CIRCULATORY RESPONSES TO ENDOTRACHEAL INTUBATION IN LIGHT GENERAL ANESTHESIA—THE EFFECT OF ATROPINE AND PHENTOLAMINE

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THE circulatory responses to tracheal intubation during anesthesia have been observed frequently and variously interpreted. Reid and Brace,[1] in 1940, ascribed the electrocardiographic changes occurring during this maneuver to a "vago-vagal" reflex, postulating that both the afferent and efferent pathways of the reflex were mediated over the vagus nerve, while Burstein [2, 3] attributed to reflex responses seen by the electrocardiogram to stimulation of the cardioaccelerator nerves or hypoxia. King [4] and associates believed the reflex mechanism to be "essentially nonspecific in character." They stated that the impulses initiating the reflex are probably carried over the vagus while the effector system is less clearly defined and may be due to decreased parasympathetic or increased sympathetico-adrenal activity.

When intubation of the trachea is performed under light general anesthesia and a relaxant drug, such as succinyldicholine, a definte pressor response and an increase in heart rate is observed. This will be demonstrated subsequently and has been reported by other investigators.[1, 4]

In an effort to help delineate the pathway over which these circulatory reflexes are mediated, we have studied the circulatory responses in patients who were given relatively large doses of atropine or phentolamine intravenously.

METHOD

Our study was performed on 26 subjects, all of whom showed no evidence of cardiac or pulmonary disease and who were to undergo general anesthesia for various surgical procedures. They ranged in age from 22 to 54 years. All received morphine 10 mg. and atropine 0.4 mg. subcutaneously approximately one hour prior to the induction of anesthesia. Arterial blood pressures were measured from the brachial artery with a Statham pressure transducer attached directly to an indwelling needle. Standard lead II of the electrocardiogram was recorded throughout the entire procedure.

The patients were anesthetized with thiamylal sodium intravenously and 100 per cent oxygen was administered from a standard anesthesia apparatus in a partial rebreathing system. Light first plane anesthesia was maintained for approximately five minutes; the subjects were then given 40 mg. of succinyldicholine intravenously. The trachea was intubated and the responses recorded on a direct writing recorder. When the blood pressure and the pulse rate returned to preintubation levels or nearly so, the trachea was extubated and the subject maintained in the anesthetized state. Respiration was either assisted or controlled when respiratory depression or apnea occurred. No specific attempt was made to hyperventilate the patients.

The patients were then divided into two groups. Sixteen patients were given 3 mg. of atropine intravenously; ten were given 5 mg. of phentolamine intravenously. Circulatory responses in the two groups were recorded after the administration of these drugs. After an interval of ten minutes, intubation of the trachea was repeated with the aid of succinyldicholine, and the responses again measured.

RESULTS

The effect of endotracheal intubation on heart rate and blood pressure in the normal lightly anesthetized subject is shown in table 1. Twenty-six subjects showed a significant increase in heart rate, systolic and diastolic pressures. Six of these patients exhibited a transitory cardiac arrhythmia, which was interpreted as premature ventricular contractions. In no

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TABLE 1

EFFECTS OF TRACHEAL INTUBATION ON BLOOD PRESSURE AND HEART RATE

(26 Subjects)

	Before Intubation	After Intubation	t	Prob.
Heart rate	101.2 ± 12.4	115.2 ± 15.9	3.466	.005
Systolic B.P.	112.6 ± 18.7	150.2 ± 28.9	5.460	.005
Diastolic B.P.	80.4 ± 14.3	107.5 ± 19.0	5.693	.005

instance did these persist for longer than 60 seconds.

Table 2 depicts the effects of intravenous atropine on the heart rate, systolic and diastolic pressure in the 16 patients given 3 mg. of atropine. These responses are consistent with those described by other investigators: [5, 7, 8] A significant increase in pulse rate but no significant change in systolic and diastolic blood pressure. Conduction defects were not noted after 3 mg. of atropine intravenously.

The effect of phentolamine on the circulation is shown in table 3. No significant alterations in blood pressure or pulse rate followed the administration of 5 mg. of this drug intravenously. This is contradictory to the statement in Goodman and Gilman [6] that intravenous phentolamine causes a rapid and marked fall in blood pressure, accompanied by tachycardia. However, it must be remembered that our data were obtained on normotensive anesthetized subjects, all of whom were supine throughout the study.

TABLE 2

EFFECT OF ATROPINE (3 MG. I.V.) ON CIRCULATION

	Before Atropine	After Atropine	t	Prob.
Heart rate	103.5 ± 11.6	119.8 ± 7.8	4.5	.005
Systolic B.P.	112.4 ± 20.5	114.8 ± 20.9	0.32	Not sig.
Diastolic B.P.	82.4 ± 14.5	82.6 ± 18	0.05	Not sig.

TABLE 3

EFFECT OF PHENTOLAMINE (5 MG. I.V.) ON CIRCULATION

	Before Phentol.	After Phentol.	t	Prob.
Heart rate	97.5 ± 12.8	110.7 ± 15.3	2.060	.05
Systolic B.P.	113.0 ± 15.5	104.1 ± 17.2	1.151	.15
Diastolic B.P.	77.1 ± 13.4	70.3 ± 12	1.134	.15

TABLE 4

EFFECT OF ATROPINE AND TRACHEAL INTUBATION

(16 Subjects)

	Before Intubation	After Intubation	t	Prob.
Heart rate	119.8 ± 7.8	122.9 ± 7.7	1.128	.15
Systolic B.P.	114.8 ± 20.9	134.9 ± 22.2	2.547	.01
Diastolic B.P.	82.6 ± 18.0	98.6 ± 16.9	2.479	.01

TABLE 5

EFFECT OF PHENTOLAMINE AND TRACHEAL INTUBATION

(10 Subjects)

	Before Intubation	After Intubation	t	Signif.
Heart rate	110.7 ± 14.3	106.4 ± 18.1	0.56	No
Systolic B.P.	104.1 ± 17.2	114.3 ± 26.1	0.98	No
Diastolic B.P.	70.3 ± 12	77.3 ± 17.1	1.00	No

The results obtained in 16 patients given atropine, followed by tracheal intubation, are in table 4. This dose of atropine abolished the increase in heart rate following intubation but did not obtund the pressor response. The systolic pressure significantly increased from a mean of 114 mm. of mercury to 134 mm. of mercury, and the diastolic blood pressure concomitantly increased approximately 15 mm. of mercury. Four subjects in this group developed transitory cardiac arrhythmias.

Those subjects to whom phentolamine was administered, followed by tracheal intubation, showed no significant increase in heart rate, systolic or diastolic blood pressure (table 5). The mean heart rate actually decreased but this is probably due to Subject 26, who exhibited a decrease of approximately 30 beats per minute. Heart rate decreased in four other subjects, the decrease ranging from three to 20 beats per minute. In one patient there was no change, and in the remaining there was an increase of three to ten beats per minute. No cardiac arrhythmias were detected in this group.

COMMENT

When intubation of the trachea is performed under light general anesthesia and a relaxant drug, such as succinyldicholine, a pressor response and an increase in heart rate is obtained. This has been demonstrated by our findings as well as by other investigators.[1, 4]

Large doses of atropine (3 mg. intravenously) will effectively block vagal transmission in man.[5, 7, 8] Although atropinization in our experience did inhibit the acceleration of the pulse rate following tracheal intubation, the pressor response was not altered. This and the fact that no bradycardia occurred, leads us to believe that factors other than "vago-vagal mechanisms" are responsible for the alterations in heart rate and blood pressure accompanying tracheal intubation.

Phentolamine possesses potent adrenergic blocking properties,[9, 10] and in 5-mg. doses, effectively blocks adrenal sympathetic response. With such blockade, the circulatory changes accompanying tracheal intubation no longer occur. This appears to confirm the hypothesis of King and his co-workers that these changes are the result of a sympathicoadrenal discharge rather than a "vago-vagal" phenomenon.

The rise in plasma carbon dioxide and the decrease in hydrogen ion concentration previously reported,[11] occurring during the so-called rapid intubation technique, may affect the tachycardia and pressor response. The moderate but significant rise in plasma carbon dioxide and the decrease in hydrogen ion concentration may be exaggerated in prolonged attempts at tracheal intubation. It is reasonable to assume that an elevated plasma carbon dioxide unaccompanied by hypoxemia could affect the reflex response to tracheal intubation. Cardiac arrest occurring during intubation or extubation has been reported; it may be that an elevated plasma carbon dioxide level could effect the sympathico-adrenal response to intubation in some deleterious manner. The possible effects of an accompanying hypoxemia under similar circumstances might further complicate this response.

SUMMARY AND CONCLUSIONS

Tracheal intubation under light general anesthesia is consistently accompanied by a pressor response, tachycardia, and in some instances cardiac arrhythmias. The circulatory changes are not obtunded by the administration of atropine but are by phentolamine. This indicates that the mechanism is not entirely vagal in nature but may be mediated via the sympathico-adrenal system. The possible consequence of an elevated plasma carbon dioxide on this reflex response is discussed. The effect of atropine and phentolamine on heart rate and blood pressure are also described.

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