia must be taken seriously regardless of electrocardiographic findings. The results here recorded do not permit advocating the use of ethyl chloride to a point beyond loss of consciousness of the patient.

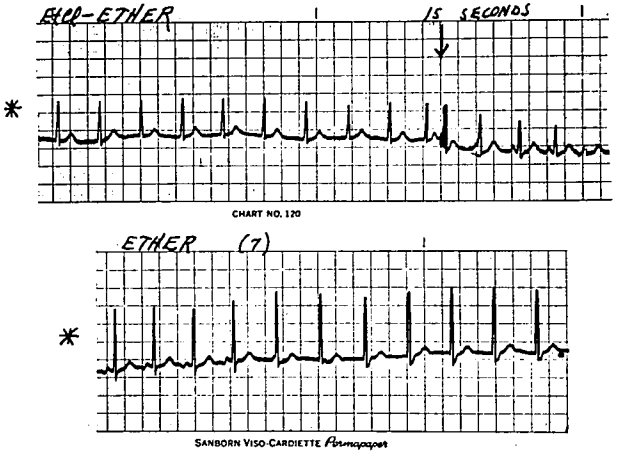

FIG. 1. * Patient was in Plane 1 of Stage 3.

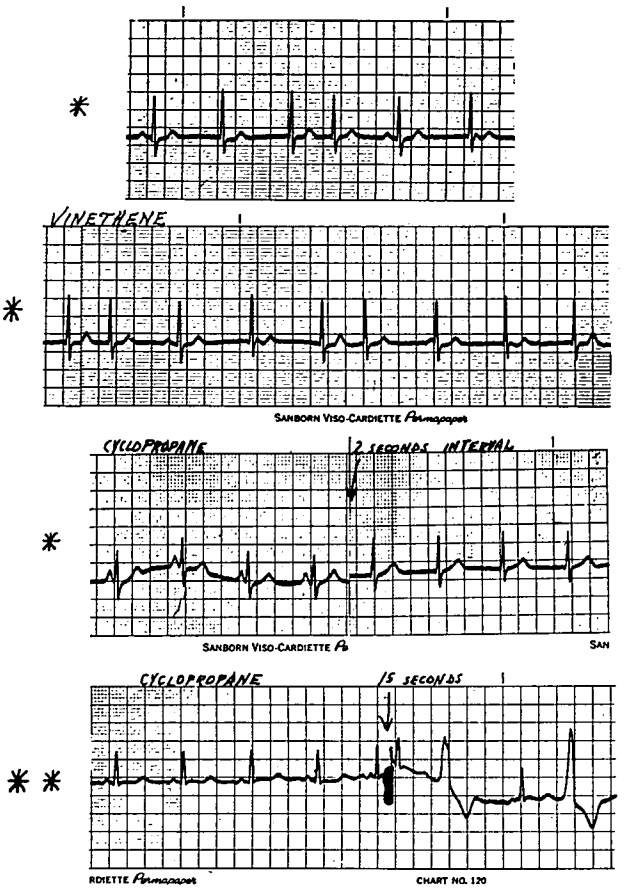

FIG. 1 (continued). * Patient was in Plane 1 of Stage 3. ** Patient was in lower Plane 2 of Stage 3. In none of these cases was there any clinical evidence of arrhythmia.

The similar results found in these experiments and those quoted by other workers with other agents, that is, loss of P wave and wandering pacemaker, lead toward the concept that these irregularities occur generally with inhalation anesthesia. The fact that they occurred when the hemoglobin was 97 or 98 per cent saturated with oxygen indicates that they were not primarily connected with anoxia.

SUMMARY

In 106 patients, when ethyl chloride was used for induction of anesthesia to the point of loss of consciousness only, no arrhythmia appeared which contraindicated the use of ethyl chloride for induction of ether anesthesia.

An electrocardiographic study revealed that a wandering pacemaker occurred frequently during first plane, third stage anesthesia using ether, vinethene or cyclopropane. This change occurred when the oxygenation of the hemoglobin was 97 to 98 per cent of saturation.

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