ELECTROCARDIOGRAPHIC STUDIES DURING ENDOTRACHEAL INTUBATION. II. EFFECTS DURING GENERAL ANESTHESIA AND INTRAVENOUS PROCAINE *

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A PRECEDING investigation indicated that electrocardiographic disturbances were manifested during endotracheal intubation in 68 per cent of the patients who had been anesthetized with various commonly used agents and technics. The majority of these disturbances could be classified as resulting from stimulation of the cardio-accelerator nerve characterized by sinus tachycardia, premature ventricular contractions and ventricular tachycardia. A smaller percentage showed disturbances of cardiac depression as evidenced by sinus bradycardia and nodal rhythm. Factors which seemed to enhance cardiac disturbances during endotracheal intubation were occasioned by insufficient depth of anesthesia, prolonged laryngoscopy with repeated attempts at intubation, respiratory obstruction before intubation and tracheal irritation after intubation (1).

Since intravenous procaine has been shown to be beneficial in the treatment of certain cardiac arrhythmias produced during general anesthesia (2, 3, 4, 5, 6), it was deemed advisable to determine whether the administration of procaine intravenously during anesthesia prior to intubation of the trachea could minimize the electrocardiographic disturbances caused by intubation. In the report herein presented, an analysis is made of the effects on the electrocardiograms in a series of 114 adult patients who were intubated during anesthesia with various anesthetic agents and in whom procaine hydrochloride—in a 1 per cent concentration—was injected intravenously one to five minutes before endotracheal intubation.

METHOD

As in the preceding series, control electrocardiograms of the first three leads were obtained in all patients before anesthesia. Subsequent lead 2 electrocardiographic tracings were taken during anes-

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thesia, during the injection of the procaine solution, one minute after the injection, during intubation, and after intubation at frequent intervals. Tracings were completed on 114 patients who underwent surgical interventions in which endotracheal intubation was an elective procedure. An attempt was made to approximate the number of different anesthetic agents employed with that of the preceding series (table 1).

RESULTS

1. *Cyclopropane and Ether.*—The largest group in this series again comprised endotracheal intubation after anesthetization with cyclopropane induction followed by ether. There were 35 cases. Of these, 26 patients were intubated at a depth of second plane and 9 were intubated in third plane. In each instance, procaine hydrochloride, 100 mg. in a 1 per cent concentration, was injected intravenously one to five minutes prior to intubation.

TABLE 1

	Cases
Cyclopropane and ether	35
Cyclopropane, ether and cocaine	20
Cyclopropane alone	17
Cyclopropane and cocaine	12
Nitrous oxide and ether	6
Nitrous oxide, ether and cocaine	5
Pentothal sodium intravenously	4
Pentothal sodium and curare	2
Pentothal sodium, curare and cocaine	2
Pentothal sodium, cyclopropane and ether	2
Pentothal sodium, cocaine and ether	3
Pentothal sodium, cocaine and cyclopropane	1
Nembutal and curare	3
Nembutal, curare and cocaine	2
Total	114

Of the 26 patients who were intubated during cyclopropane-ether anesthesia in the second plane, 11 showed no change at any time in the electrocardiograms. This is a significant improvement over the preceding series in which only 3 of 24 patients showed no electrocardiographic changes when the only difference was omission of the intravenous procaine. In addition to the 11 uneventful cases, 2 patients developed a transitory nodal rhythm immediately after the injection of procaine. This returned to normal in one to two minutes and remained normal during and after intubation.

Sinus tachycardia was produced at the time of intubation in 5 instances—this is in contrast to an incidence of 11 in the series in which procaine had been omitted. The tachycardia ranged from 100 to 125 per minute and lasted an average of four minutes.

In 5 of the patients, there was no electrocardiographic change before or during intubation, but following intubation the patients manifested a "bucking" reaction and in 3 of the cases sinus tachycardia de-

veloped while the other 2 developed ventricular premature contractions with bigeminal rhythm.

Two patients had some cardiac disease in the preanesthetic state as evidenced by the control electrocardiograms. In one of these, there was sinus tachycardia with auricular premature contractions; in the other, there were prominent S2 and S3 waves indicative of left ventricular preponderance. In both of these patients a first degree heart block developed following the intravenous administration of the procaine, characterized by prolongation of the PR interval which was sustained for ten to fifteen minutes (fig. 1).

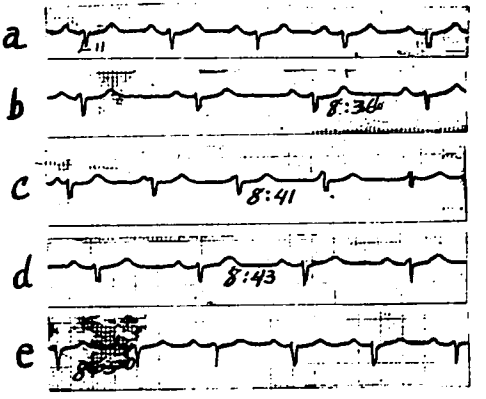

FIG. 1. Cardiac depression characterized by nodal rhythm and first degree heart block following intravenous procaine during cyclopropane-ether anesthesia. *a*—Control lead 2: prominent S wave; left ventricular preponderance. *b*—First plane cyclopropane: sinus bradycardia, rate 55. *c*—One minute after procaine: shifting pacemaker with nodal rhythm. *d*—Three minutes after procaine: regular sinus rhythm, rate 65; PR interval 0.24 seconds (first degree heart block). *e*—Five minutes after intubation: regular sinus rhythm, rate 90; PR interval 0.22 seconds.

Only one patient in this group developed ventricular premature contractions with bigeminal rhythm at the moment of intubation. This was a difficult case that required several attempts before intubation could be accomplished. A regular sinus rhythm returned ninety seconds later and was maintained thereafter.

When endotracheal intubation was performed following cyclopropane induction supplemented by ether until the third plane was reached and procaine was injected prior to intubation, there were no electrocardiographic changes in 12 of 13 cases. Three of these, although they manifested no electrocardiographic disturbance at the time of intubation, "bucked" considerably following the insertion of the endotracheal

tube and in each instance a sinus tachycardia developed one to two minutes following intubation. In one of these, a tachycardia of 125 per minute lasted for two minutes; in another, a tachycardia of 145 per minute lasted for two minutes, and in the third, a tachycardia of 130 per minute lasted for thirty seconds (fig. 2). These three instances of tachycardia, which did not occur at the moment of intubation but followed a "bucking" reaction, presumably can be attributed to temporary respiratory asphyxia.

Only one patient in this group of 13 showed an electrocardiographic change at the time of intubation; this consisted of a temporary rise in heart rate from 100 to 130 per minute which lasted thirty seconds.

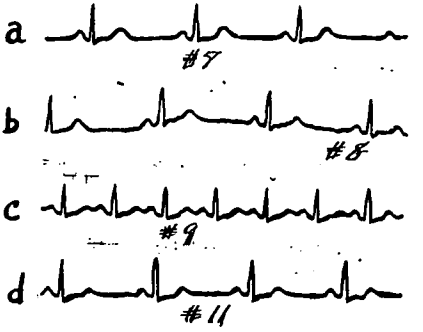

FIG. 2. Tachycardia produced by a "bucking" reaction following endotracheal intubation during third plane cyclopropane-ether. *a*—Time of endotracheal intubation: regular sinus rhythm, rate 72. *b*—During "bucking" reaction following intubation: regular sinus rhythm, rate 60. *c*—One minute later: sinus tachycardia, rate 130. *d*—Four minutes after intubation: regular sinus rhythm, rate 75.

2. *Cyclopropane, Ether and Cocainization.*—Endotracheal intubation performed during cyclopropane-ether anesthesia and intravenous procaine combined with 10 per cent cocaine spray of the glottis was studied in a group of 20 cases. Twelve patients were intubated during second plane and 8 during third plane anesthesia. The results showed somewhat fewer disturbances compared to the previous series when intravenous procaine was not administered. But, again, it would seem that the greater incidence of results of cardio-accelerator nerve stimulation may be the result of the absorption of cocaine following its topical application.

Six of the 12 patients who were intubated during second plane under these conditions showed no electrocardiographic disturbance at the time of intubation.

One of the patients in this group developed ventricular premature

contractions and bigeminal rhythm five minutes after the injection of 100 mg. of procaine during second plane cyclopropane-ether anesthesia. This arrhythmia persisted for twelve minutes. A second dose of 100 mg. of procaine was injected at this time. One minute later a regular sinus rhythm returned and was sustained. Laryngoscopy, cocaine spray of the glottis and intubation performed nine minutes later caused no further disturbance (fig. 3).

Three other patients whose electrocardiograms showed no disturbance during intubation reacted by "bucking" immediately following intubation, and one minute after intubation a sinus tachycardia developed in all 3, which lasted three to five minutes. In one case, a sinus

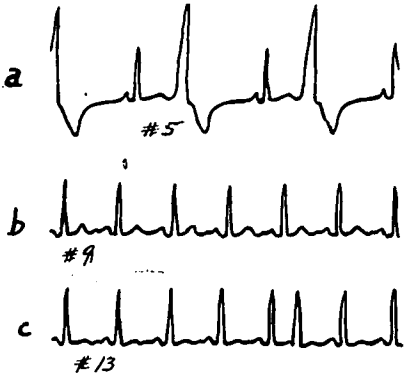

FIG. 3. Ventricular premature contractions and bigeminal rhythm during cyclopropane-ether anesthesia sustained until after the intravenous injection of a second dose of 100 mg. of procaine. *a*—Ventricular premature contractions and bigeminal rhythm during cyclopropane-ether anesthesia persisting five minutes after the intravenous injection of a first dose of procaine. *b*—Regular sinus rhythm, rate 110, three minutes after the intravenous injection of a second dose of 100 mg. of procaine. *c*—At the time of endotracheal intubation: regular sinus rhythm, rate 95.

tachycardia of 120 per minute lasted for three minutes. In the second, a tachycardia of 115 per minute was produced one minute after intubation and this was increased to 140 per minute when inflation of the cuff on the endotracheal tube had resulted in severe "bucking." In the third case, a sinus tachycardia of 125 per minute persisted for three minutes.

Sinus tachycardia at rates of 120 to 140 per minute developed at the very instant of intubation in 4 of the 12 patients in this series.

Ventricular premature contractions with bigeminal rhythm which persisted for eleven minutes were manifested by another patient at intubation. A second dose of 100 mg. of procaine was administered

intravenously and two minutes later the electrocardiogram returned to normal, with maintenance of normal rhythm.

Electrocardiographic tracings were obtained in 8 patients who were intubated following cyclopropane-ether anesthesia in the third plane complemented by 100 mg. of procaine intravenously and also cocainization of the glottis. Six of these 8 patients had no electrocardiographic change at the time of intubation. Four of these, however, developed sinus tachycardia one minute after intubation at rates of 120 to 130 per minute, lasting three to four minutes.

The seventh patient manifested ventricular premature contractions and bigeminal rhythm following spraying of the glottis with 10 per cent cocaine. This type of arrhythmia persisted until one minute after the intravenous injection of 100 mg. of procaine, when the rhythm returned to normal. Intubation then showed no electrocardiographic disturbance.

The eighth patient in this group developed first degree heart block at the time of a first attempt at intubation. A second attempt, three minutes later, produced ventricular premature contractions and bigeminal rhythm. On the third attempt, ten minutes later, intubation was completed and this coincided with a return to a normal sinus rhythm at a rate of 95 per minute. There was a moderate amount of "bucking" which resulted in an increase in heart rate to 110 per minute for three minutes.

3. *Cyclopropane.*—Electrocardiographic tracings were obtained in 17 patients who were intubated following anesthetization with cyclopropane and the intravenous injection of 100 mg. of procaine. Thirteen were intubated in second plane and 4 in third plane.

Eight of the 13 cases in which intubation was performed during second plane anesthesia showed no electrocardiographic change. This is a significant improvement over the previous series in which intravenous procaine had been omitted, for then only one of the 9 patients intubated during second plane cyclopropane anesthesia manifested no change.

Two other patients showed no change at intubation but electrocardiographic disturbances occurred following a "bucking" reaction caused by the inserted endotracheal airway. In one patient, a sinus tachycardia at a rate of 110 per minute developed one minute after intubation and three minutes later when the cuff was inflated the rate increased to 140 per minute. In the other patient, violent "bucking" was also produced five minutes after intubation, and this was followed by ventricular premature contractions with trigeminal rhythm (fig. 4).

Sinus tachycardia at a rate of 130 per minute occurred in 2 of the cases just as the endotracheal tube was passed through the larynx.

One patient developed ventricular premature contractions with bigeminal rhythm during second plane cyclopropane anesthesia. It was

not altered by the intravenous injection of 100 mg. of procaine, and it persisted during and after endotracheal intubation. A second injection of 100 mg. of procaine was followed one minute later by a return to regular sinus rhythm.

When endotracheal intubation was performed during third plane cyclopropane anesthesia and intravenous procaine, 3 of 4 patients showed no electrocardiographic change. The one who sustained a cardiac arrhythmia developed ventricular premature contractions with bigeminal rhythm which were produced at the time of intubation, and this type of arrhythmia persisted for two minutes.

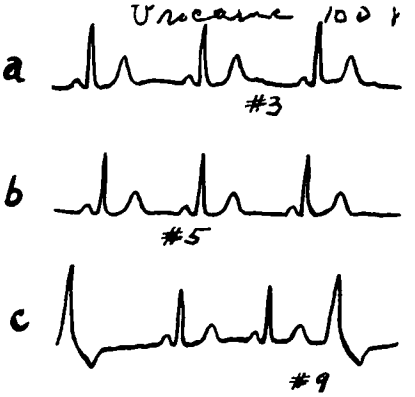

FIG. 4. Ventricular premature contractions and trigeminal rhythm produced by "bucking" reaction five minutes after endotracheal intubation during second plane cyclopropane anesthesia. *a*—Regular sinus rhythm at time of injection of 100 mg. of procaine during second plane cyclopropane anesthesia. *b*—Regular sinus rhythm persists during endotracheal intubation. *c*—Ventricular premature contractions and trigeminal rhythm five minutes after intubation following a "bucking" reaction.

4. *Cyclopropane and Cocaine.*—In 12 cases, endotracheal intubation was performed during cyclopropane anesthesia, intravenous procaine, and following the spraying of the glottis with 10 per cent cocaine. Nine were intubated in second plane and 3 in the third plane.

Nine of these 12 cases showed no electrocardiographic change at intubation. This is in sharp contrast to the previous series in which procaine intravenously had been omitted and in which only one of 9 patients had no electrocardiographic disturbance at the time of intubation.

Four of the 9 patients intubated under these conditions in the second plane had no electrocardiographic disturbance at any time prior to, during, or after intubation.

Two other patients showed no disturbance at the time of intubation although they had developed arrhythmias immediately following cocainization of the larynx during cyclopropane anesthesia. In one patient the arrhythmia consisted of ventricular premature contractions and bigeminal rhythm. In the other, there were ventricular premature contractions with short runs of ventricular tachycardia. In both of these cases, the arrhythmias returned to a regular sinus rhythm about one minute after the intravenous injection of 100 mg. of procaine. Endotracheal intubation, as already mentioned, then showed no disturbance (fig. 5).

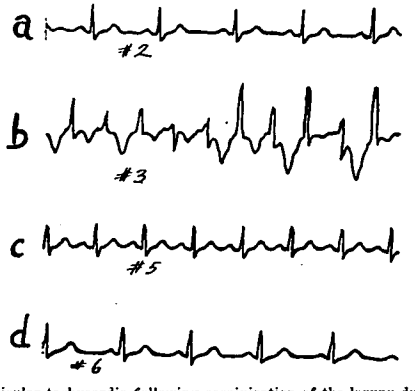

FIG. 5. Ventricular tachycardia following cocainization of the larynx during cyclopropane anesthesia and return to normal rhythm one minute after the intravenous injection of procaine. *a*—First plane cyclopropane anesthesia: regular sinus rhythm. *b*—Seven minutes after start of cocainization of the larynx: ventricular premature contractions and short runs of ventricular tachycardia. *c*—One minute after the intravenous injection of 100 mg. procaine: sinus tachycardia, rate 120. *d*—During endotracheal intubation, five minutes after the procaine: regular sinus rhythm, rate 75.

In 2 of the cases, a nodal rhythm occurred about one minute after the intravenous injection of procaine. This was transitory, lasting fifty seconds in one case and two minutes in the other. The regular sinus rhythms were then maintained in both cases during and after intubation.

The last of the 9 cases intubated during these conditions developed a sinus tachycardia from 65 to 115 per minute after cocainization. The injection of 100 mg. of procaine was then followed by a return in the heart rate to 80 per minute. When the endotracheal tube was inserted a sinus tachycardia at 140 per minute was observed.

None of the 3 patients intubated during third plane cyclopropane anesthesia in which the glottis had been sprayed with 10 per cent co-

caine and 100 mg. of procaine injected intravenously showed any electrocardiographic change at any time.

5. *Nitrous Oxide and Ether.*—Electrocardiographic tracings were obtained in 6 patients who were intubated after nitrous oxide induction followed by ether and oxygen and in whom procaine was injected intravenously prior to intubation.

Three of these patients were intubated during second plane. Of these, 2 showed no change at intubation, the other manifested an increase in heart rate to 110 per minute which lasted five minutes.

None of the 3 patients intubated in third plane had any electrocardiographic disturbance.

6. *Nitrous Oxide, Ether and Cocaine.*—The glottis was sprayed with 10 per cent cocaine in 5 patients who had been anesthetized with nitrous oxide and ether. This was followed by the injection of 100 mg. of procaine intravenously prior to intubation.

Three of these patients were intubated at a depth of second plane and none showed any electrocardiographic change.

Both of the patients who were intubated at a depth of third plane showed no change at the time of intubation. However, one of them "bucked" violently when the tube was introduced into the trachea and this was followed one minute later by a rise in heart rate from 90 to 130 per minute which lasted for five minutes. This case illustrates that the laryngeal and tracheal reflexes are not obtunded at the same depth in all patients. The tachycardia produced in this case may be attributed to the temporary asphyxia following intubation; the "bucking" reaction was so severe that apparent bronchospasm prevented respiratory tidal exchange.

7. *Pentothal Sodium.*—Three of 4 patients who were intubated following the intravenous injection of pentothal sodium (750 mg.) supplemented by the intravenous injection of 100 mg. of procaine showed no electrocardiographic disturbance at the time of intubation. The fourth patient developed a tachycardia of 115 per minute at the time of intubation which was sustained for two minutes.

Two of these 4 patients manifested a significant decrease in the voltage of the T wave two minutes after the injection of pentothal, which lasted ten to fifteen minutes (fig. 6). A similar observation had been noted in the previous series when endotracheal intubation was performed following pentothal sodium alone.

8. *Pentothal Sodium and Curare.*—Two patients were intubated after the intravenous injection of a mixture of pentothal sodium (500 mg. in 20 cc.) and d-tubocurarine (50 units in 2.5 cc.) followed one minute later by the intravenous injection of procaine (100 mg. in 10 cc.). There were no circulatory changes that could be depicted in these 2 cases before, during or after endotracheal intubation.

9. *Pentothal Sodium, Curare and Cocaine.*—Electrocardiographic tracings were obtained in 2 patients who were anesthetized by first

spraying the pharynx and glottis with 10 per cent cocaine. This was followed by the intravenous injection of a mixture of pentothal sodium with d-tubocurarine as in the preceding group. One minute later, procaine (100 mg. in 10 cc.) was injected intravenously and endotracheal intubation was performed one minute after the last injection.

In one patient, intubation produced a rise in heart rate from 100 to 125 per minute which persisted for four minutes.

The second patient was apprehensive despite what had been deemed adequate preanesthetic medication. His heart rate was 130 per minute. Five minutes after cocainization, the tachycardia had increased to 165 per minute. This tachycardia was not abated following the injection of pentothal-curare nor following the procaine injection. Endotracheal intubation was uncomplicated but the sinus tachycardia persisted until six minutes after intubation.

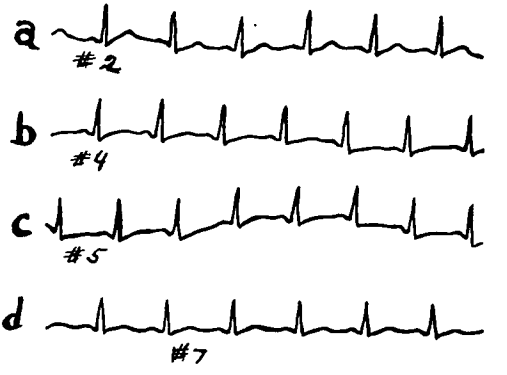

FIG. 6. Decrease in voltage of T wave following the intravenous injection of pentothal sodium. *a*—Just prior to intravenous pentothal: regular sinus rhythm, rate 95. *b*—Two minutes after pentothal: decrease in voltage of T wave; regular sinus rhythm, rate 105. *c*—During intubation: same as *b*. *d*—Fifteen minutes after pentothal: increase in voltage of T wave; regular sinus rhythm, rate 95.

10. *Pentothal Sodium, Cyclopropane and Ether.*—Electrocardiograms were secured in 2 patients who were intubated following the intravenous injection of pentothal sodium (400 mg. in 20 cc.) succeeded by cyclopropane and ether anesthesia to mid-second plane. There were no circulatory changes in one patient. The second patient developed ventricular premature contractions with bigeminal rhythm at the time of intubation which persisted for three minutes.

11. *Pentothal Sodium, Cocaine and Ether.*—Three patients were intubated after being anesthetized in the following sequence: intravenous injection of pentothal sodium (400 mg. in 20 cc.), cocainization

of the pharynx and glottis with 10 per cent cocaine, ether by inhalation to mid-second plane, and the intravenous injection of procaine (100 mg. in 10 cc.).

One patient showed no electrocardiographic change. After the injection of procaine a second patient developed a nodal rhythm which lasted for two minutes at which time the patient was intubated and the tracing reverted to a regular sinus rhythm which was maintained.

The third patient showed no change until five minutes after intubation when the heart rate increased from 100 to 145 per minute; this tachycardia lasted for eight minutes.

12. *Pentothal Sodium, Cocaine and Cyclopropane.*—Electrocardiographic tracings were obtained in one patient who was intubated after anesthetization as in the preceding group but cyclopropane was substituted for ether. The electrocardiogram remained normal throughout.

13. *Nembutal and Curare.*—Three patients were intubated after anesthesia was induced in the following manner: nembutal (500 mg. in 20 cc.) was first injected intravenously. This was followed by d-tubocurarine (70 units in 3.5 cc.). One minute later, procaine (100 mg. in 10 cc.) was injected through the same intravenous route. Endotracheal intubation was accomplished one minute later.

One subject maintained a normal electrocardiogram.

At the time of intubation a second patient developed a sinus tachycardia from 80 to 120 per minute which lasted sixty seconds.

The third patient manifested a lowered T wave for four minutes.

14. *Nembutal, Curare and Cocaine.*—In 2 patients, the same technic as in the preceding group was carried out and in addition cocaine spray of the glottis was performed.

One patient showed no electrocardiographic disturbance.

Immediately after the injection of nembutal the other patient developed a depressed ST segment with diphasic T wave which lasted for four minutes. At intubation, two ventricular premature contractions were recorded and then the tracing returned to normal.

DISCUSSION

Under the conditions herein described, the majority of the electrocardiographic changes produced by endotracheal intubation during various types of general anesthesia consisted of changes which may be attributed to stimulation of the cardio-accelerator nerve. Of 114 cases studied in this series, 27 (24 per cent) showed some electrocardiographic disturbances at the time of intubation which consisted of sinus tachycardia, 18 cases; ventricular premature contractions with bigeminal rhythm, 4 cases; increase in the PR interval, 2 cases; and one case each of nodal rhythm; first degree heart block, and isolated premature ventricular contractions.

The incidence of 24 per cent, in this series, manifesting electrocardiographic changes at the time of endotracheal intubation compares

favorably with a preceding series in which the main disparity had been omission of intravenous procaine prior to intubation (1). Then, the incidence of electrocardiographic changes produced at the time of intubation of the trachea was 68 per cent. The addition of intravenous procaine not only reduced the total number of cardiac disturbances but also seemed to minimize the severity of such disturbances. Thus, whereas sinus tachycardia was produced in 43 cases of the series without procaine, there were only 18 cases of sinus tachycardia when procaine was administered intravenously prior to intubation. Furthermore, whereas the average heart rate of the sinus tachycardia was 140 per minute when procaine was omitted, it was 120 per minute when procaine was used. Similarly, in the present series, not only were there 4 cases instead of 10 in which ventricular premature contractions with bigeminal rhythm developed at the time of intubation, but the severity and duration of this type of arrhythmia were markedly reduced.

Depth of anesthesia at the time of intubation was found to be a similarly prominent factor in both series. Of 95 patients anesthetized with inhalation anesthesia, 66 were intubated during second plane and electrocardiographic changes were observed in 16 (24 per cent). When intubation was performed in third plane, 4 of 29 patients showed some cardiac disturbance (14 per cent).

The addition of cocaine by spraying the glottis and pharynx with 10 per cent cocaine solution during general anesthesia again demonstrated an increased number of electrocardiographic changes characterized by sinus tachycardia, ventricular premature contractions, or ventricular tachycardia. These cardiac arrhythmias were observed to occur from one to three minutes after cocainization. Intravenous administration of procaine tended to correct these arrhythmias and endotracheal intubation could then be performed, in most instances, without producing any further electrocardiographic disturbance (fig. 5).

The barbituric acid derivatives, pentothal sodium and nembutal, were used relatively infrequently in this series. Endotracheal intubation following the administration of these agents was attended with fewer disturbances in rhythm and in rate. As in the previous series, however, there were some indications that these barbiturates may affect the cardiac myocardium as manifested by the changes in the T waves and in the ST segment. Of 19 patients anesthetized with pentothal sodium or nembutal, 4 developed such abnormalities; significant depression of the T wave occurred in 3 patients, and another showed a depressed ST segment and diphasic T wave.

As in the previous series, it was noted that prolonged laryngeal exploration and excessive instrumentation with repeated attempts at tracheal intubation enhanced and aggravated the electrocardiographic disturbances.

Transitory respiratory obstruction due to "bucking" following the

insertion of a tube into the trachea frequently caused electrocardiographic disturbances. It was observed in 19 cases in this series. Circulatory changes appeared about one minute after the insertion of the endotracheal airway and lasted three to five minutes. In 16 cases, a sinus tachycardia of 125 to 140 per minute was produced. In 2 cases ventricular premature contractions with bigeminal rhythm developed and in one case the ventricular premature contractions were in trigeminal form.

Another observation was that the intravenous injection of 100 mg. of procaine during general anesthesia was followed in 6 instances by a nodal rhythm which lasted one to two minutes. This transitory effect may be attributed to the effect of procaine in depressing cardiac conduction. It is of no grave consequence. On the contrary, it corroborates the thesis that intravenous procaine during general anesthesia tends to diminish the increased sensitization engendered by the state of general anesthesia.

The recommended dose of 100 mg. of procaine in a 1 per cent concentration is an arbitrary one. Occasionally, as occurred in 3 cases in this series, this dose may be insufficient to effect any change in a cardiac disturbance but a second identical dose, injected five to ten minutes later, may produce the desired result.

SUMMARY

Electrocardiographic tracings were obtained in 114 adult patients who were given general anesthesia complemented by the intravenous injection of 100 mg. of procaine hydrochloride in a 1 per cent concentration prior to endotracheal intubation. Electrocardiographic disturbances occurred in 24 per cent of the cases at the time of intubation. In a previous series under similar conditions but in which procaine had been omitted, the incidence of electrocardiographic disturbances was 68 per cent.

The changes in the electrocardiograms produced at the moment of endotracheal intubation were similar to those in the previous series in which procaine was not administered, although they were less severe as well as less frequent. The majority of the disturbances (18 out of 27) again consisted of transitory sinus tachycardia. Other changes included ventricular premature contractions in bigeminous form, increase in the PR interval, nodal rhythm and first degree heart block. As in the previous series, insufficient depth of anesthesia, prolonged laryngoscopy and respiratory obstruction were important factors in the development of such electrocardiographic disturbances.

Topical cocainization of the pharynx and larynx during general anesthesia seemed to enhance cardiac disorders which were manifested by sinus tachycardia, ventricular premature contractions and ventricular tachycardia. Presumably, these effects may be attributed to sym-

pathetic stimulation from the systemic absorption of cocaine with potentiation of these effects during the state of general anesthesia.

In cases in which there was "bucking" following the insertion of an endotracheal airway, electrocardiographic changes were observed to occur one minute after the start of the "bucking" reaction. The disturbances consisted of sinus tachycardia or ventricular premature contractions.

All the cardiac disturbances noted before, during or after endotracheal intubation were transitory and of no grave consequence. It seems worthy, however, to make every effort to minimize such disturbances. The intravenous injection of procaine during general anesthesia before endotracheal intubation was found to be a worthwhile procedure.

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Seldon—"Blood and Plasma Substitutes"

Slocum—"Influence of Position on Vital Capacity"

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Thomas—"Explosion Hazards and Their Control"

Barbour—"Functions and Management of Inhalation Therapy Service"

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Tuohy—"Continuous Spinal Anesthesia"

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