REFLEX CIRCULATORY RESPONSES TO DIRECT LARYNGOSCOPY AND TRACHEAL INTUBATION PERFORMED DURING GENERAL ANESTHESIA * †

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The effects of laryngoscopy and tracheal intubation on the cardiovascular system can be readily overlooked during clinical anesthesia. The anesthetist may become so engrossed in the technical aspects of intubation that he has little opportunity to note any abnormal circulatory reaction save it be severe or prolonged. Even conventional records of pulse rate and blood pressure are ordinarily not obtained during the procedure.

The purpose of this study was to record and evaluate whatever cardiovascular changes might accompany these maneuvers. The effect of light as well as deep general anesthesia has been observed for several reasons. First, reflex phenomena are more easily elicited during light anesthesia. Second, intubation of the trachea is frequently performed during light anesthesia supplemented by a curariform drug. Third, Burstein and co-workers have observed that deep anesthesia minimizes electrocardiographic changes incident to tracheal intubation (1).

As intimated, one approach to the problem has been the recording of electrocardiograms. The interpretation of records obtained in this way has been varied (1, 2). Reid and Brace (2) concluded that cardiac reflexes could originate in the trachea, larynx, bronchi or lungs and effect a response by a sudden increase in vagal tone. These reflexes were termed "vagovagal" since both the afferent and efferent paths of the reflex were assumed to be the vagus nerve. Burstein and others (1, 3), using the same method in a larger series of cases, reached a different conclusion, that is, that the majority of changes in the electrocardiogram could be attributed to stimulation of the cardio-accelerator nerves. This would imply an increase in cardiac sympathetic tone rather than the increase in vagal tone suggested by Reid and Brace.

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The electrocardiograph alone is inadequate for the study of cardiovascular reflexes, since it can only indicate changes in rate, rhythm or electrical activity of the heart, and does not necessarily reflect the functional status of the cardiovascular system as a whole. Electrical derangement in the heart may not be accompanied by serious functional changes in the circulation. Conversely, the effects of marked circulatory alterations such as transient hypertension or hypotension may not affect the electrocardiogram. Arterial pulse pressure curves afford a more accurate measure of the functional response of the entire circulatory system. We have, therefore, resorted to the continuous, direct recording of brachial artery pressure and pulse rate during tracheal intubation.

In this series, the majority of observations have been made upon man. A preliminary report of a portion of this study has been presented (4). In addition, observations on the effects of tracheal intubation of the dog have been included in order to ascertain if a species difference in circulatory response to these maneuvers exists.

METHODS

Man

The 46 subjects of this study were patients who required general anesthesia for surgical procedures. All had received morphine or meperidine plus atropine or scopolamine for preanesthetic medication.

Recording.—Direct continuous arterial blood pressure and pulse rate were recorded from a brachial artery, using a Lilly capacitance-type electromanometer and direct inking oscillograph (5, 6). Simultaneous electrocardiograms (lead 2) were secured in 23 patients before and during intubation.

Anesthesia.—General anesthesia was obtained with various combinations of the following agents (tables 1 and 2): Thiopental, nitrous oxide, cyclopropane and ethyl ether. D-tubocuraine and decamethonium bromide were also employed. Inhalation agents and oxygen were administered from an anesthesia machine equipped with a circle filter.

Thiopental alone or thiopental and a curariform drug, when used as the sole anesthetic agents, were administered in the indicated dosages (table 1). In these instances it is our opinion that only first plane surgical anesthesia was attained. Tracheal intubation was not attempted in the 2 patients receiving thiopental alone because of the frequency of coughing following tracheal intubation with thiopental as the sole anesthetic agent.

When inhalation agents were administered an attempt was made to maintain the desired plane of anesthesia sufficiently long so that the removal of the mask at the time of tracheal intubation would not cause a rapid change in the depth of anesthesia. When nitrous oxide, oxygen and a curariform drug were administered, anesthesia was the deepest

TABLE 1

ALTERATIONS IN BLOOD PRESSURE AND PULSE RATE CAUSED BY LARYNGOSCOPY AND TRACHEAL INTUBATION DURING FIRST PLANE ANESTHESIA

Case	Sodium Thiopental, mg.	Decameth. Bromide, mg.	d-Tubocurarine Chloride, mg.	Before Laryngoscopy		During Laryngoscopy		After Tracheal Intubation	
				Pulse Rate	Blood Pressure, mm.	Pulse Rate	Blood Pressure, mm.	Pulse Rate	Blood Pressure, mm.
A. Pentothal									
1	350	—	6	90	100/54	106	141/87	—	—
2	600	—	15	94	162/100	98	191/118	102	195/122
3	600	—	12	96	134/92	102	172/130	112	176/130
4	350	—	14	98	107/68	100	122/80	100	165/100
5	450	—	18	100	118/80	120	152/108	128	162/107
6	350	—	15	92	120/78	138	158/129	134	162/126
7	800	—	12	112	114/73	120	153/97	—	—
8	500	—	—	86	118/92	100	190/123	—	—
9	500	—	—	70	116/77	72	157/107	—	—
10	750	4	—	80	118/73	94	172/110	100	178/115
11	400	4	—	82	166/106	106	207/137	118	255/158
12	250	4	—	100	116/66	108	186/120	124*	210/105
13	175	4	—	120	107/73	127	162/105	136	182/115
14	250	4	—	82	105/65	112	120/75	124	152/100
15	800	4	—	92	106/70	114	134/92	100	149/98
16	400	6	—	104	135/77	106	140/77	120	178/115
17	400	4	—	142	160/94	130	218/119	148	232/132
18	700	6	—	84	134/90	100	147/112	100	177/136
19	300	6	—	62	106/61	48†	131/80	98	166/105
20	200	4	—	84	123/76	88	159/103	100	175/120
21	200	4	—	78	123/78	96	193/119	—	—
22	500	3.5	—	100	93/75	100	115/90	132	140/110
23	400	4	—	80	242/188	80	249/186	102	240/191
24	250	4	—	80	146/72	80	175/100	80	184/95
B. Nitrous Oxide									
25	—	4	—	78	190/110	120	224/140	126	236/142
26	—	4	—	68	152/110	112	240/153	110	250/142
27	—	5	—	90	160/90	90	166/93	118	190/114
28	—	4	—	64	108/72	64	120/80	66	135/85
29	—	4	—	118	138/92	122	168/117	152	198/138
C. Miscellaneous									
30	Thiopental-C_2H_5-Ether (500 mg.)			58	100/67	58	150/91	72	154/94
31	C_2H_5-d-Tubocurarine (15 mg.)			110	120/75	120	136/91	120	150/103
32	N_2O-Ether-d-Tubo. (9 mg.)			98	165/84	114	230/115	164	260/136

* Premature ventricular contractions (E.C.G.) for one minute after insertion of tube. This is the rate when rhythm reverted to normal.

† Pulse rate decrease associated with passage of tube through nose.

obtainable with a flow of 4 liters of nitrous oxide and 1 liter of oxygen or a comparable percentage at higher flows.

In the patients receiving a curariform drug and in 2 individuals to whom cyclopropane was administered, marked respiratory depression or apnea resulted. In these instances assisted or controlled respiration was employed as soon as respirations became inadequate. When the thiopental-curariform mixtures were used, the patient breathed 100 per cent oxygen for several minutes before the administration of the drugs. When respirations became inadequate, a normal or greater than normal ventilation was maintained until laryngoscopy. Laryngoscopy was usually complete in less than fifteen seconds after discontinuing ventilation.

TABLE 2

ALTERATIONS IN BLOOD PRESSURE AND PULSE RATE CAUSED BY LARYNGOSCOPY AND TRACHEAL INTUBATION DURING SECOND AND THIRD PLANE ANESTHESIA

Case	Plane of Stage 3 Anesthesia	Agent	Before Laryngoscopy		During Laryngoscopy		After Tracheal Intubation	
			Pulse Rate	Blood Pressure, mm.	Pulse Rate	Blood Pressure, mm.	Pulse Rate	Blood Pressure, mm.
	Plane 2							
33	Upper	C_3H_6	66	178/100	64	178/100	92	210/120
34	Upper	C_3H_6-Ether	72	154/88	78	156/88	98	195/107
35	Upper	C_3H_6-Decamethonium Bromide (4 mg.)	82	118/72	72	116/72	104	144/98
36	Middle	Thiopent.-C_3H_6-Ether-d-Tubo. (6 mg.)	84	130/74	84	126/72	104	140/82
37	Middle	C_3H_6-Ether	94	236/150	92	236/150	108	238/162
38	Middle	C_3H_6	80	200/98	82	200/98	90	216/108
39	Middle	N_2O-Ether	104	190/112	104	200/122	115	226/134
40	Middle	Thiopent.-C_3H_6-Ether	58	110/74	58	110/74	75	114/80
41	Lower	N_2O-Ether	90	194/98	96	208/98	110	214/98
42	Lower	N_2O-Ether	126	172/108	126	178/114	126	180/116
43	Lower	C_3H_6	80	145/80	80	145/80	80	145/80
	Plane 3							
44	Upper	N_2O-Ether	152	106/60	152	106/60	152	110/60
45	Upper	N_2O-Ether	102	148/100	106	154/104	112	158/102
46	Upper	N_2O-Ether	114	134/90	114	134/90	114	138/86

Coughing, breath holding, or straining at the time of intubation produce circulatory responses independent of those caused by instrumentation of the pharynx, larynx and trachea. For this reason, records obtained during such disturbing reflexes have been excluded from this report.

Dog

Three dogs were studied in a manner similar to that described for man except that blood pressure was obtained from a femoral artery and no preanesthetic medication was administered. Anesthesia was produced by thiopental and decamethonium bromide. Following laryngo-

scopy and tracheal intubation, the endotracheal tube was removed and atropine or scopolamine was repeatedly administered intravenously in 0.05 mg. doses. The effects of tracheal intubation were observed after each dose of the belladonna drug.

RESULTS

Man

The results have been grouped according to the clinical estimate of the depth of anesthesia at the time of laryngoscopy and tracheal intubation. Table 1 presents the data from patients assumed to be in first plane anesthesia and table 2 from those estimated to be in second or third plane anesthesia. In the latter table the patients are listed in order of increasing depth of anesthesia. Blood pressure figures represent maximal levels of systolic and diastolic pressure measured before and after manipulation.

The cardiovascular effects of direct laryngoscopy and tracheal intubation performed in a light plane of anesthesia were generally similar (table 1). When the epiglottis was elevated by direct laryngoscopy

FIG. 1. The rise in blood pressure caused by laryngoscopy and tracheal intubation of man during light general anesthesia (apnea during thiopental-decamethonium bromide anesthesia, case 20). 1. Mask removed and laryngoscope inserted into mouth. 2. Epiglottis raised. The abducted vocal cords were kept exposed while the blood pressure reached a plateau at a maximal value. 3. Tube inserted into trachea. The part of the pressor response contributed by this maneuver is clearly demonstrated. Pressure levels indicated at left margin. Marks on baseline represent five second intervals.

(either with the conventional or the MacIntosh laryngoscope) there was usually an elevation in systolic and diastolic blood pressure within five seconds. Upon insertion of the tube into the trachea a further increase in blood pressure occurred (fig. 1). The average rise in systolic pressure in the 27 patients whose tracheas were intubated was 53 mm. of mercury, while the average rise in diastolic pressure was 34 mm. of mercury. A plateau at or about this peak pressure was maintained for one or two minutes followed by a gradual return to the prelaryngoscopic levels within five minutes.

Cardiac rate was increased an average of 23 beats per minute in the 27 patients whose tracheas were intubated. In no instance did the rate become slower than the control. Laryngoscopy alone affected rate less consistently, for in only 19 of the 32 patients was the rate increased significantly. In one patient (Case 17) laryngoscopy resulted in slowing of the rate. In one other (Case 19) bradycardia occurred with passage of the endotracheal tube through the nose.

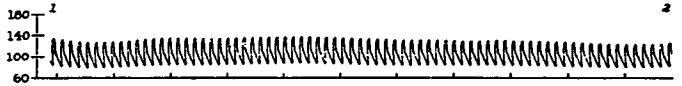

FIG. 2. Blood pressure during apnea. This record, with same pressure and time scale as the previous figure, was obtained from the same patient (Case 20) before any instrumentation was performed. Curarization had produced complete apnea. 1. Mask removed. 2. Mask replaced and artificial ventilation resumed.

As anesthesia was deepened to second or third plane (table 2), changes in blood pressure and pulse rate incident to laryngoscopy and tracheal intubation became less prominent. The pattern of response was of the same type as observed in first plane anesthesia. The average increase in systolic blood pressure in this group, however, was only 16 mm. and in diastolic pressure 10 mm. of mercury. Cardiac rate was increased an average of 13 beats per minute. Again, in one instance (Case 35), laryngoscopy caused some slowing of the heart rate.

No instance of cardiac arrhythmia was noted during laryngoscopy in the entire series of 46 patients. Abnormalities of rhythm developed in 7 patients with tracheal intubation. Six of these were in the group of lightly anesthetized patients. The abnormalities noted were: several extra systoles in 5 patients (Cases 15, 26, 27, 32, 33); extra systoles for fifteen seconds followed by tachycardia of 128 in one (Case 30), and a run of premature ventricular contractions lasting for a minute in another (Case 13). The arrhythmias occurred simultaneously with insertion of the tube into the trachea in 4; in the remainder they developed some seconds later.

All of these circulatory responses discussed appeared to be independent of anesthetic agents used and were unaltered by the omission of curariform drugs (Cases 8, 9, 30, 33, 34). The most important factor appeared to be the depth of anesthesia attained.

Carbon dioxide retention could be ruled out as an etiologic factor in the production of the rise in blood pressure and pulse rate, since adequate artificial ventilation was provided when necessary until the moment the mask was removed; then the laryngoscopy and intubation were accomplished within thirty seconds. Anoxia could likewise be excluded since in 24 patients the artificial ventilation was accomplished with 100 per cent oxygen. A crucial experiment indicating that anoxia or hypercapnia was not involved was carried out as follows: Several patients,

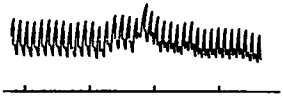

FIG. 3. Influence of cough upon blood pressure. The abrupt peak in blood pressure is of the same duration as the expulsive effort of the cough.

after artificial ventilation had been maintained for a few minutes, were allowed to remain apneic for a period longer than that usually required for tracheal intubation. Changes in blood pressure and pulse rate were insignificant (fig. 2). When artificial ventilation was resumed and laryngoscopy accomplished later, the typical pressor response appeared (fig. 1).

FIG. 4. The effects of laryngoscopy and tracheal intubation of the dog during light anesthesia (apnea during thiopental-decamethonium bromide anesthesia without preanesthetic medication). 1. Mask removed. 2. Epiglottis raised. 3. Tube inserted into trachea. Pressure levels indicated at left margin. Constant recording speed. Marks on baseline represent five second intervals.

As mentioned previously, the circulatory responses recorded were different from those obtained during coughing, with or without a tube in the trachea. Figure 3 is an example of the type of record obtained from a patient who was coughing.

Dog

The effects of laryngoscopy or tracheal intubation in the dog were uniform and were entirely different from those observed in man. There appeared to be marked vagal inhibition of the heart with slowing of the pulse rate and some fall in blood pressure (fig. 4). This effect was

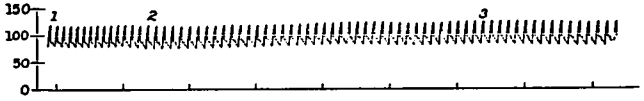

FIG. 5. Laryngoscopy and tracheal intubation of the dog after a belladonna derivative (scopolamine 0.2 mg. total intravenously; 15 kg. dog). This is the same animal as in figure 4. Conditions are the same except for the intravenous administration of scopolamine and small additional doses of thiopental and decamethonium bromide. Legend the same as for figure 4.

readily reproducible. The effects of vagal inhibition could be progressively decreased and finally abolished by the intravenous administration of atropine or scopolamine in successive doses of 0.05 mg. (fig. 5).

COMMENT

During light general anesthesia, direct laryngoscopy or tracheal intubation, uncomplicated by coughing, anoxia or hypercapnia, is capable of producing marked circulatory effects characterized by a rise in

blood pressure and an increase in heart rate. These changes are initiated by the laryngoscope pressing on the base of the tongue or lifting the epiglottis, and are independent of the type of laryngoscope blade employed. Deeper anesthesia obtunds or abolishes these phenomena. The pressor response, however, appears to be more completely blocked by deep anesthesia than does the increase in pulse rate. The fact that increasing depth of anesthesia minimizes the change in cardiac rate during tracheal intubation has been demonstrated by Burstein (1). As far as we are aware this is the first time that a similar effect on blood pressure response has been measured.

Previous studies (1, 2) have emphasized the role of the endotracheal tube in eliciting a reflex circulatory response. It has been demonstrated in this investigation that laryngoscopy alone can cause a pressor effect; insertion of the tube into the trachea augments this effect and, in addition, is capable of producing cardiac arrhythmias.

The increased arterial pressures and the cardiac acceleration which accompany tracheal intubation during light anesthesia indicate that stress may be placed upon the myocardium. The increases of pressure may be of considerable magnitude; systolic pressure rises of 90 mm. of mercury or more were recorded in 4 patients in this series. Since the extent of this response appears to be unpredictable, it would seem advisable to use discretion in performing tracheal intubation during very light anesthesia. A damaged myocardium might not tolerate the additional burden placed upon it by such a procedure. These results support our clinical belief that it is usually wiser to intubate the trachea when the patient is in second or third plane anesthesia. The comparative value of topical anesthesia of the larynx and trachea in mitigating reflex circulatory disturbances is now under investigation.

In this series of patients, tracheal intubation did not produce a vasodepressor response or vagal inhibition of the heart. Vagal-induced cardiac slowing has been reported, and cardiac arrest as a result of vagal activity has been hypothesized as following intubation of the trachea in man. The reaction of Case 19, with cardiac slowing upon passage of the tube through the nose, suggests that the origin of such a reflex may be supraglottic. The belladonna derivative administered preanesthetically to every human subject may have diminished vagal influence upon the heart despite the small doses used (0.4 to 0.6 mg.). This aspect deserves study.

In sharp contrast, intubation of the dog's trachea produced marked vagal inhibition of the heart and some depression of the blood pressure (fig. 4). Atropine or scopolamine could block this response, but at no time could a pressor effect or cardiac acceleration be demonstrated. Apparently the balance between sympathetic and vagal tone in the dog is different from that in man.

The fact that marked vagal reflexes can be evoked by stimulation of the pharynx and larynx in lower animals such as the rabbit (7) has

suggested to many observers that such a reflex may assume great importance during intubation of the human trachea especially under light anesthesia. The data reported herein suggest that such a conclusion is unwarranted.

There is no evidence that the carbon dioxide tension in the blood will rise sufficiently during a thirty second period of apnea occurring after a period of hyperventilation to affect blood pressure significantly. It would be almost impossible to develop anoxia during this period, especially in the 24 instances in which the previous ventilation had been accomplished with 100 per cent oxygen. We have maintained patients without ventilation for periods of a minute without significant changes in blood pressure or pulse rate after an interval of adequate ventilation (fig. 2). The prompt pressor response with laryngoscopy and tracheal intubation usually has attained its peak within thirty seconds of removal of the mask. A delayed and much less abrupt response would be characteristic of gradual accumulation of carbon dioxide or anoxia.

It is difficult to assign a teleologic role to this reflex which we have elicited. We believe it to be essentially nonspecific in character. Evidence in support of such a contention comes from the fact that although we were unable to demonstrate a pressor response by placement of an oral airway, extension of the head or external pressure over the trachea and carotid region, several rather painful maneuvers were capable of producing a rise in pressure during *light* anesthesia. These included pressure on the angles of the jaw (Case 44), traction on the tongue (Cases 6, 38 and 42), and pinching of the ear lobe (Cases 7 and 11). In the first 3 patients, systolic pressure rises averaging 45 mm. of mercury were produced; in the latter 3 there were rises of 30 mm. of mercury.‡

If clinicians attempt to verify the observations reported in this paper, they must recognize that, after intubation, there is a gradual return of blood pressure and pulse rate toward prelaryngoscopy levels. In all probability this is owing to fatigue of reflex receptors, but subsequent deepening of anesthesia or the intervention of buffer mechanisms must also be considered.

Since the vagus is the sensory nerve to the root of the tongue, epiglottis and trachea, sensory impulses initiating the reflex are probably carried over this nerve. The effector system is less clearly defined. The over-all circulatory response noted in these studies might have been produced by decreased parasympathetic or increased sympathetico-adrenal activity.

The hemodynamic alterations responsible for the rise in blood pressure noted in nearly every patient studied during light anesthesia are difficult to analyze completely. With due allowance for the actual level of blood pressure (8), it appears from examination of the pulse pressure that cardiac stroke volume is unchanged or increased at the time of the

‡ It is interesting to note that in the dog, tongue traction produced the same type of vagal inhibition of the heart as did intubation.

increase in pressure (fig. 1). This suggests myocardial stimulation and an adequate venous return. An increase in total peripheral resistance as the result of arteriolar constriction may also have contributed to the pressure rise.

Increases in cardiac rate could be the result of cardio-accelerator action. One is impressed by the fact that the increased blood pressure does not immediately reflexly slow the heart, indicating that cardio-accelerator forces may be even more potent than at first evident. Cardiac acceleration, although often present with laryngoscopy alone, appears to be more marked after insertion of the tube (tables 1 and 2). This confirms the observations of Burstein and others (1) who reported that the majority of the electrocardiograph abnormalities noted during the process of tracheal intubation were sinus tachycardias occurring at the instant the tube was inserted.

Coughing and straining may be complications of laryngoscopy and intubation. Some of the effects of coughing upon blood pressure should, therefore, be mentioned. Coughing superimposes jagged, bizarre peaks upon a normal pulse pressure tracing (fig. 3), even to some extent when the glottis is held open by a tube. These pressure peaks are the result primarily of the physical transmission of increased intrathoracic pressure to the heart and intrathoracic arteries. Paroxysms of coughing may hinder venous return into the thorax. The resultant diminution in cardiac filling is then reflected as decreased pulse pressure. The effects of cough and strain on circulatory pressures have been well described by Hamilton (9, 10).

Circulatory effects have been noted which may continue for a period after the cough and do not appear to be the result of the simple propagation of intrathoracic pressure. Of these, tachycardia appears to be the most common and persistent. In addition, we have noted that the blood pressure level may be altered, occasionally with a diminution in pulse pressure.

SUMMARY

During light general anesthesia, direct laryngoscopy or tracheal intubation, uncomplicated by coughing, anoxia or hypercapnia, is capable of producing decided circulatory effects characterized by a rise in blood pressure and an increase in heart rate. These changes are initiated by the laryngoscope pressing on the base of the tongue or lifting the epiglottis, and are independent of the type of laryngoscope blade employed. Deeper anesthesia obtunds or abolishes these phenomena.

In lightly anesthetized man, cardiac arrhythmias did not appear during laryngoscopy, but were observed as the trachea was intubated. Second or third plane anesthesia diminished this response.

In the lightly anesthetized dog, laryngoscopy and tracheal intubation led to bradycardia and decreased blood pressure.

The mechanism of production of these effects and their clinical significance are discussed.

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The New England Society of Anesthesiologists and The Harvard Medical Society will have a joint meeting Tuesday Evening at 8:00 p.m., October 9, 1951, at the Harvard Medical School Amphitheater D.

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Anesthesia: "Some Problems of Anesthesia," Henry K. Beecher, M.D.

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Nutrition: "Abnormalities of Nutrition and Their Correction," Fredrick Stare, M.D.

Mental Disturbances: "Psychiatric Risks Involved in Surgical Care," Lincoln D. Clark, M.D.

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