

ELECTROCARDIOGRAPHIC STUDIES DURING ENDO- TRACHEAL INTUBATION.* I. EFFECTS DURING USUAL ROUTINE TECHNICS

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WITH the advent of instantaneous electrocardiography, a new important method of cardiac evaluation is available to the clinical anesthesiologist. Electrocardiographic studies during endotracheal intubation comprise one phase of interest. To clarify the observations in these studies, it is intended to present several papers in this series. Our first paper represents the observations obtained in over one hundred cases with varying agents and technics. The majority consists of intubations performed during anesthesia with cyclopropane alone or combined with ether. Other agents were used in smaller numbers, but the results are included in order to obtain a general glimpse of what may occur. We intend to study various phases independently and more thoroughly. It is hoped that other anesthesiology centers will take up this mode of investigation to further increase our knowledge.

In a previous study on cardiocirculatory disturbances during intrathoracic surgery it was observed that 24 of 33 patients showed electrocardiographic changes when an endotracheal tube was passed through the larynx (1). Because of the surprisingly high incidence of cardiac disturbances it was deemed advisable to study electrocardiographic effects during endotracheal intubation in a larger series. An analysis of the electrocardiographic effects during intubation with various anesthetic agents in 109 cases is made in the present report. The effects of intubation in the different planes of surgical anesthesia as well as the effects of certain attending technical complications such as respiratory obstruction or trauma are also correlated. In a subsequent report, the value of intravenous procaine prior to endotracheal intubation will be presented.

METHOD

Control electrocardiograms were obtained in all patients prior to anesthetization. Subsequent lead 2 electrocardiographic tracings were

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TABLE 1

Cyclopropane and ether	37
Cyclopropane, ether and cocaine	15
Cyclopropane alone	15
Cyclopropane and cocaine	8
Nitrous oxide and ether	7
Nitrous oxide, ether and cocaine	3
Pentothal sodium	5
Pentothal and cocaine	4
Pentothal, curare and cocaine	2
Pentothal, cocaine and ether	2
Nembutal and curare	3
Nembutal, curare and cocaine	2
Cocaine topically in conscious state	6
Total	109

taken during anesthesia prior to, during, and after intubation at frequent intervals. Electrocardiographic tracings were also taken if and when the patients manifested an undue complication such as laryngeal spasm before intubation, excessive mucous secretions, or "bucking" reaction on the inserted endotracheal tube. Tracings were completed on 109 adult patients who underwent surgical interventions in which endotracheal intubation was an elective technic. The anesthetic agents employed prior to endotracheal intubation are listed in table 1.

RESULTS

1. *Cyclopropane and Ether*.—Endotracheal intubation after anesthesia induced with cyclopropane followed by ether comprised the largest group. This consisted of 37 cases. Of these, 24 were intubated at a depth of second plane anesthesia and 13 were intubated when third plane was attained.

Of the 24 patients intubated during cyclopropane and ether anesthesia in the second plane, 21 showed some cardiac disturbance at the time the tube was passed into the trachea. The majority (11 instances)

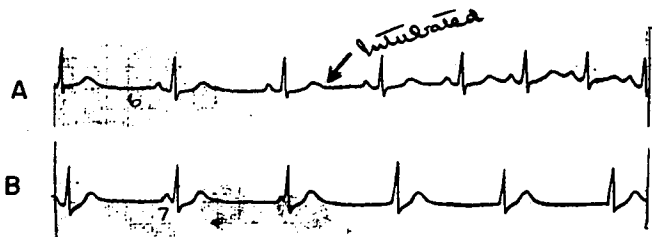


FIG. 1. Sinus tachycardia and nodal rhythm following endotracheal intubation. The upper tracing (A) shows the transition from a heart rate of 50 per minute to a sudden sinus tachycardia of 110 per minute at the instant an endotracheal tube was inserted into a patient anesthetized with cyclopropane and ether in second plane. The lower tracing (B), taken ten minutes later, shows a transition to nodal rhythm.

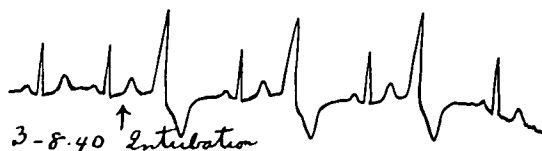


Fig. 2. Premature ventricular contractions with runs of bigeminal rhythm occur immediately after endotracheal intubation during cyclopropane-ether anesthesia.

showed the development of a sinus tachycardia at a rate of from 110 to 160 per minute which was observed at the very instant the endotracheal tube was passed. Figure 1 illustrates such an effect. The upper tracing shows the transition from a previous rate of 50 per minute to one of 110 per minute at the time of intubation. The lower tracing, taken three minutes later, shows the development of a nodal rhythm. Similar nodal rhythms were observed in 4 other cases in this series, all 5 of which had developed in three to five minutes after intubation and all 5 patients showed immediate but temporary sinus tachycardia at the time of intubation.

Three of the 21 patients who showed some cardiac disturbance during intubation in this series manifested premature ventricular contractions with runs of either bigeminal or trigeminal rhythm. Figure 2 shows one instance in which premature ventricular contractions with runs of bigeminal rhythm occurred immediately following intubation.

Other electrocardiographic abnormalities produced during intubation in this series consisted of: decrease in the voltage of the T waves,

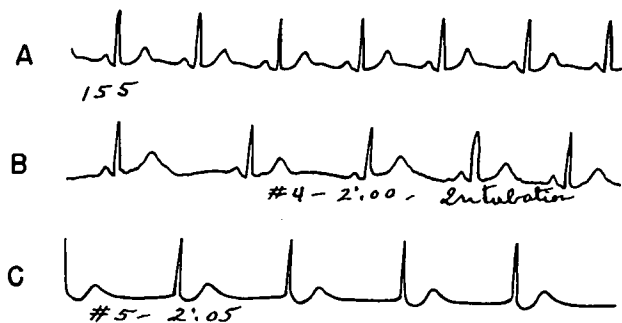


Fig. 3. Sinus bradycardia and nodal rhythm following endotracheal intubation. The upper tracing (A) shows a regular sinus rhythm at a rate of 85 per minute in a patient under cyclopropane-ether anesthesia in second plane before intubation. In the middle tracing (B), taken immediately after intubation, the rate was diminished from 85 to 60 per minute. The lower tracing (C), taken five minutes after intubation, shows a nodal rhythm.

2 cases; sinus bradycardia, 2 cases (in one, the previous heart rate of 85 per minute was diminished to 60) (fig. 3); in the second case the heart rate diminished from 140 to 65 per minute); increase in the PR interval from 0.16 to 0.28, one case; depression and sagging of the ST segment, one case, and nodal rhythm, one case. (Nodal rhythm, as previously mentioned, occurred five minutes after intubation in 4 of the cases which had manifested a sinus tachycardia at the time of intubation.)

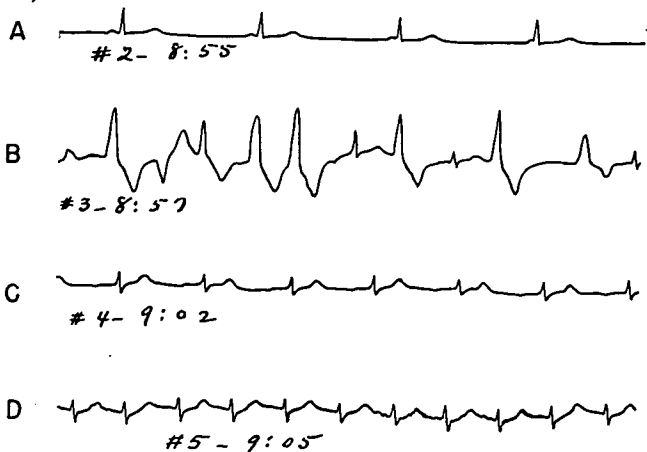


FIG. 4. Ventricular tachycardia following endotracheal intubation after cyclopropane-ether anesthesia and cocaineization of the larynx. Tracing (A), prior to intubation, shows a nodal rhythm with ventricular rate of 48 followed by a return to regular sinus rhythm with a rate of 60 per minute. Tracing (B), immediately following intubation, shows the appearance of frequent multifocal ventricular premature contractions with runs of ventricular tachycardia, separated by occasional nodal beats and followed by a definite nodal rhythm at a rate of 95 per minute. Tracing (C) taken five minutes after intubation, shows persistence of nodal rhythm at a rate of 75 per minute. Tracing (D), taken ten minutes after intubation, shows persistence of the nodal rhythm at a rate of 130 per minute.

Only 3 of the 24 patients who were intubated during second plane cyclopropane-ether anesthesia showed no electrocardiographic changes.

A marked difference in reaction was observed when intubation was performed during third plane cyclopropane-ether anesthesia in contrast to intubation in a lighter plane. Of 13 cases so studied, 7 showed no change in the electrocardiogram. In another case, a nodal rhythm which was present before intubation, changed to a normal sinus rhythm at the time the endotracheal tube was passed. In 4 of 5 cases (out of 13) in which an electrocardiographic change was manifested, it consisted of a sinus tachycardia which was less pronounced than in the

series of intubation during second plane; the heart rates in this instance increased from 70 or 80 per minute to 110, 115, 120 and 135. The fifth abnormality was the production of several ventricular premature contractions which lasted less than one minute.

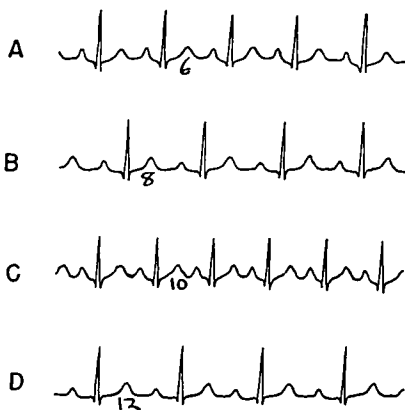


FIG. 5. Increase in PR interval following endotracheal intubation during cyclopropane anesthesia. Tracing (A), during second plane cyclopropane anesthesia, shows a regular sinus rhythm at a rate of 80 per minute and a PR interval of 0.18 second. Tracing (B), following an unsuccessful attempt at intubation, shows a prolongation of the PR interval from 0.18 to 0.22 second. In tracing (C), following a bout of coughing, the heart rate is increased from 80 to 120 per minute. Tracing (D), immediately following intubation, demonstrates that the PR interval has increased to 0.26 second; this persisted for seven minutes.

The important inference, therefore, is that when intubation is performed in the deeper plane of cyclopropane-ether anesthesia the incidence of electrocardiographic changes is reduced (from 87 per cent to 39 per cent).

2. *Cyclopropane, Ether and Cocainization.*—Endotracheal intubation performed during cyclopropane-ether anesthesia combined with 10 per cent cocaine spray of the glottis was studied as a separate series.

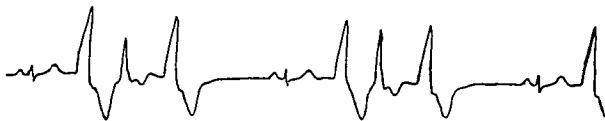


FIG. 6. Ventricular premature contractions and ventricular tachycardia which developed when a patient anesthetized and intubated during cyclopropane anesthesia "bucked" on the tube thirty seconds after intubation.

Electrocardiographic tracings were obtained in 15 cases. In 9, intubation was carried out during second plane and in 6 during third plane anesthesia.

Only 2 of the 9 patients intubated during second plane anesthesia showed no electrocardiographic changes. The disturbances produced in the other 7 patients consisted of:—sinus tachycardia (to 120, 140, 150 per minute), 3 cases; sinus arrhythmia, one case; nodal rhythm, one case, and ventricular premature contractions, 2 cases. In one case it was characterized by ventricular premature contractions with bigeminal rhythm; in the other there were ventricular premature contractions with runs of ventricular tachycardia, separated by occasional nodal beats (fig. 4).

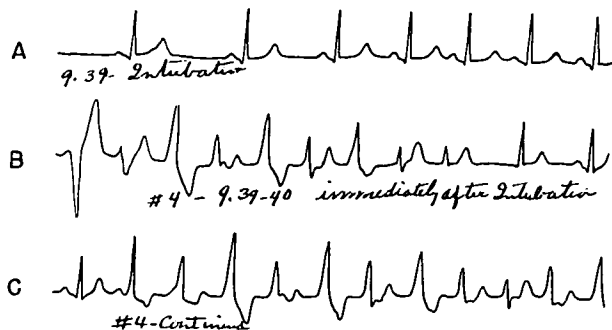


FIG. 7. Ventricular tachycardia following endotracheal intubation during cyclopropane anesthesia. Tracing (A), taken at the time of intubation, shows an increase in heart rate from 55 to 100 per minute. Tracings (B) and (C), continuous strips taken less than a minute after intubation, illustrate the appearance of frequent multifocal alternating ventricular extra systoles with a short run of ventricular tachycardia and brief return to regular sinus rhythm and then resumption of the ventricular tachycardia.

Of 6 patients intubated following cocainization and cyclopropane-ether anesthesia to third plane, 2 showed no electrocardiographic changes.

Sinus tachycardia was produced in 3 cases (with heart rates at 110, 130 and 140 per minute). In one this disturbance was complicated further by decreased voltage of the T waves and depression of the ST segment. The fourth case showed a ventricular tachycardia just before intubation which shifted to a sinus bradycardia at a rate of 45 per minute during the first attempt at intubation, and a persistence of the bradycardia at 60 per minute during intubation proper.

3. *Cyclopropane*.—Electrocardiographic tracings were obtained in 15 patients who were intubated following anesthetization with cyclo-

propane alone. Nine were intubated in second plane, and 6 in third plane.

Only one of 9 cases in which intubation was performed during second plane showed no electrocardiographic changes. The others showed the following changes at the time of intubation: Sinus tachycardia was produced in 4 cases (2 from 80 to 110, and 2 from 84 and 88 to 120 per minute. Nodal rhythm developed in 2 cases. In one patient prolongation of the PR interval from 0.18 to 0.22 developed when intubation was

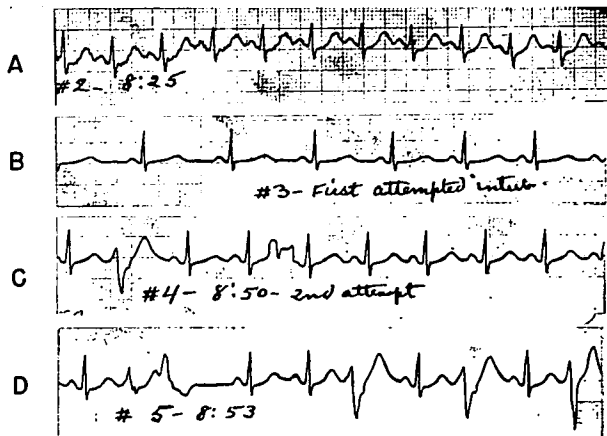


FIG. 8. Aggravation of electrocardiographic disturbances following repeated attempts at endotracheal intubation. Tracing (A), taken soon after a moderate excitement stage during cyclopropane anesthesia, shows a sinus tachycardia at a rate of 130 per minute. Tracing (B), taken during third plane cyclopropane anesthesia and during the first attempt at intubation, shows a regular sinus rhythm which was not altered by this first attempt. Tracing (C), taken during a second attempt at intubation, resulted in a sinus tachycardia at a rate of 105 per minute with some rare ventricular premature contractions. Tracing (D), taken during the third attempt at intubation, shows a sinus tachycardia at a rate of 110 per minute and the development of frequent ventricular premature contractions with runs of bigeminal rhythm.

attempted. This was followed by coughing which increased the heart rate from 80 to 120 per minute. At the time the endotracheal tube was passed, the PR interval increased further from 0.22 to 0.26 (fig. 5) and this was sustained for seven minutes. Another patient showed no change at the time of intubation but when he "bucked" on the tube a half minute later this was followed promptly by ventricular premature contractions and ventricular tachycardia (fig. 6).

When endotracheal intubation was performed during third plane

cyclopropane anesthesia, 3 of 6 patients showed no electrocardiographic change. Sinus tachycardia developed in one case. Nodal rhythm occurred in another. In the third case, intubation was associated with ventricular premature contractions and ventricular tachycardia (fig. 7).

4. *Cyclopropane and Cocaine*.—In 8 cases, endotracheal intubation was performed during cyclopropane anesthesia supplemented by 10 per cent cocaine spray of the glottis. Five were intubated in second plane and three in third plane anesthesia.

Only one patient showed no electrocardiographic change at the time of intubation which was during second plane anesthesia. The other 4 patients who were intubated in second plane anesthesia developed ventricular premature contractions, in three of which the contractions were

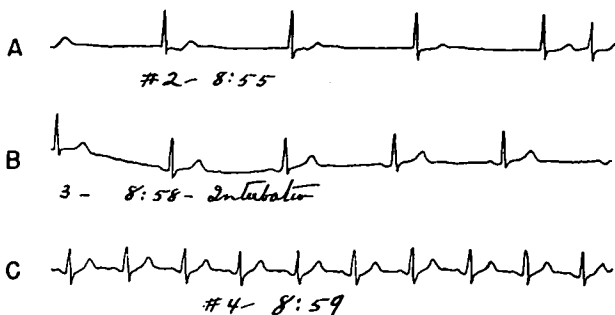


FIG. 9. Bradycardia followed by tachycardia after endotracheal intubation during ether anesthesia. Tracing (A), during second plane nitrous oxide-ether anesthesia, shows a nodal rhythm at a rate of 50 per minute with rare supraventricular premature contraction. Tracing (B), taken three minutes later during intubation, shows a sinus bradycardia at a rate of 55 per minute. Tracing (C), one minute after intubation, shows tachycardia at a rate of 110 per minute.

characterized by persistent bigeminal rhythm. In one of these, this rhythm occurred when intubation was attempted at three successive intervals and the ventricular premature complexes were more frequent in each succeeding attempt (fig. 8).

The 3 patients intubated during third plane cyclopropane anesthesia combined with cocaine topically developed sinus tachycardia (with heart rates at 120, 130 and 140 per minute) at the time of intubation.

5. *Nitrous Oxide and Ether*.—Electrocardiographic tracings were taken in 7 cases in which intubation was carried out after nitrous oxide induction followed by ether and oxygen in a closed rebreathing system.

In 4 of the cases, endotracheal intubation was performed during second plane anesthesia and all showed some electrocardiographic changes. Two had a sinus tachycardia at a rate of 130 per minute.

One developed a nodal rhythm. The fourth manifested a nodal rhythm prior to intubation; at the time of intubation, this changed to a sinus bradycardia at a rate of 55 per minute which lasted just one minute and then doubled its rate to 110 per minute (fig. 9).

Endotracheal intubation was performed in 3 cases during third plane nitrous oxide-ether anesthesia while electrocardiograms were taken. None of these displayed any electrocardiographic changes.

6. *Nitrous Oxide, Ether and Cocaine.*—Spraying of the glottis with 10 per cent cocaine was done in 3 patients who had been anesthetized with nitrous oxide-ether to third plane. Electrocardiographic records during endotracheal intubation showed no change in one case. The second patient developed a nodal rhythm which later changed to sinus

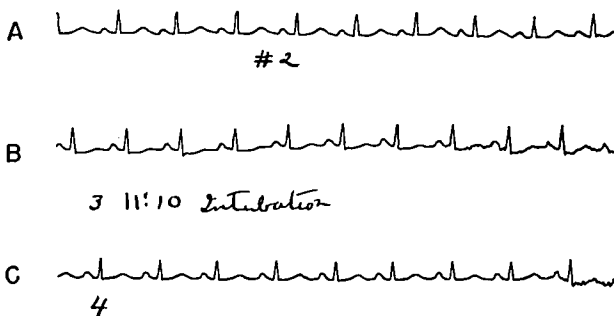


FIG. 10. Decrease in voltage of the T wave during endotracheal intubation after anesthesia with pentothal sodium. Tracing (A), after the administration of 1 Gm. of pentothal sodium intravenously into an adult male patient, a sinus tachycardia at a rate of 110 per minute is noted. Tracing (B), during endotracheal intubation, there is a decrease in the voltage of the T wave. Tracing (C), taken two minutes after intubation, shows a return of the T wave to its original height.

tachycardia at a rate of 120 per minute. The third case manifested a sinus tachycardia at a rate of 120 per minute which lasted less than a minute and then changed to a nodal rhythm.

7. *Pentothal Sodium.*—Four of 5 patients intubated following the intravenous administration of a relatively large dose of pentothal sodium (750 mg. to 1 Gm.) showed no electrocardiographic changes. The fifth case showed a depressed ST segment and decrease in the voltage of the T wave which lasted for two minutes (fig. 10).

8. *Pentothal Sodium and Cocaine.*—In 4 patients the pharynx and glottis were first sprayed with 10 per cent cocaine before pentothal sodium was injected intravenously. All 4 showed some electrocardiographic changes during intubation. Three of these displayed a sinus tachycardia at a rate of 120 per minute with depression of the ST seg-

ment and decrease in voltage of the T waves. The fourth case manifested bradycardia at the instant of intubation from 105 to 45 per minute; this was followed two minutes later by a sinus tachycardia at 120 per minute which persisted for two minutes (fig. 11).

9. *Pentothal Sodium, Curare and Cocaine.*—Electrocardiographic tracings were obtained in 2 cases in which intubation was carried out following the intravenous injection of a mixture of pentothal sodium (500 mg.) and curare (60 to 80 units) as well as the spraying of the glottis with 10 per cent cocaine. Both of these patients showed no electrocardiographic change at the time of endotracheal intubation.

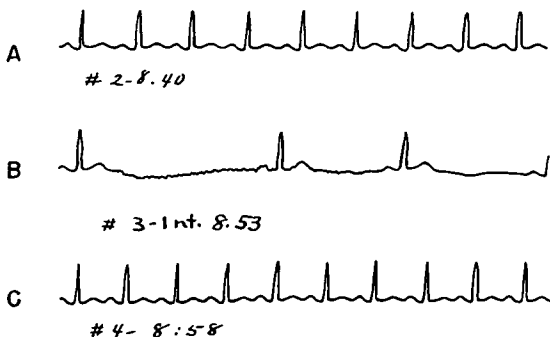


FIG. 11. Sinus bradycardia during endotracheal intubation. Tracing (A), taken following anesthesia by cocainization of the pharynx and larynx, then intravenous administration of 0.8 Gm. of pentothal sodium, shows a slight tachycardia at a rate of 105 per minute. Tracing (B), during intubation, shows a change to a sinus bradycardia at a rate of 45 per minute. Tracing (C), taken five minutes after intubation shows a return in the heart rate to 120 per minute.

10. *Pentothal Sodium, Cocaine and Ether.*—Two patients were anesthetized by first spraying the pharynx and glottis with 10 per cent cocaine and then injecting pentothal sodium intravenously (750 mg.) followed by inhalation of ether in a closed carbon dioxide absorption system until mid-second plane was attained. Electrocardiograms during intubation in one case showed a sinus tachycardia with a change in rate from 80 to 140 per minute. In the other case a sinus arrhythmia with decrease in the T voltage was observed.

11. *Nembutal-Curare.*—Electrocardiographic tracings were obtained in 3 cases intubated following the intravenous administration of pentobarbital sodium (500 mg. in 5 per cent solution) combined with 100 units of curare. Sinus tachycardia developed in all 3 cases: from 65 to 125 per minute in one case, from 100 to 150 per minute in the second case, and from 70 to 135 per minute in the third case. In the latter,

the change to sinus tachycardia lasted fifteen seconds and was succeeded later by frequent ventricular premature contractions and runs of trigeminal rhythm which persisted for ten minutes.

12. *Nembutal, Curare and Cocaine.*—In 2 cases, pentobarbital sodium and curare were used as in the preceding group and 10 per cent cocaine was sprayed into the glottis prior to endotracheal intubation. In both of these cases sinus tachycardia developed at the time of passing the tube into the trachea.

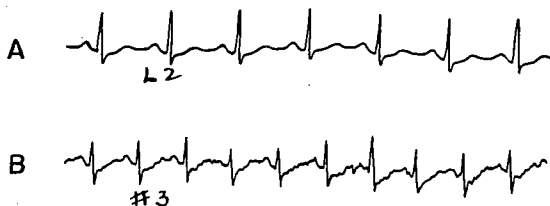


FIG. 12. Tachycardia during endotracheal intubation in the conscious state. Tracing (A), taken during intubation following cocainization of the pharynx and larynx, shows a regular sinus rhythm at a rate of 105 per minute. Tracing (B), taken during inflation of the cuff in the endotracheal tube caused a further increase in the heart rate to 130 per minute.

13. *Cocaine and Conscious State.*—Electrocardiographic tracings were taken in 6 patients who were intubated while awake but in whom the pharynx and larynx had been sprayed with 10 per cent cocaine solution. Three showed no electrocardiographic changes during such intubation. In the 3 who displayed some electrocardiographic disturbance, the first developed a sinus tachycardia at a rate of 140 per minute with considerable depression of the RST segment. In a second patient a sinus tachycardia from 76 to 105 per minute also developed and this increased to 130 per minute when the cuff on the endotracheal tube was inflated (fig. 12.) The third patient had paroxysmal tachycardia at a rate of 170 per minute and auricular fibrillation.

DISCUSSION

As Reid and Brace have shown in 10 cases, irritation of the respiratory tract by endotracheal intubation may be followed by a reflex effect upon the heart (2). That the electrocardiographic disturbances involved are of a reflex nature is substantiated by the fact that the changes occur at the very instant the tube is passed through the larynx into the trachea. The conclusion, however, that this reflex is a "reflex of a vagovagal type" cannot be fully corroborated. The nerve endings in the larynx and in the trachea are unquestionably the afferent portions of such a reflex. To explain the disparity of the efferent effects upon

the heart it may be noteworthy to recall that the vagus nerve trunk is composed of mixed fibers and that though the usual predominant efferent effect upon the heart is one of cardiac depression, it has been shown that the vagi also contain cardio-accelerator fibers which may become predominant particularly after sympathectomy (3, 4, 5). Furthermore, the vagal impulses may spread to the sympathetic nerves.

In the present series consisting of electrocardiographic determinations during endotracheal intubation in 109 cases there were electrocardiographic disturbances in 73 (68 per cent). In the majority, 43 cases, a sinus tachycardia developed with heart rates from 110 to 170 per minute at the time the endotracheal tube was introduced. The other electrocardiographic changes produced at the time of intubation consisted of: 10 cases of premature ventricular contractions with bigeminal or trigeminal rhythm; 5 cases of nodal rhythm; 4 cases of sinus bradycardia; 3 cases of decreased voltage of the T wave (in addition, there were 4 other cases which showed reduction of the T waves along with some other abnormalities); 3 cases of marked increase in the PR interval; 2 cases of sinus arrhythmia; 2 cases of ventricular tachycardia and 1 case of auricular fibrillation.

Depth of anesthesia at the time of intubation was a prominent factor. Of 84 patients anesthetized with inhalation anesthesia, 50 were intubated during second plane and electrocardiographic changes were observed in 43 (86 per cent). When intubation was performed during third plane, 19 of 34 patients showed some electrocardiographic disturbance (56 per cent).

Type of anesthesia may be a factor but was not too consistent in this small series. The addition of cocaine by spraying the glottis or the pharynx or both with 10 per cent cocaine solution seemed to increase the number of electrocardiographic changes—particularly favoring the production of sinus tachycardia. This might be attributed to a general sympathetic stimulating effect of cocaine. The patients who were intubated following the intravenous administration of pentothal sodium either alone or combined with laryngeal cocainization or intravenous curare or both seemed to show fewer electrocardiographic changes but there was an increase in the number showing myocardial or coronary disturbance or both as indicated by the development of decreased T waves and depressed ST segments.

Prolonged exploration and excessive instrumentation during laryngoscopy and necessity of repeated attempts at intubation enhanced and aggravated the electrocardiographic disturbances during intubation. This is well illustrated in figure 8 in which a first attempt at intubation caused no change; a second attempt resulted in sinus tachycardia with rare ventricular premature contractions, and a third attempt resulted in sinus tachycardia with frequent ventricular premature contractions and runs of bigeminal rhythm. Phases of hypoxia owing to pharyn-

geal or glottic obstruction before intubation also enhanced and aggravated the electrocardiographic disturbances during intubation.

Intubation during the conscious state following preanesthetic medication and topical cocaineization of the pharynx and larynx showed no electrocardiographic changes in 3 of 6 patients. The other 3 demonstrated effects of sympathetic stimulation (sinus tachycardia in 2 of them and paroxysmal tachycardia with auricular fibrillation in the other) which might possibly be influenced both by the psychic reaction and by the sympathetic stimulating effect owing to the absorption of cocaine enhanced by local reflex action.

All the electrocardiographic disturbances observed following endotracheal intubation in this series were transitory, lasting from fifteen seconds to ten minutes. In only one case may a postoperative cardiac complication be attributed to an electrocardiographic disturbance occasioned by endotracheal intubation. This was a patient who underwent a pneumonectomy; during intubation, a decrease in the voltage of the T wave was observed, lasting five minutes. The postoperative course was uneventful for seven days when he was stricken suddenly and died. The autopsy revealed coronary sclerosis.

There have been reports of patients who died suddenly the instant an endotracheal tube was inserted (6, 7).

In the present series of cases all the electrocardiographic changes were *transitory and of no grave consequence*. Because of the high incidence of such disturbances, however, efforts should be made to avoid or minimize them as much as possible. Further studies are now in progress to determine what such efforts should comprise.

SUMMARY

Electrocardiographic determinations during endotracheal intubation in 109 cases following anesthesia with various usual technics demonstrated electrocardiographic disturbances in 68 per cent of the cases at the time of intubation.

The majority of these disturbances, all of which were transitory, consisted of sinus tachycardia.

Other disturbances, in decreasing incidence, consisted of premature ventricular contractions, nodal rhythm, sinus bradycardia, decrease in voltage of the T wave, depression of the ST segment, increase in the PR interval, sinus arrhythmia, ventricular tachycardia and auricular fibrillation.

Insufficient depth of anesthesia, prolonged laryngoscopy with numerous attempts at intubation, respiratory obstruction before intubation, and tracheal irritation after intubation were found to be important factors in the production of such electrocardiographic disturbances.

The authors wish to express their gratitude to the residents of the Anesthesia Staff, the technicians of the Cardiac Section and the members of the Medical Illustration Division at the Bronx Veterans' Administration Hospital for their help and participation in this investigation.

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SOUTHERN SOCIETY OF ANESTHESIOLOGISTS

The first meeting of the newly organized Southern Society of Anesthesiologists will be held at the Hotel Jefferson, St. Louis, Missouri, on March 31-April 1, 1950. All meetings will be held in the Ivory Room. Technical exhibits will open at 9:00 A.M. March 31, and will close 12:00 noon April 1. The program for the meeting follows:

FRIDAY, MARCH 31, 1950

DR. PERRY P. VOLPITTO, Presiding

Technics for Stellate Ganglion Block—An Evaluation

Drs. H. J. Freiheit and R. M. S. Barrett, St. Louis University, St. Louis, Missouri.

Avertin for Basal Anesthesia

Drs. Paul Elsberg, John Andrina, Donald Stubbs, Doctors Hospital, Washington, D. C.

Nembutal in the Fenestration Operation

Dr. Harold Carron, Tampa, Florida.

Prolongation of Spinal Anesthesia Using Levophen

Dr. H. M. Ausherman, Chattanooga, Tennessee.

Luncheon

Dr. John Adriani, Presiding.

Hypertensive Episodes During Anesthesia—With a discussion of Pheochromocytoma

Dr. Edward B. Tuohy, Georgetown University, Washington, D. C.

Epidural Anesthesia in Thoracic Surgery

Dr. Oral B. Crawford, Springfield, Mo.

Oxygen and Carbon Dioxide Tensions as Affected by Amnesic and Analgesic Agents in Obstetrics

Drs. Perry P. Volpitto and J. M. Brown, University of Georgia, Augusta, Ga.

Business Meeting and Election of Officers

(Continued on page 244)