CARDIAC ARRHYTHMIA: EFFECT OF VAGAL STIMULATION AND HYPOXIA * † ‡ §

J. JACOBY, M.D., C. ZIEGLER, M.D., W. HAMELBERG, M.D.,
A. MOGG, M.D., K. KLASSEN, M.D., AND F. FLORY, M.D.

Columbus, Ohio

CARDIAC irregularities which develop during anesthesia are a matter of concern because they may precede serious or fatal cardiac disturbances. Arrhythmias which develop during anesthesia have been attributed to the use of certain anesthetics, such as chloroform and cyclopropane; to drugs, particularly certain vasopressors; to hypoxia; or to vagal reflexes. Among the vagal stimuli which are considered responsible for cardiac irregularities are those associated with instrumentation of the larynx and trachea. A number of articles have indicated that the stimulation of intubation and extubation is frequently responsible for cardiac irregularities (1, 2, 3).

Previous work at this institution indicated that vagal stimulation alone is probably not the cause of cardiac irregularities (4). This work was done on human subjects using direct stimulation of the intact vagus nerve in the thorax, and stimulation of both the proximal and the distal ends of the severed vagus nerve. The stimuli used were both mechanical and electrical. Electrocardiographic tracings taken during stimulation showed no evidence of cardiac arrhythmia when adequate oxygenation was present.

In our previous work a strong stimulus was used, but its nature was different from that which occurs clinically. It was decided, therefore, that an experimental procedure should be devised to test the effect of clinical vagal stimulation (with and without hypoxia) on the cardiac rhythm. Stimulation of the larynx and trachea by endotracheal intubation, having been reported to be a frequent cause of arrhythmias, was used as the means of vagal stimulation. Blood oxygen saturation was continuously observed by means of an ear oximeter which was attached to the patient prior to the induction of anesthesia. Electrocardiographic tracings were taken prior to the induction, following induction, and during and after intubation. Blood oxygen saturations were recorded and correlated with the electrocardiographic pattern.

* From the Department of Surgery (Anesthesia), Ohio State University, Columbus, Ohio.

† Read before the annual meeting of the American Society of Anesthesiologists, Inc., Cincinnati, Ohio, October 26, 1954.

‡ This work was supported by a grant from the Central Ohio Heart Association.

† Accepted for publication February 8, 1955.

METHOD

A separate operating room was set aside for this work and patients who were good surgical risks were selected for study. The procedure was begun an hour before the scheduled time of surgery, so that an ample amount of time would be available for the study.

The usual moderate premedication was used: demerol® 75–100 mg., and atropine or scopolamine 0.3–0.4 mg. Induction of anesthesia was accomplished with Pentothal® and relaxation was obtained with *d*-tubocurarine. The patient's chest was then repeatedly inflated with oxygen by manual pressure on the breathing bag.

Most of the intubations were done by new members of the department who were learning the technique. Intubation was accomplished under direct vision using a Macintosh laryngoscope. As was expected, because the intubations were not done by experts, hypoxia developed in some cases.

After considerable experience had been gained with the combined use of the oximeter and the electrocardiograph, good risk patients were selected in whom anoxia was created by the inhalation of high concentrations of nitrous oxide and low concentrations of oxygen.

The limits of safety were not exceeded by the experimental procedure. In every case the patient was very closely observed by members of the senior staff. Apnea was not produced in any patient. The periods of hypoxia were brief, never exceeding three minutes. The oxygen saturation was not allowed to decrease below 50 per cent; and the degree of hypoxia was less than that which sometimes occurs during intubation by beginners when there is no study being carried out.

RESULTS

A total of 121 patients were included in this study. Group 1 consisted of 110 patients intubated by beginners. The lungs were repeatedly inflated with oxygen, and the blood oxygen saturation was above 96 per cent prior to the beginning of intubation. Group 2 consisted of eleven patients whose blood oxygen saturation was reduced prior to intubation by inhalation of a gas mixture low in oxygen.

Group 1. The 110 patients in this group had oxygen saturations above 96 per cent prior to intubation. During the intubation oxygen saturation remained above 80 per cent in eighty-five patients; oxygen saturation fell below 80 per cent in twenty-five. Of these 25, 15 patients had oxygen saturations below 80 per cent but above 60 per cent, and 10 patients had oxygen saturations of 60 per cent. At times it was necessary to interrupt the procedure to re-oxygenate the patients.

Group 2. Eleven patients inhaled 5–10 per cent oxygen with nitrous oxide until the blood oxygen saturation fell to between 50 and 60 per cent. These patients were then promptly intubated before air breathing could again elevate the oxygen saturation.

TABLE 1

ELECTROCARDIOGRAPHIC CHANGES: OXYGEN SATURATION ABOVE 80%

85 Cases

	Anesthesia Induction		Endotracheal Intubation	
	Cases	%	Cases	%
No change	39	46	37	44
Tachycardia	33	38	40	47
Bradycardia	8	9	0	0
Arrhythmia	5	6	8	9

The *electrocardiographic tracings* were classified into four categories: (1) unchanged, (2) tachycardia of 10 per cent or more, (3) bradycardia of 10 per cent or more, and (4) changes in the electrocardiographic pattern.

Some patients developed electrocardiographic changes after the induction of anesthesia but before tracheal manipulation or intubation. This is in accord with the findings of others. Of the 85 patients whose oxygen saturation was 80 per cent or more, 39 had no change following induction and 37 had no change following intubation. Tachycardia

TABLE 2

ELECTROCARDIOGRAPHIC CHANGES: OXYGEN SATURATION BELOW 80%

36 Cases

	Anesthesia Induction		Endotracheal Intubation	
	Cases	%	Cases	%
No change	17	47	13	37
Tachycardia	13	37	16	44
Bradycardia	3	8	1	3
Arrhythmia	3	8	6	17

was the most common change following both induction of anesthesia and intubation. Slowing of the heart resulted in 8 patients following induction but in none following intubation. Arrhythmia developed in 5 patients (6 per cent) following induction and in 8 (9 per cent) following intubation. Transient extrasystoles were the most common arrhythmias noted (table 1).

Of the total of 121 patients, thirty-six developed blood oxygen saturations below 80 per cent. When oxygen saturation was between 80 and 60 per cent, the incidence of alterations in pulse rate was about

TABLE 3

ELECTROCARDIOGRAPHIC CHANGES DURING INTUBATION
OXYGEN SATURATION 50–60%

11 Cases

	Cases	%
No change	0	0
Tachycardia	9	82
Bradycardia	1	9
Arrhythmia	4	36

the same as that in the well oxygenated patients, with the exception that bradycardia occurred once following intubation in the poorly oxygenated group but did not occur in the well oxygenated group. Arrhythmias, however, occurred in 17 per cent of the poorly oxygenated patients, but in only 9 per cent of the well oxygenated patients (table 2).

There were 11 patients in whom the blood oxygen saturation was reduced to between 50 and 60 per cent. All but one of these had marked changes in pulse rate (91 per cent). Four patients (36 per cent) also developed alterations in electrocardiographic pattern, consisting of coupled beats, auricular flutter, wandering pacemaker, and depressed S–T segment (table 3).

Color changes were not observed in most patients until the blood oxygen saturation was reduced to below 75 per cent. Between 75 and 65 per cent of the patients were considered "dusky"; cyanosis was observed to be mild to moderate when the blood oxygen saturation was between 65 and 50 per cent. No patient became deeply cyanotic because the procedure was interrupted for re-inflation of the lungs with

TABLE 4

ELECTROCARDIOGRAPHIC CHANGES DURING INTUBATION

Oxygen Saturation, %	Pulse, %	Arrhythmia, %
80–100	47	9
60– 80	47	17
50– 60	91	36

oxygen when necessary. The effect of anemia on the appearance of cyanosis was not observed since all patients in this study had normal hemoglobin levels.

COMMENT

Vagal stimulation initiated by tracheal intubation may cause changes in electrocardiographic tracings. When the patient is well oxygenated, these changes are similar in nature and frequency of occurrence to those which follow induction of anesthesia, and appear to be of little consequence.

When blood oxygen saturation is markedly reduced (50 to 60 per cent), pulse changes occur in almost all patients (91 per cent), and the incidence of arrhythmias is four times as great as in well oxygenated patients. Arrhythmias occur in one out of every three poorly oxygenated patients, but only in one out of every ten well oxygenated patients. In the poorly oxygenated patients, the arrhythmias not only are more frequent but are more serious in nature. Carbon dioxide excess probably played no part in producing the high incidence of arrhythmias. These patients were hyperventilated with a mixture of gases low in oxygen, but carbon dioxide removal was normal or greater than normal (table 4).

Patients frequently develop hypoxia when intubated by beginning students who lack skill in the technique, even when the patients' lungs are first inflated with oxygen. In individuals with normal hemoglobin, the onset of slight cyanosis during intubation indicates hypoxia sufficiently great to require a pause for re-inflation of the lungs with oxygen. Anemic patients should not be intubated by beginners, since cyanosis would develop only after more severe hypoxia had occurred and anemic patients are less able to tolerate hypoxia than normal individuals.

The greater the hypoxia, the more serious are the cardiac responses to vagal stimulation.

SUMMARY

1. Vagal stimulation was initiated by tracheal intubation. The resulting electrocardiographic changes were correlated with the blood oxygen saturation.

2. Hypoxia occurred frequently when intubation was performed by beginning students. Hypoxia was also produced in some patients who were good surgical risks by the inhalation of gas mixtures low in oxygen content.

3. Intubation produced arrhythmias in 9 per cent of well oxygenated patients, and in 36 per cent of the most hypoxic patients. Alterations of pulse rate occurred in 47 per cent of the former, and in 91 per cent of the latter.

4. Cardiac responses to vagal stimulation are more frequent and more serious when hypoxia is present.

REFERENCES

1. Burstein, C. L.; LoPinto, F. J., and Newman, W.: Electrocardiographic Studies During Endotracheal Intubation; Effects During Usual Routine Techniques, Anesthesiology **11**: 224 (March) 1950.
2. Rosner, S.; Newman, W., and Burstein, C. L.: Electrocardiographic Studies During Endotracheal Intubation; Effects During Anesthesia with Thiopental Sodium Combined with Muscle Relaxant, Anesthesiology **14**: 591 (Nov.) 1953.
3. Stephen, C. R.; Martin, R., and Nowill, W. K.: Cardiovascular Reactions of Surital, Pentothal or Evipal Combined with Muscle Relaxants for Rapid Anesthesia Induction, Anesth. & Analg. **32**: 361 (Nov.–Dec.) 1953.
4. Morton, D. R.; Klassen, K. R.; Jacoby, J. J., and Curtis, G. M.: Effect of Intrathoracic Vagal Stimulation on Electrocardiographic Tracings in Man, Surg. Gynec. & Obst. **96**: 724 (June) 1953.