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ELECTROCARDIOGRAPHIC STUDIES DURING ENDOTRACHEAL INTUBATION. IV. EFFECTS DURING CYCLOPROPANE OR CYCLOPROPANE-ETHER ANESTHESIA AND INTRAVENOUS PROCAINE AMIDE * † ‡ §

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IN a previous report it was observed that during endotracheal intubation there were electrocardiographic disturbances in over 60 per cent of patients who had been anesthetized with various commonly used anesthetic agents (1). Subsequent reports have indicated that the incidence and severity of these cardiac arrhythmias which occurred during endotracheal intubation were significantly reduced when procaine hydrochloride or diethylaminoethanol was used before intubation during general anesthesia (2, 3). Since procaine amide has been shown to have an action similar to that of procaine hydrochloride on cardiac arrhythmias (4, 5), it was deemed desirable to study the effects of procaine amide on the electrocardiogram during endotracheal intubation.

Procaine amide differs from procaine hydrochloride by the presence of the amide grouping (.CO.NH.) in lieu of the ester grouping (.CO.O.). This chemical alteration results in a loss of the local anesthetic prop-

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§ The procaine amide used in these studies was obtained from E. R. Squibb and Sons, under the name "pronestyl."

erties. It also reduces the stimulating toxicity on the central nervous system so that it may be administered intravenously to conscious subjects without producing convulsions.

In the present report, an analysis is made of the effects of procaine amide on the electrocardiograms in a series of 100 patients who were intubated during general anesthesia with cyclopropane or with cyclopropane and ether. Following induction of general anesthesia, a dose of 300 mg. of procaine amide in a 10 per cent solution was injected intravenously one to five minutes before endotracheal intubation.

METHOD

Control electrocardiograms of the first three leads were obtained in all patients before anesthesia. Subsequent lead 2 electrocardiographic tracings were taken during anesthesia, during the injection of procaine amide, one minute after the injection, during intubation and after intubation. Tracings were obtained in 100 patients who were to undergo surgical intervention in which endotracheal intubation was a desired procedure.

RESULTS

Cyclopropane.—In 51 patients, cyclopropane was the sole anesthetic agent used. Of these, 26 were intubated at a depth of second plane and 25 were intubated in third plane.

Of the 26 patients who were intubated during cyclopropane anesthesia in second plane, 22 showed no change in the electrocardiogram during intubation.

Sinus tachycardia developed at the time of intubation in 3 patients. The heart rate ranged from 110 to 145 per minute, lasting an average of three minutes.

Ventricular premature contractions developed at the time of intubation in one case. This was transitory as the arrhythmia reverted to regular sinus rhythm within two minutes.

Of 5 patients whose electrocardiograms were normal in the controls, 4 had transitory nodal rhythms one minute after the injection of procaine amide. These nodal rhythms changed to sinus rhythm before intubation.

In 2 patients nodal rhythm had developed during cyclopropane anesthesia. After the injection of procaine amide there was a change to regular sinus rhythm in each case and the electrocardiograms remained normal during and after intubation.

Ventricular premature contractions with bigeminal rhythm developed during cyclopropane anesthesia in one patient. After the intravenous injection of procaine amide there was a return to a regular sinus rhythm and the electrocardiogram remained normal thereafter.

In 2 patients who displayed low T-waves in the control electrocardiograms, the amplitude of the T-waves increased during cyclopropane

anesthesia. (This may indicate improvement of myocardial oxygenation during cyclopropane-high oxygen anesthesia.)

In 5 patients, there were no electrocardiographic changes before or during endotracheal intubation, but one minute after intubation, a sinus tachycardia developed in 3 patients which lasted two minutes while in the other 2 patients ventricular premature contractions with bigeminal rhythm developed. Regular sinus rhythm recurred in less than three minutes in all these cases.

Of the 25 patients who were intubated during third plane cyclopropane anesthesia, 23 showed no change in the electrocardiograms during endotracheal intubation.

In 2 of the patients in this group bigeminal ventricular premature contractions developed during laryngoscopy and intubation. In both instances a normal rhythm returned two minutes after intubation.

Two patients manifested a nodal rhythm one minute after the injection of procaine amide. This lasted two minutes. Regular sinus rhythm recurred and remained unchanged during and after intubation.

In 2 of the patients, transitory bigeminal ventricular premature contractions developed during induction with cyclopropane. These contractions were associated with respiratory obstruction; when respiratory patency was restored the electrocardiograms returned to normal.

Nodal rhythm developed during third plane cyclopropane anesthesia in 5 patients. In 4 of these, there was a return to a regular sinus rhythm one minute after the administration of procaine amide. In the fifth case, the nodal rhythm persisted for eight minutes until three minutes after intubation before it returned to a regular sinus rhythm.

In 5 of the patients, the electrocardiogram remained normal before and during intubation, but two to five minutes after intubation there were transient runs of bigeminal ventricular premature contractions associated with "bucking" or bronchospasm.

In 4 other patients electrocardiographic disturbances were observed two minutes after intubation. In one of these there was a notable reduction in the amplitude of the T-wave. In the other 3, sinus tachycardia (from 120 to 135 per minute) developed which lasted about four minutes.

Cyclopropane and Ether.—Electrocardiograms were obtained of 49 patients who were intubated following anesthetization with cyclopropane and ether. Twenty-four were intubated in second plane and 25 in third plane anesthesia.

Eighteen of the 24 patients in which intubation was performed during second plane anesthesia showed no electrocardiographic change during endotracheal intubation.

Five patients displayed sinus tachycardia at the time of intubation at rates of 110 to 140 per minute lasting for an average of six minutes. Sinus tachycardia at a rate of 120 per minute developed in one patient during the first attempt at intubation. On the second attempt at intubation, the normal sinus rate was maintained.

Two patients manifested bigeminal ventricular premature contractions during cyclopropane-ether anesthesia. One minute after the injection of procaine amide, a regular sinus rhythm returned in both of these cases and was maintained thereafter.

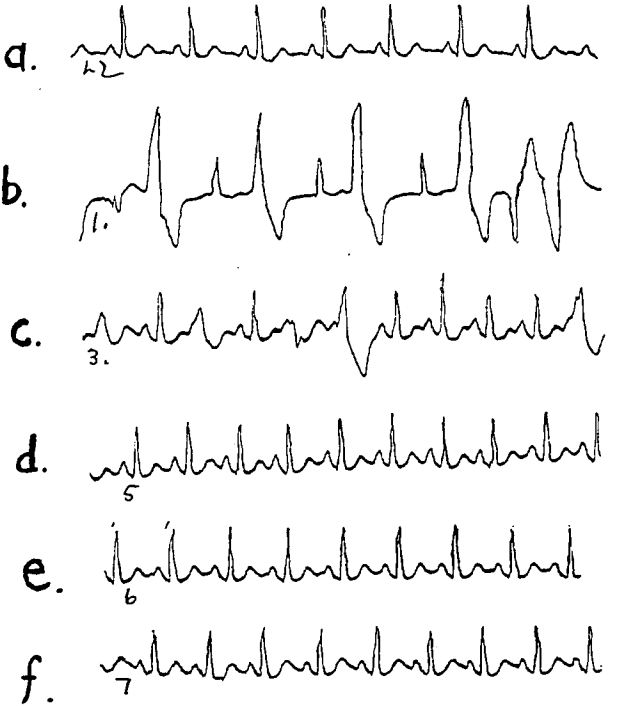

FIG. 1. Bigeminal ventricular premature contractions with brief runs of bifocal ventricular tachycardia during second plane cyclopropane-ether anesthesia until after the intravenous injection of procaine amide.

a, Control electrocardiogram, lead 2 showing regular sinus rhythm at a rate of 90 per minute.

b, During first plane of cyclopropane-ether anesthesia: bigeminal ventricular premature contractions with brief run of bifocal ventricular tachycardia.

c, During injection of procaine amide: bigeminal ventricular premature contractions.

d, During endotracheal intubation, three minutes after the injection of procaine amide: regular sinus rhythm with sinus tachycardia.

e, Four minutes after intubation: sinus tachycardia, rate 100.

f, Two minutes later: no change.

Severe laryngeal spasm developed immediately before intubation in one case. During this interval the patient had bigeminal ventricular contractions. At the time of intubation a regular sinus rhythm returned and was maintained.

Another patient in this group demonstrated bigeminal ventricular premature contractions with brief runs of bifocal ventricular tachycardia. One minute after the injection of procaine amide, a regular sinus rhythm with occasional bigeminal ventricular premature contractions occurred. At the time of intubation, there was a sinus tachycardia at a rate of 135 per minute. Three minutes later a sinus rhythm of 100 per minute occurred and was maintained (fig. 1).

In one patient an intraventricular conduction defect of an undetermined type developed one minute after injection of procaine amide. This lasted for five minutes. At the time of intubation, there was a regular normal sinus rhythm at a rate of 100 per minute which was maintained.

One patient had nodal rhythm when control electrocardiograms were taken. One minute after the injection of procaine amide there was a change to a regular sinus rhythm which was sustained.

In one patient a sinus tachycardia at a rate of 135 per minute developed after the injection of procaine amide. Immediately following intubation the rate diminished to 100 per minute.

In one case of this group a sinus tachycardia was observed one minute after intubation; this was at a rate of 120 to 140 per minute and it lasted for four minutes.

Twenty-five patients were intubated during third plane cyclopropane-ether anesthesia. Nineteen showed no electrocardiographic change during intubation.

Four patients manifested a transitory sinus tachycardia at the moment of intubation. In 2 of these the rates were 120 per minute, in another it was 130 per minute, while in the fourth it was 145 per minute.

In one patient in this group a nodal rhythm developed with ventricular premature contractions of bigeminal type at the moment of intubation. This was in a difficult case in which three attempts at intubation were required. A regular sinus rhythm returned immediately after intubation.

One patient displayed a nodal rhythm during the time of intubation. Immediately after intubation a regular sinus rhythm returned.

A nodal rhythm developed in 2 patients before the injection of procaine amide. In each case a regular sinus rhythm returned one minute after the intravenous injection of procaine amide.

In 3 patients, ventricular premature contractions of bigeminal type were observed before the injection of procaine amide. Regular sinus rhythm returned after the injection of procaine amide and endotracheal intubation produced no change.

In one patient the electrocardiogram remained normal until five minutes after intubation when a sinus tachycardia at a rate of 135 per minute was observed. This lasted five minutes when the patient's tidal volume, which had markedly diminished immediately following intubation, returned to normal limits.

COMMENT

Of the 100 cases investigated in this series, 17 (17 per cent) showed some electrocardiographic disturbances at the time of endotracheal intubation which consisted of sinus tachycardia, 13 cases, ventricular premature contractions, 3 cases, and nodal rhythm, one case. These results are comparable to those in the previous two series in which procaine hydrochloride and diethylaminoethanol were employed instead of procaine amide (2, 3).

The incidence of 17 per cent showing electrocardiographic changes at the time of endotracheal intubation in this series compares favorably with the first series in which no drug was used before intubation (1). The incidence of electrocardiographic changes in the latter group, produced at the time of intubation, was more than 60 per cent. The addition of intravenous procaine amide not only reduced the total number of cardiac disturbances but also seemed to reduce the severity of such disturbances. Thus, whereas sinus tachycardia was produced in 43 of 109 patients in the series in which no procaine or procaine derivative was used, sinus tachycardia developed in only 13 of 100 patients when procaine amide was administered intravenously before intubation. Similarly, in the present series, there were only 3 cases of ventricular premature contractions which were produced at the time of intubation as compared to 10 of 109 patients who were intubated without procaine or its derivatives. The duration and severity of this type of arrhythmia were markedly reduced.

A nodal rhythm developed in 6 patients immediately after the intravenous injection of 300 mg. of procaine amide. This rhythm lasted one to two minutes. Possibly, this effect may be the result of a depressant action of procaine amide on ventricular musculature. In 9 patients, however, a nodal rhythm developed before procaine amide was injected, and a regular sinus rhythm returned in each of these cases after procaine amide was administered.

The anesthetic agents used in this series were limited to cyclopropane and cyclopropane-ether. They comprised the agents used in the majority of cases in the previous series. There has been justifiable criticism that there were too few cases in which other anesthetic agents were used, such as nitrous oxide and ether, pentothal sodium in various combinations and the complemental use of topical anesthetic drugs. It is not our intention to omit these agents entirely; rather, we intend to study and to report separately on the results with these agents when

sufficient numbers of cases have been accumulated. The ultimate goal of these studies is to learn more about this tracheocardiac reflex and to determine what methods of endotracheal intubation are least injurious to the patient.

Of particular interest are the electrocardiographic disturbances which occur one to three minutes after introduction of the endotracheal tube in subjects who manifest "bucking" effects. It is thought that these reactions are precipitated by a temporary asphyxial condition incident to bronchiolar constriction initiated reflexly by the presence of the tube in the trachea. Further studies are being conducted in an attempt to obviate these reactions by the application of a nontoxic topical anesthetic agent.

SUMMARY

Electrocardiographic determinations were obtained on 100 patients who were intubated during cyclopropane and cyclopropane-ether anesthesia. Each patient received an intravenous injection of 300 mg. of procaine amide before intubation of the trachea. Electrocardiographic disturbances occurred in 17 per cent at the time of intubation. In the series in which procaine or its derivatives had been omitted under similar conditions, the incidence of electrocardiographic disturbances was more than 60 per cent.

Although electrocardiographic changes produced at the moment of endotracheal intubation were reduced, they were similar to those in the first series in which procaine amide was not administered. The majority of the disturbances consisted of transitory sinus tachycardia and ventricular premature contractions.

Hypoxia, insufficient depth of anesthesia and repeated attempts at intubation were among the factors which seemed to enhance the development of these reflexogenic electrocardiographic disturbances.

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