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STUDIES OF ANAESTHESIA IN RELATION TO HYPERTENSION
II: HAEMODYNAMIC CONSEQUENCES OF INDUCTION AND ENDOTRACHEAL INTUBATION

BY

C. PRYS-ROBERTS, L. T. GREENE, R. MELOCHE AND P. FOËX

SUMMARY

The electrocardiographic and haemodynamic responses to the induction of anaesthesia, followed by laryngoscopy and endotracheal intubation have been studied in a group of 16 untreated hypertensive patients, and a group of 20 patients receiving antihypertensive therapy up to and including the day of operation. The influence of five different induction agents, thiopentone, methohexitone, propanidid, diazepam, and neuroleptanalgesia induced by a combination of phenoperidine and droperidol were compared. Neuroleptanalgesia caused less arterial hypotension than any of the other agents, but afforded only marginally more protection than other agents against hypertension, tachycardia and dysrhythmia associated with laryngoscopy and tracheal intubation. Both propanidid and diazepam caused dramatic but transient hypotension in a small number of patients and were not investigated further. Unlike its effects in normotensive subjects, methohexitone caused greater hypotension than thiopentone in hypertensive patients. The rationale is presented for the prophylactic blockade of beta-adrenergic receptors to prevent hypertensive crises during laryngoscopy and intubation in both treated and untreated hypertensive patients.

During preliminary studies of the circulatory effects of anaesthesia in treated and untreated hypertensive patients (Prys-Roberts, Meloche and Foëx, 1971) we observed that most of the patients experienced three periods of circulatory instability: during induction, during and after tracheal intubation, and during the immediate period surrounding awakening. Tachycardia and hypertension are well documented complications of laryngoscopy and tracheal intubation in normotensive patients under a variety of anaesthetic techniques (Reid and Brace, 1940; Burstein, Lo Pinto and Newman, 1950; King et al., 1951; Rosner, Newman and Burstein, 1953; Takeshima, Noda and Higaki, 1964; Forbes and Dally, 1970) and during tracheal suction in patients with tracheostomy (Corbett, Kerr and Prys-Roberts, 1969). Dingle (1966) and Forbes and Dally (1970) suggested that the hypertensive response of normal subjects to laryngoscopy and intubation might be enhanced and prove dangerous to hypertensive subjects.

We have compared the haemodynamic effects of five agents for the induction of anaesthesia in treated and untreated hypertensive patients, in the hope of establishing the relative safety of each, and have studied the haemodynamic and electrocardiographic responses of these patients to the subsequent laryngoscopy and tracheal intubation.

CLINICAL MATERIAL AND PROCEDURES

The haemodynamic responses of 36 hypertensive patients were studied during the induction of anaesthesia, and during the sequence of short-term paralysis with suxamethonium followed by laryngoscopy and tracheal intubation. The patients were aged between 24 and 72, and were divided into two groups: those whose hypertension was untreated, and those who had been treated for varying periods of time with antihypertensive drugs, and who were receiving their drugs in the prescribed dosage up to and including the day of operation. Details of the patients are shown in table I.

C. PRYS-ROBERTS, M.A., PH.D., M.B., F.F.A.R.C.S.; L. T. GREENE,* M.D.; R. MELOCHE,† M.D., F.R.C.P(C); P. FOËX, M.D.; The Nuffield Department of Anaesthetics, University of Oxford.

Present addresses:

*Department of Anesthesiology, Columbia-Presbyterian Medical Center, New York, U.S.A.

† Hôpital Notre Dame, Montreal 133, P.Q., Canada.

TABLE I

Details of patients included in the study, their anti-hypertensive treatment, and haemodynamic values measured during the awake control period. Patients are divided into groups according to the induction agent used.

Patient	Sex	Age	Arterial pressure (mm Hg)	Cardiac output (l./min)	Heart rate	Systemic vascular resistance (units*)	Stroke vol. (ml)	Anti-hypertensive treatment	Premedication
Thiopentone induction									
01	F	63	162/72	6.26	66	1302	95	AMD	Nil
02	M	52	136/76	—	62	—	—	AMD, N–K	Nil
03	M	50	212/120	7.24	78	1667	93	AMD, B, N–K	Nil
09	M	59	147/83	4.73	78	1824	61	AMD, N–K	Nil
13	F	61	128/84	—	105	—	—	B, N–K	Nil
14	F	27	208/112	4.50	46	2557	98	AMD, B, N–K	Nil
15	F	68	178/76	4.27	63	2115	68	Reserpine, tranquillizer	Nil
16	F	69	180/86	3.60	77	2641	47	AMD	Nil
19	F	68	214/90	3.86	85	2836	45	Reserpine	Nil
20	F	47	215/98	5.97	99	1914	60	B, N–K	Nil
04	M	59	200/99	4.90	62	2625	79	Nil	Nil
05	F	72	218/84	4.14	64	2267	65	Nil	Nil
07	M	57	184/103	6.02	72	1752	84	Nil	Nil
10	M	71	229/118	4.78	50	2591	100	Nil	Pa 10, H 0.2
12	M	55	162/82	6.24	64	1359	100	Nil	Pa 20, H 0.4
Neuroleptanalgesia induction									
21	F	70	204/102	4.05	82	2644	49	Nil	Nil
22	F	61	230/116	—	81	—	—	Nil	Nil
25	M	68	232/104	—	54	—	—	B. N–K	Nil
26	F	44	170/92	6.01	67	1444	90	Dbq, N–K, tranquillizer	Nil
27	M	24	196/104	6.56	85	1681	77	Nil	Nil
28	M	44	178/100	4.28	59	2353	73	B. N–K	Nil
29	M	51	165/95	4.66	84	2126	44	Guanethidine	Nil
31	M	51	160/86	6.74	60	1352	110	Reserpine, tranquillizer	Nil
38	F	69	183/88	4.59	75	2176	55	Nil	Nil
Propanidid induction									
32	F	49	288/128	—	54	—	—	Nil	Pa 20, H 0.4
33	F	37	164/88	—	58	—	—	Nil	Pa 20, H 0.4
34	F	51	206/110	—	72	—	—	AMD, N–K	Pt 100, A 0.6
35	F	54	281/143	4.38	99	3631	44	Nil	Pa 20, H 0.4
Methohexitone induction									
40	F	65	195/80	5.59	84	1715	67	Nil	Nil
41	M	46	176/92	—	67	—	—	Nil	Nil
42	F	45	208/112	—	60	—	—	Nil	Nil
43	F	63	175/105	—	100	—	—	Nil	Pa 20, A 0.6
44	M	69	195/80	—	75	—	—	AMD, tranquillizer	Pt 50, A 0.6
45	M	69	200/125	—	115	—	—	AMD, phenobarbitone	Pt 50, A 0.6
Diazepam induction									
36	F	72	190/105	—	92	—	—	AMD, N–K	Nil
37	F	69	160/70	—	75	—	—	AMD, N–K	Nil

Premedication: Dose in mg; Pa = papaveretum; Pt = pethidine; H = hyoscine; A = atropine.

Anti-hypertensive drugs: AMD = methyldopa; B = bethanidine; N–K = Navidrex–K (cyclopenthiazide with slow release potassium chloride); Dbq = debrisoquine sulphate.

*Resistance units: dyne sec cm^{-5}

The patients were interviewed and examined by one of us on the day preceding the study, and their consents to the observations were obtained after a full explanation of the purpose of the studies, and pre-operative assessment as described in full by Prys-Roberts, Meloche and Foëx (1971). Although most of the patients were not premedicated since baseline measurements on the conscious, undepressed, resting patient formed an integral part of the complete studies, some patients were premedicated according to their own or their anaesthetist's wishes.

The patients were brought to the anaesthetic room in their own beds, and lay in a supine position with one pillow throughout the studies. Under local analgesia, an 18-gauge Teflon cannula (Becton-Dickinson Longdwel) was inserted percutaneously into a brachial artery. In some patients, a larger version of the same cannula was inserted into the contralateral basilic vein, and a 90-cm nylon catheter was floated centrally until its tip lay in the right atrium, right ventricle or pulmonary artery. Standard electrocardiographic limb and chest leads were applied.

Arterial pressure was measured with a Statham P37 miniature pressure transducer connected directly to the arterial cannula, and right heart pressures were measured with a Statham PM131 TC transducer. Instantaneous heart rate was measured with a Neilson tachometer (Devices Ltd) triggered by the R wave of the e.c.g. Cardiac output was measured by the dye dilution method, using a Waters XC-302 cuvette densitometer and amplifier to detect and amplify the changing concentration of indocyanine green in arterial blood. All variables were simultaneously recorded on an Elema-Schönander EM 81 ink-jet recorder, and displayed on a four-channel oscilloscope (LAN electronics). Details of the methods were given in the previous communication (Prys-Roberts, Meloche and Foëx, 1971).

The choice of induction agent was determined chronologically; the first 15 patients received thiopentone sodium (2.5 per cent) in sufficient dosage (50–200 mg) to ensure loss of consciousness and acceptance of face-mask administration of the maintenance gas mixture—1 per cent halothane in nitrous oxide (7 l./min) and oxygen (3 l./min). The subsequent 9 patients were induced by neuroleptanalgesia (NLA) produced by incremental doses of phenoperidine (0.1 mg) and droperidol (1.0 mg) given over a period of 5–15 minutes until the patient appeared to be asleep (though responding to command) and accepted an anaesthetic facemask. The range of the accumulated doses of the drugs used to produce NLA were: phenoperidine 0.8–1.6 mg and droperidol 5–9 mg.

Four patients received propanidid (200–300 mg) in a 5 per cent solution, administered through the right heart catheter over a period of 15–20 seconds, followed by facemask administration of the maintenance gas mixture as soon as the hyperventilatory phase had commenced. Two patients were induced with 10–20 mg diazepam given over a period of 30 seconds, followed immediately by maintenance anaesthesia.

The last group of 6 patients received methohexitone (Brietal Sodium) in a dose of 80–100 mg, administered as a 1 per cent solution over a period of 30 seconds.

Prior to the moment of induction, all the patients were allowed to lie undisturbed for about 5 minutes while continuous recordings were made of the electrocardiograph (usually lead II), instantaneous heart rate, arterial pressure and right heart or pulmonary arterial pressures. Measurements of cardiac output were performed in duplicate during this period. The patients were not told when the induction agent would be administered, in the hope that pre-induction apprehension would not cause haemodynamic changes which would obscure the pharmacological effects of the induction agents.

Following the administration of the induction agent, the patients breathed the maintenance gas mixture through a Magill attachment and facemask for 5–10 minutes until arterial pressure and heart rate had stabilized. This stage was designated the post-induction steady-state period (1), and in many patients served also as a pre-intubation control period. In other patients, studies of baroreceptor function were performed during this interval, and a further measurement was made to serve as a pre-intubation control value during steady-state anaesthesia (2).

Endotracheal intubation.

When a steady state of spontaneous ventilation was established, suxamethonium 75–100 mg was administered intravenously, and when the muscle fasciculations appeared gentle manual inflation of

TABLE II

Responses of treated and untreated hypertensive patients to induction of anaesthesia with thiopentone, and the subsequent sequence of muscular paralysis, laryngoscopy and tracheal intubation, and maintenance anaesthesia. Mean values are shown with SD in parentheses.

	TREATED HYPERTENSIVES ($n=10$)						UNTREATED HYPERTENSIVES ($n=5$)					
	SAP (mm Hg)	DAP (mm Hg)	MAP (mm Hg)	HR (beats/ min)	$\dot{Q}$ (l./min)	SVR (dyne sec cm^{-5})	SAP (mm Hg)	DAP (mm Hg)	MAP (mm Hg)	HR (beats/ min)	$\dot{Q}$ (l./min)	SVR (dyne see cm^{-5})
Awake (control)	178 (34)	90 (16)	121 (21)	76 (18)	5.05 (1.29)	2107 (531)	199 (27)	97 (15)	134 (17)	71 (14)	5.25 (0.94)	2118 (550)
After induction												
1 min	175 (41)	96 (29)	123 (32)	88 (16)	—	—	161 (17)	88 (13)	113 (11)	80 (8)	—	—
3 min	157** (40)	87 (26)	110** (30)	82 (18)	—	—	163 (26)	86 (7)	111 (13)	70 (7)	—	—
Steady-state (1)	130*** (35)	72** (20)	91*** (22)	68 (13)	3.94** (0.92)	2022 (395)	130* (24)	71* (10)	91* (12)	63 (6)	4.53 (1.10)	1645* (296)
Steady-state (2)	127 (34)	71 (19)	90 (21)	70 (15)	4.03 (1.13)	1950 (288)	126 (25)	69 (10)	88 (12)	63 (7)	4.08[2]	1868[2]
After suxamethonium	124 (26)	71 (15)	89 (18)	75 (13)	—	—	114 (26)	63 (13)	81 (16)	63 (3)	—	—
After laryngoscopy (maximum values)	177††† (25)	101†† (14)	126†† (17)	96†† (13)	—	—	170†† (34)	95†† (8)	120††† (14)	80 (5)	—	—
Steady-state (3)	121 (19)	68 (17)	86 (16)	70 (14)	3.74 (0.98)	1838 (320)	109 (16)	58† (12)	74 (12)	58† (7)	3.26[2]	1869[2]

Paired two-tailed *t*-test: comparison of values against awake control

$P<0.05$	*
$P<0.01$	**
$P<0.001$	***

Comparison of values with steady-state (2)

$P<0.05$	†
$P<0.01$	††
$P<0.001$	†††

SAP = systolic arterial pressure
DAP = diastolic arterial pressure
MAP = mean arterial pressure
HR = heart rate
$\dot{Q}$ = cardiac output
SVR = systemic vascular resistance

[2] means based on two measurements only

the lungs was started. It was established in an early group of patients that haemodynamic changes did not occur within the first 30–40 seconds if the lungs were not inflated, and that manual inflation for a period of 1 minute without further interference produced no significant changes from the control values. Laryngoscopy was therefore delayed for 20 seconds after visible fasciculations had ceased, and the laryngoscope (Macintosh) was held in position for optimal visualization of the vocal cords for 15–20 seconds before spraying the larynx and trachea with 2 ml of 4 per cent lignocaine. A cuffed oral endotracheal tube (Oxford pattern), lightly lubricated with lignocaine gel, was inserted and its cuff immediately inflated. Manual inflation of the lungs was maintained with the same gas mixture as that used before intubation, until adequate spontaneous ventilation had returned. The patients then breathed spontaneously for as long as was necessary to achieve a further steady-state (3) of ventilation and circulation.

Estimates of cardiac output, accompanied by sampling of arterial blood (and in some patients mixed venous blood) for estimation of P_{O_2}, P_{CO_2} and pH, were obtained during the pre-induction control period, after the induction of NLA in the appropriate patients, during the post-induction and pre-intubation steady-state period, and finally during the post-induction steady-state period.

In 4 patients who underwent dental operations, blind nasotracheal intubation was performed after inhalation of approximately 8–10 per cent carbon dioxide in 60 per cent nitrous oxide and 30 per cent oxygen for about 60–90 seconds. The tube entered the larynx on the first attempt in every patient, and a pharyngeal pack was then manually inserted.

RESULTS

The data from this study are presented in two ways. Firstly as a comparison of the circulatory responses to the induction and tracheal intubation sequences in five groups of patients according to the induction agents used, each group comprising both treated and untreated patients. Secondly, as a comparison of the same sequences in two groups: treated (Group 1) and untreated (Group 2) hypertensive patients, irrespective of the agents used for induction.

Induction of anaesthesia.

Thiopentone. In 10 treated hypertensive patients (table II) a mean dose of 155 mg (SD 54) produced a 12 per cent fall of systolic arterial pressure within 3 minutes, and the steady-state values of both systolic and mean arterial pressures for the whole group were more than 25 per cent below the control values, and fell by more than 50 per cent in 2 patients, both female and over 65 years old, though with well controlled arterial pressure in everyday life. A 22 per cent fall in cardiac output was largely responsible for this arterial hypotension, since the systemic vascular resistance fell by only 4 per cent (n.s.). Heart rate increased transiently during the first minute without a significant change of arterial pressure, but the steady-state values of heart rate were not significantly lower than the control values.

In a smaller group of 5 untreated hypertensive patients (table II) arterial pressures fell progressively to a maximum of 35 per cent below the awake controls ($P < 0.02$) during the steady-state period of maintenance, but unlike the treated hypertensive group, this arterial hypotension was predominantly due to a significant reduction of systemic vascular resistance (22 per cent, $P < 0.05$), and although cardiac output fell by 0.72 l./min (mean), this difference did not reach statistical significance.

Neuroleptanalgesia. Before applying the facemask, a state of neuroleptanalgesia (NLA) was achieved in which the patients would lie motionless, apparently asleep, but responding to command. In this state the mean arterial pressure was 15 per cent below the control value ($P < 0.02$) in a combined group of treated and untreated hypertensive patients (table III). This fall was almost entirely due to a reduction of systemic vascular resistance ($P < 0.05$). No significant changes occurred during the first 3 minutes after starting maintenance anaesthesia, but by the time a steady-state had been achieved, a further fall in both systolic and mean arterial pressures had occurred as a result of a 14 per cent reduction of cardiac output ($P < 0.001$). No significant changes in heart rate occurred at any stage.

With one exception, there was no obvious difference between the responses of the treated and untreated patients in this group. One patient, a 70-year-old fit lady with untreated essential hyper-

tension, developed a severe sinus bradycardia leading to heart block and hypotension within 4 minutes of the slow intravenous administration of droperidol 5 mg (fig. 1). She responded well to intravenous atropine (0.6 mg), but despite an increase in heart rate from 25 to 89 beats/min, she remained hypotensive (112/70 compared with 204/102 mm Hg) when full NLA had been established, and her cardiac output had fallen from 4.05 l./min to 2.74 l./min. Although the changes were transient in this patient, the findings emphasize the need for continuous monitoring of e.c.g., heart rate and arterial pressure in elderly hypertensive patients.

Propanidid. In a group of 3 untreated hypertensive and 1 treated hypertensive patients, all of whom had high arterial pressures at rest (table III), there was a significant reduction (32 per cent, $P < 0.05$) of systolic and mean arterial pressures, together with a marked tachycardia, within 1 minute of completing the rapid intravenous administration of propanidid. After 3 minutes, there was little further change in the arterial pressure, though heart rate had returned to control levels. When a steady-state had been achieved, both systolic and mean arterial pressures were more than 32 per cent below the conscious resting values, with a moderate reduction of heart rate (18 per cent).

Diazepam. In 2 patients, diazepam caused marked but transient hypotension during the induction period (table III), and was associated with excessive hypertension following laryngoscopy.

Methohexitone. In 6 patients induced with methohexitone (2 treated and 4 untreated) there was a significant reduction of systolic (-19 per cent) and mean (-15 per cent) arterial pressure (table III), together with an 18 per cent increase of heart rate. Cardiac output changes were measured in only 1 patient in this group, whose values fell from 5.59 l./min in the awake control period to 3.98 l./min in the steady-state of maintenance anaesthesia. In this patient, there was a slight increase in systemic vascular resistance, thus the hypotension was entirely due to a decrease in cardiac output related to decreased stroke volume. The arterial hypotension was progressive during the first 5 minutes after induction, the values of systolic and mean arterial pressure in the steady-state period being 41 and 36 per cent respectively below the awake control values.

Since systolic pressures fell much more than the diastolic pressures during the first minute, it is probable that the hypotension at this stage was also largely due to reduced cardiac output rather than decreased vascular resistance.

Comparison of treated and untreated patients. There were significant differences ($P < 0.05$) between the values of systolic, diastolic and mean arterial pressures in untreated and treated hypertensive patients during the awake control period, but not during any other stage of the investigations. The mean values for all variables after the induction of anaesthesia were remarkably similar in the two groups of patients at each stage of measurement (table IV). In the untreated hypertensive group, the greater fall in arterial pressure from the awake control to the steady-state value was associated with a greater reduction of systemic vascular resistance (2269 to 1993 dyne sec cm^{-5}) compared with the treated hypertensive patients (1925 to 1811 dyne sec cm^{-5}), though the difference between the two groups did not reach statistical significance at the 5 per cent level.

Sequence of tracheal intubation.

Thiopentone. In 10 treated hypertensive patients, no significant change of arterial pressure or heart rate occurred following the administration of suxamethonium (75–100 mg) intravenously. The maximum values of systolic, diastolic and mean arterial pressures, and of heart rate occurring within 30–60 seconds of laryngoscopy and intubation, were significantly higher than the steady-state values during maintenance anaesthesia (table II), and the mean values for the group following laryngoscopy were almost identical with the awake control values for arterial pressure, but heart rate was significantly higher ($P < 0.05$) than in the conscious patient. Three patients had systolic arterial pressures not more than 50 mm Hg higher than their conscious values, and one patient exceeded his conscious systolic arterial pressure by 92 mm Hg. The steady-state values achieved after intubation did not differ significantly from those before intubation.

TABLE III

Circulatory responses of four mixed groups (treated and untreated) of hypertensive patients to induction of anaesthesia with neuroleptanalgesia (group A), propanidid (group B), methoexitone (group C) and diazepam (group D), and the subsequent sequence of muscular paralysis, laryngoscopy and endotracheal intubation, and maintenance anaesthesia. Mean values are shown with SD in brackets.

	NEUROLEPTANALGESIA $n=9$ (5 T, 4 UT)						PROPANIDID $n=4$ (1 T, 3 UT)						METHOHEXITONE $n=6$ (2 T, 4 UT)						DIAZEPAM $n=2$ (2 UT)			
	SAP	DAP	MAP	HR	$\dot{Q}$	SVR	SAP	DAP	MAP	HR	$\dot{Q}$	SVR	SAP	DAP	MAP	HR	$\dot{Q}$	SVR	SAP	DAP	MAP	HR
Awake (control)	191 (27)	99 (9)	133 (12)	72 (12)	5.27 (1.13)	2010 (442)	235 (60)	117 (24)	159 (39)	71 (20)	4.38[1]	2901[1]	192 (13)	99 (18)	130 (14)	84 (21)	5.59[1]	1715[1]	190	105	133	84
Awake (after NLA)	164* (33)	88* (13)	113* (18)	74 (16)	5.03 (1.34)	1795* (308)																
After induction																						
1 min	—	—	—	—	—	—	161* (26)	95* (17)	117* (23)	113* (17)	—	—	155** (20)	89 (15)	111* (14)	99 (15)	—	—	145	75	98	81
3 min	167* (23)	93 (11)	116** (15)	74 (12)	—	—	166* (41)	94 (19)	117 (27)	73 (15)	—	—	132*** (14)	78** (14)	96*** (12)	91 (11)	—	—	—	—	—	—
Steady-state (1)	151*** (29)	84*** (14)	108*** (18)	73 (16)	4.53*** (0.96)	1905 (302)	150* (32)	83* (14)	105* (20)	58 (13)	—	—	114*** (24)	67** (15)	83** (17)	78 (21)	3.98[1]	1807[1]	135	70	92	82
Steady-state (2)	149 (35)	83 (15)	107 (20)	71 (14)	4.30 (1.01)	1989 (330)	150 (32)	83 (14)	105 (20)	58 (13)	—	—	112 (26)	64 (15)	80 (17)	78 (24)	3.98[1]	1807[1]	135	70	92	82
After suxamethonium	141 (38)	75 (14)	97 (21)	72 (14)	—	—	165 (56)	96 (38)	118 (45)	67 (16)	—	—	115 (31)	67 (20)	83 (23)	76 (17)	—	—	—	—	—	—
After laryngoscopy (maximum values)	194†† (35)	103†† (14)	135†† (22)	90† (25)	—	—	229††† (44)	134† (35)	166†† (37)	89†† (7)	—	—	171† (66)	98† (32)	122† (42)	96† (9)	—	—	250	130	170	115
Steady-state (3)	141 (46)	79 (20)	102 (26)	77 (17)	4.53 (1.17)	1799 (280)	148 (36)	82 (20)	106 (23)	62 (17)	2.57[1]	3296[1]	110 (40)	65 (23)	80 (28)	65 (16)	3.56[1]	1796[1]	173	90	117	87

Key Symbols, abbreviations and units as in table II. [1] = measurements in one patient only. T = treated. UT = untreated.

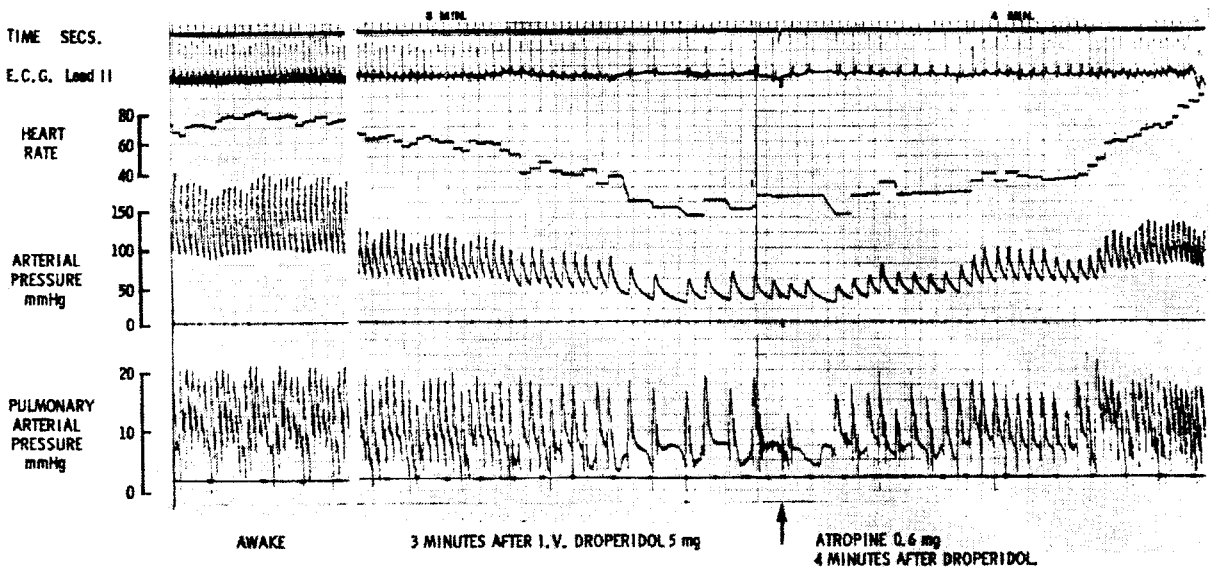

FIG. 1

Development of marked sinus bradycardia leading to heart block following the administration of droperidol (5 mg) in patient No. 21, and the return to sinus rhythm following atropine (0.6 mg).

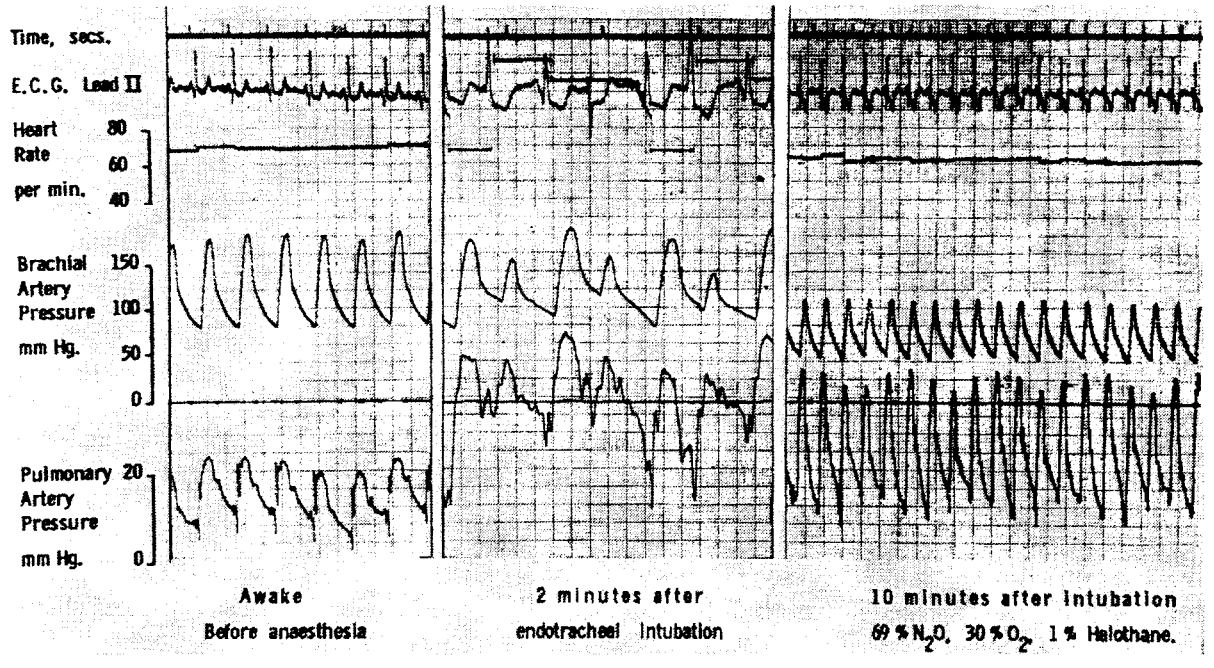

FIG 2

Circulatory changes occuring during and after laryngoscopy and tracheal intubation (pateint No. 15). The lefthand panel shows the values during the awake control period, at which time the patient's cardiac output was 4.27 l./min. Arterial hypertension occurred within 15 seconds of laryngoscopy, despite having fallen to 100/50 mm Hg after induction. Tracheal intubation was accompanied by prolonged ventricular bigeminy and further systemic hypertension. Pulmonary hypertension was also noted at this stage (centre panel) and persisted into the steady-state of maintenance anaesthesia. Depression of the S-T segment and T-Wave inversion also persisted into maintenance anaesthesia, indicating myocardial ischaemia in association with the low arterial pressures. Cardiac output fell to 2.84 l./min during maintenance anaesthesia; thus, in association with the elevated pulmonary arterial pressures, it may be estimated (even in the absence of left atrial pressures) that the resistance of the pulmonary vascular bed had more than doubled.

Similar directional changes were observed in 4 untreated hypertensive patients (table II), and although the data are not amenable to statistical comparison because of the small numbers in this group, the mean values for all variables are sufficiently similar to those of the treated hypertensive group, at each stage, that it is unlikely that a real difference exists.

Neuroleptanalgesia. No significant changes in arterial pressure or heart rate accompanied the administration of suxamethonium to 9 patients induced by neuroleptanalgesia (table III). The changes which followed laryngoscopy and intubation were of a similar magnitude to those in patients receiving thiopentone, and there was no significant difference between these values for arterial pressure and those in the awake control period, although heart rate was elevated by 18 beats/min ($P < 0.02$). In individual patients, the systolic arterial pressure after intubation exceeded their awake controls in 5 patients, 1 of whom had an increase of 66 mm Hg.

Propanidid. No significant haemodynamic changes accompanied the administration of suxamethonium in 4 patients induced with propanidid. Arterial pressures increased significantly (table III) following laryngoscopy and intubation, and the mean values for the group were not significantly different from the awake control values. Systolic arterial pressure exceeded the awake control in 2 patients, one by 22 mm Hg, and in the other by 76 mm Hg reaching a maximum value of 276/176 mm Hg. Heart rate also increased significantly in the group as a whole, although the fastest rate recorded (96) was rather lower than the fastest rates recorded in the thiopentone group (7 out of 10 developed heart rates in excess of 100) and the NLA group (maximum rate 140 beats/min, 3 out of 9 over 100 beats/min).

Methohexitone. There was a greater percentage increase in systolic and mean arterial pressures (53 per cent, $P < 0.05$) following laryngoscopy and intubation (table III), as compared with the other groups (thiopentone 39.4 per cent; NLA 27 per cent; propanidid 51 per cent). Individual systolic pressures exceeded the awake control values in 2 patients, +39 mm Hg in one and +72 mm Hg in the other.

Comparison of treated and untreated patients. Mean values for arterial pressures, heart rate and cardiac output in the pre-intubation steady-state were not significantly different in the two groups of patients (table IV), and the administration of suxamethonium had no significant effect in either group. Following laryngoscopy and intubation, arterial systolic, diastolic and mean pressures, and heart rate were significantly elevated as compared with the pre-intubation steady-state, and there was no difference between the responses of the treated and untreated patients. Postintubation steady-state values were similar in both groups, and were not significantly different from the pre-intubation levels. Following intubation, there was a higher incidence of hypertension exceeding the awake control value in the treated hypertensive patients. Mean arterial pressure was elevated above control values in 70 per cent of this group as compared with 42 per cent of the untreated hypertensive patients, and 4 treated hypertensives had excessive hypertension following laryngoscopy and intubation (>50 mm Hg above awake control systolic arterial pressure) compared with only 1 untreated hypertensive patient. Three of these four treated patients were well controlled and almost normotensive as a result of treatment, and there were no obvious explanations as to why they should produce such a marked sympathetic response to intubation.

Disorders of cardiac rhythm.

Many of the patients in both treated and untreated groups had disorders of cardiac rhythm during the awake control period, usually premature ventricular contractions. The incidence was higher in the untreated group (6 out of 13) than in the treated group (2 out of 16) ($\chi^2 = 2.56$, n.s.), although there was no consistent relationship between the incidence of cardiac dysrhythmia in the conscious patient, and the arterial pressure. Four patients developed premature ventricular contractions during the induction of anaesthesia having previously been free from dysrhythmia, and in 4 others the incidence of dysrhythmia increased during induction.

Administration of suxamethonium was not associated with the onset of dysrhythmia in any patient, but was associated with the cessation of pre-existing dysrhythmia in 8 patients (6 untreated and 2 treated).

Laryngoscopy, laryngeal and tracheal spraying,

TABLE IV

Responses of treated and untreated hypertensive patients to induction of anaesthesia, and the sequence of muscular paralysis, laryngoscopy, endotracheal intubation and subsequent maintenance anaesthesia. Mean values are shown with SD in parentheses.

	TREATED HYPERTENSIVES ($n=16$)						UNTREATED HYPERTENSIVES ($n=14$)					
	SAP	DAP	MAP	HR	$\dot{Q}$	SVR	SAP	DAP	MAP	HR	$\dot{Q}$	SVR
Awake (control)	181 (31)	93 (14)	125 (18)	72 (16)	5.19 (1.18)	1925 (207)	211 (36)	104 (18)	142 (24)	74 (14)	5.00 (0.87)	2269 (114)
After induction												
1 min	169 (36)	92 (24)	118 (27)	82* (18)	—	—	165*** (26)	92* (15)	117*** (19)	92* (18)	—	—
3 min	161** (35)	90 (22)	113** (25)	78 (17)	—	—	159*** (30)	86*** (13)	110*** (18)	77 (10)	—	—
Steady-state (1)	139*** (35)	76*** (18)	97*** (22)	67 (14)	4.28** (1.02)	1811 (105)	137*** (28)	76*** (14)	97*** (18)	69 (15)	3.89** (0.70)	1993 (200)
Steady-state (2)	134 (33)	74 (17)	95 (21)	67 (14)	4.14 (1.11)	1833 (115)	137 (36)	75 (17)	97 (22)	68 (16)	3.80 (0.82)	2040 (185)
After suxamethonium	132 (41)	76 (24)	95 (29)	72 (12)	—	—	136 (31)	74 (15)	94 (20)	69 (15)	—	—
After laryngoscopy (maximum values)	186††† (39)	105††† (23)	133††† (28)	91††† (16)	—	—	194††† (43)	106††† (24)	136††† (29)	90††† (19)	—	—
Steady-state (3)	133 (36)	74 (19)	95 (24)	69 (14)	4.03 (1.22)	1883 (128)	126 (34)	70 (20)	90 (23)	66 (18)	3.80 (0.95)	1892 (170)

	Treated	Untreated
Thiopentone	10	4
Neuroleptanalgesia	5	4
Propanidid	1	3
Methohexitone	0	3

Significance levels are denoted by the following symbols:

Paired two-tailed *t*-test: comparison of values against awake control	$P<0.05$	*
	$P<0.01$	**
	$P<0.001$	***
comparison of values with steady-state (2)	$P<0.05$	†
	$P<0.01$	††
	$P<0.001$	†††

Key Symbols, abbreviations and units as in table II.

and tracheal intubation were associated with a high incidence of dysrhythmia, usually premature ventricular contractions or ventricular bigeminy (fig. 2). These dysrhythmias occurred in 6 out of 13 untreated patients, and 5 out of 16 treated patients, and in every case they followed the onset of tachycardia and hypertension. In only 1 patient of the 44 hypertensive patients studied to date have we observed a bradycardia in response to laryngoscopy and intubation (fig. 3). One patient, a 65-year-old untreated hypertensive lady (195/80 mm Hg awake), developed wandering pacemaker shortly after induction with methohexitone 100 mg, together with occasional premature ventricular contractions, but immediately after laryngoscopy she developed ventricular tachycardia for a period of 10 seconds followed by further premature ventricular contractions.

Quantification of the changing incidence of dysrhythmia in patients with pre-existing dysrhythmia presents more difficulty than the quantification and statistical analysis of those arising de novo. We have applied cumulative sum (cusums) procedures (Woodward and Goldsmith, 1964) to identify statistically significant changes in the incidence of dysrhythmias associated with clinical events (fig. 4). Since these procedures are not widely known and used in the medical field, details of their application, and the manner in which the results are presented, the "Manhattan diagram", are described in brief in an appendix to this paper.

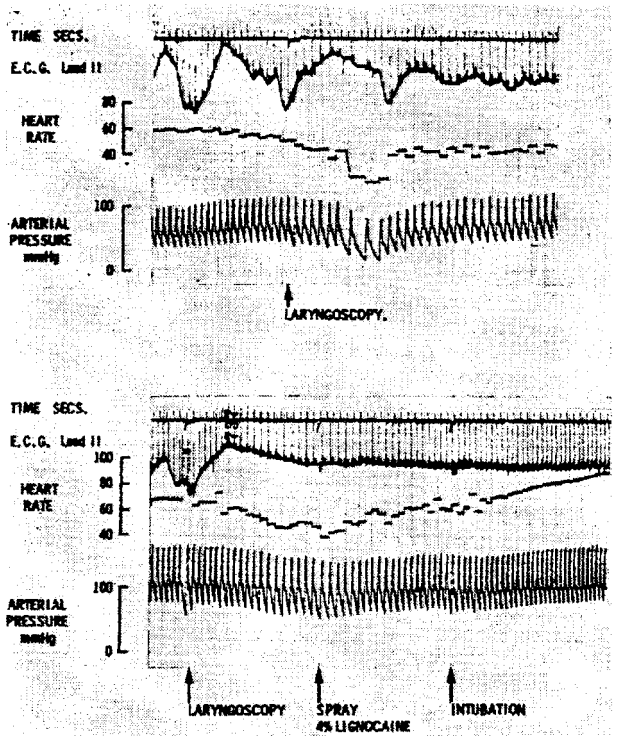

FIG. 3

Vagal reflex response to laryngoscopy. The upper panel shows the development of marked bradycardia shortly after introduction of the laryngoscope in patient No. 27 who had not previously received atropine. Following atropine 0.6 mg (lower panel), the initial heart rate was higher, but laryngoscopy still produced a bradycardia of slightly less severity before the sympathetic responses appeared.

Effect of blind nasotracheal intubation.

In 4 patents, nasotracheal intubation after inhalation of 10 per cent carbon dixoide caused no significant increase in systolic or mean arterial pressure, nor of heart rate. In 2 of these patients, previous laryngoscopy had produced a marked hypertensive response.

DISCUSSION

Our previous communication (Prys-Roberts, Meloche and Foëx, 1971) concluded that untreated high arterial pressures constituted a serious risk to patients undergoing anaesthesia and surgery. This conclusion was based on our finding of a high incidence of evidence of myocardial ischaemia related to arterial hypotension during steady-state anaesthesia in both treated and untreated patients. The results of the present study serve to emphasize that hypertensive patients are as much if not more at risk during induction of anaesthesia, and during subsequent laryngoscopy and tracheal intubation, as they are during steady-state anaesthesia. It is difficult to compare the relative risk of hypotension with myocardial ischaemia during established anaesthesia, with the excessive swing from hypertension in the conscious state to sudden hypotension during induction, followed by excessive hypertension during and after laryngeal stimulation. It is clear that these responses are not consequent upon the drugs used to manage patients with high blood pressure, but are to some extent related to the initial blood pressure. Following our findings during steady-state anaesthesia, it was surprising to find that patients on anti-hypertensive therapy with well controlled blood pressures, were equally prone to develop a hypertensive response to

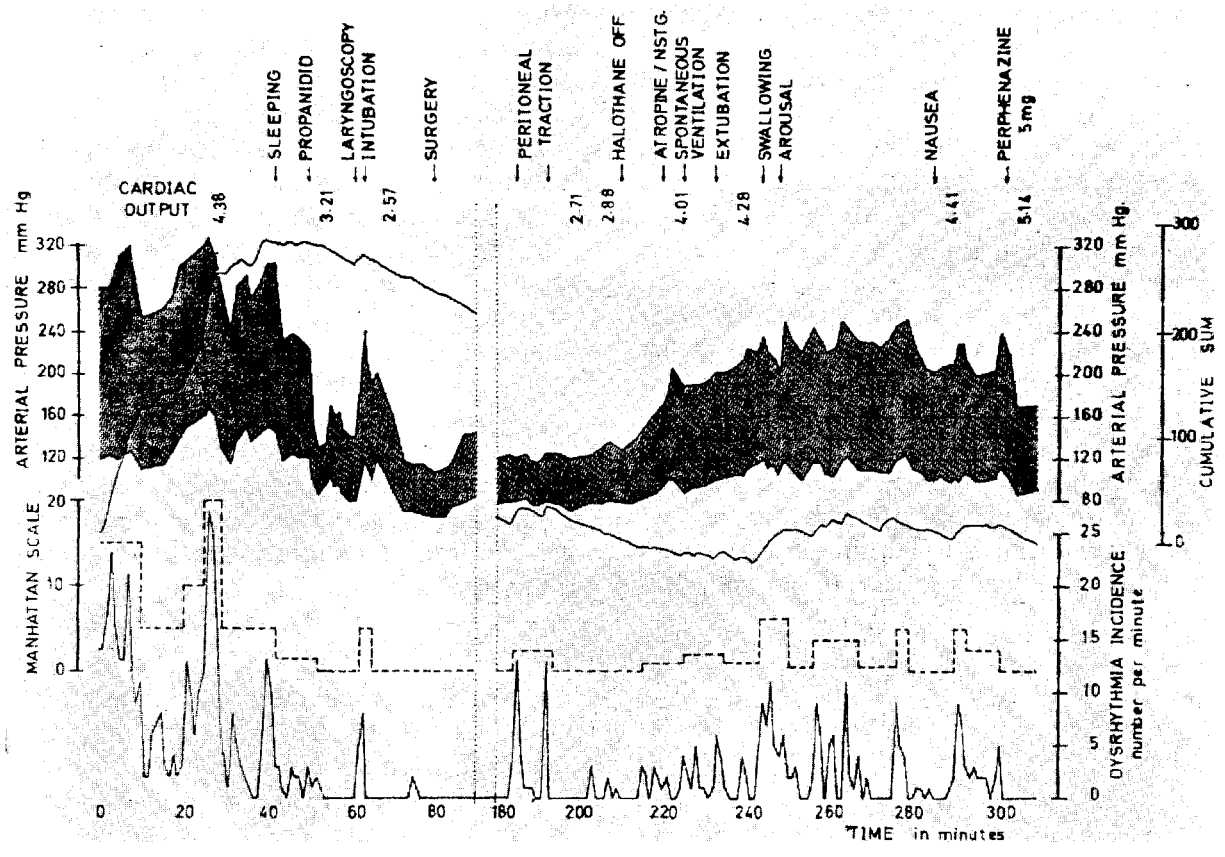

FIG. 4

Interrelation of arterial pressure and the incidence of cardiac dysrhythmia in patient No. 35. The lowest graph (continuous line) indicates the incidence of dysrhythmia, interpreted as the number of abnormal e.c.g. complexes per minute. The upper continuous line represents the cumulative sum (cusum) of this dysrhythmia incidence, and the intermediate dotted-line graph is the Manhattan diagram representing the changes in slope of the cusum graph, assessed at an arbitrary 5 per cent significance level. Assessment at the more critical 1 and 0.1 per cent significance levels showed changes to occur at the same points, but the "corners" of the Manhattan graph, representing the time of onset of a significant change in the cusum, were less obvious and of smaller magnitude.

During the pre-anaesthetic control period, the highest incidence of dysrhythmia was related to the highest arterial pressure levels which varied in a random manner, and were not associated with recognizable events. At the first event marked "sleeping" the patient fell into a natural sleep at the end of the control measurements of pulmonary gas exchange, and a significant reduction occurred in both the arterial pressure and the incidence of dysrhythmia. Although the patient could not be aroused by normal spoken command at this stage, anaesthesia was induced with propanidid in the usual way, and this event was associated with a further significant reduction of both arterial pressure and the incidence of dysrhythmia. Laryngoscopy and intubation caused a significant increase in both variables. During surgery, a 90-minute period of the record is not shown since no dysrhythmia occurred, which accounts for the progressive decline of the cusum value between the start and finish of the period. At the end of surgery, traction on the peritoneum of the pelvic floor, and of the peritoneal edges of the abdominal wall during closure, caused reflex increases in arterial pressure associated with the reappearance of dysrhythmia. Extubation, tracheal suction, and the period of arousal were associated with a progressive increase in arterial pressure and the incidence of dysrhythmia, with coincident peaks in both variables.

Cardiac output values are in l./min.

intubation, even though they were less likely to suffer drastic reduction of arterial pressure during induction.

Circulatory responses to laryngoscopy and tracheal intubation.

Reflex cardiovascular responses to mechanical stimulation of the upper respiratory tract have been described in cats (Tomori and Widdicombe 1969) and to the aspiration of secretions from the trachea in patients with normal cardiovascular reflexes (Corbett, Kerr and Prys-Roberts, 1969) and in patients with exaggerated cardiovascular reflexes due to autonomic overactivity (Corbett et al., 1969). The predominant response in man is a tachycardia and arterial hypertension, the latter being due to increased cardiac output rather than increased systemic vascular resistance, and is associated with a transient rise in the central venous pressure (Corbett et al., 1969). That these reflex responses are mediated by increased sympathetic nervous activity is based on circumstantial evidence in man, but is supported by the findings of Tomori and Widdicombe (1969) in the cat. They observed that mechanical stimulation of four areas of the upper respiratory tract, the nose, the epipharynx, the laryngopharynx and the tracheobronchial tree, induced reflex cardiovascular responses associated with enhanced neuronal activity in the cervical sympathetic efferent fibres. These cardiovascular responses, tachycardia and hypertension, and the enhanced neuronal activity were most pronounced during stimulation of the epipharynx, whereas those arising from stimulation of the tracheobronchial tree were least marked. In the present study, attempts were made to differentiate between the effects of laryngoscopy and those of tracheal intubation, and it was clear that the majority of the patients studied by us produced a reflex tachycardia and hypertension well before the act of intubation, though the effect was often enhanced by either spraying the larynx, or by intubation. Once the endotracheal tube was in position, and the laryngoscope withdrawn, the hypertension and tachycardia quickly subsided, but disturbing dysrhythmia tended to persist for up to 2 or 3 minutes. In the hypertensive patient, the extent of these cardiovascular reflexes is much more marked than in the normotensive patient.

Of the studies in normotensive man during anaesthesia, those of King and his colleagues (1951), Colon-Yordan, Mackrell and Stone (1953) and Wycoff (1960) were based on continuous records of intra-arterial pressure. Their findings were similar to ours, although the magnitude of the changes was much smaller. The study of King and associates included some patients who were hypertensive before intubation during maintenance anaesthesia with cyclopropane or diethyl ether, but they gave no indication as to whether these patients were known to be hypertensive before anaesthesia. During deep anaesthesia with cyclopropane or diethyl-ether, King and his colleagues found that the cardiovascular responses of these patients to laryngoscopy and intubation were effectively suppressed, while Colon - Yordan, Mackrell and Stone found a significant incidence (33 per cent) of ventricular dysrhythmia during intubation under deep cyclopropane-ether anaesthesia. Wycoff (1960) compared laryngoscopy and tracheal intubation under general anaesthesia with the same procedure under surface analgesia produced by cricothyroid block, and although the latter technique produced smaller changes in both blood pressure and heart rate, a sympathetic response was still present. Wycoff attributed this to soft tissue pressure by the laryngoscope (Macintosh), and also concluded that premedication with a belladonna derivative did not alter its incidence.

Takeshima, Noda and Higaki (1964) compared the effects of laryngoscopy with different laryngoscope blades, and concluded that the Macintosh blade which compressed the soft tissues of the anterior epipharynx produced a significantly greater hypertensive response than the straight-bladed Wis-Foregger laryngoscope. Simpler studies of young normotensive subjects (Takeshima, Noda and Higaki, 1964; Forbes and Dally, 1970) have established that the mean increase in arterial pressure due to laryngoscopy and intubation was of the order of 20–25 mm Hg, with maximum changes of about 40–45 mm Hg in a few subjects. In a group of normotensive patients with mitral stenosis or constrictive pericarditis, Dottori, Löf and Ygge (1970) observed significant hypertension in response to laryngoscopy and intubation following induction with nitrous oxide/oxygen/muscle relaxant (m.a.p. increased 40 per cent above conscious level), thio-

pentone/nitrous oxide/oxygen/relaxant (m.a.p. increased 57 per cent), and hexobarbitone/nitrous oxide/oxygen/relaxant (m.a.p. increased 18 per cent). Sinus tachycardia occurred in all patients, but was most marked in patients receiving thiopentone, and least marked following induction with nitrous oxide alone.

Hypertensive patients, whether treated or not, are prone to much greater changes in arterial pressure than normotensive patients of the same age. Dingle (1966) in a series of 19 hypertensive patients, found a mean increase in systolic arterial pressure of 35 mm Hg, with excessive increases in a few patients, but gave no details of the resting values, nor of the induction and maintenance techniques he used. The excessive swings of arterial pressure in some of our patients gave rise to considerable concern, particularly since they were accompanied by electrocardiographic evidence of prolonged ischaemia (fig. 2). Neuroleptanalgesia was used in the second group of patients in the hope that under its influence the hypertensive response to laryngoscopy would be attenuated, and that less circulatory depression would occur during maintenance anaesthesia. The lack of stability during laryngoscopy with this method was surprising in view of the cardiovascular stability which is commonly associated with NLA during surgery. However, the percentage increase in arterial pressure following NLA induction was less than with the other agents, and the different character of the arterial hypotension implies a lesser degree of myocardial depression and a decreased resistance to left ventricular ejection. These characteristics commend the technique for the induction of anaesthesia in hypertensive patients, but further measures to prevent the occurrence of severe hypertensive crises during intubation would seem to be necessary. Although the use of deep anaesthesia may suffice in the normotensive patient, our previous findings would strongly suggest that the associated hypotension and myocardial ischaemia would be undersirable in the hypertensive patient. It must be remembered, too, that in all our patients the hypertensive response to laryngoscopy and intubation occurred despite the prior establishment of a steady-state of maintenance anaesthesia with 1 per cent halothane.

Since the hypertensive response is due to increased activity in the cardiac sympathetic nerves (Tomori and Widdicombe, 1969) in the cat, and the pattern of their cardiovascular findings was almost identical with ours, it seems rational to block these responses by using a specific beta-adrenergic blocking agent. Propranolol has proved extremely effective in suppressing the hypotension, tachycardia and dysrhythmias associated with patients with sympathetic over-activity in severe tetanus (Prys-Roberts et al., 1969). In these patients, the exaggerated circulatory response to tracheal and nasopharyngeal suction of secretions was effectively suppressed by small doses of propranolol. The main disadvantage attending the intravenous use of propranolol is its quinidine-like effect of myocardial depression (Fitzgerald, 1970), although this has not been a problem in the management of patients with severe tetanus when smaller doses (0.1-0.2 mg) have been given incrementally. Propranolol and other beta-adrenergic blockers have been widely used and advocated for the management of hypertension (Gillam and Prichard, 1964; Richards, 1966; Frohlich, Tarazi and Dustan, 1969; Leishman et al., 1970), and of angina pectoris in hypertensive and normotensive subjects (Gillam and Prichard, 1965; Rabkin et al., 1966; Björntorp, 1967).

Choice of induction agent.

None of the agents used for induction of anaesthesia in this series was entirely satisfactory, although the NLA combination was less unsatisfactory than the others. Caution is required during the induction of NLA in hypertensive patients, however, and our experience with patient 21 (fig. 1) emphasizes the vascular instability of these patients, and the need for careful drug administration and continuous monitoring. We cannot agree with the recommendation of Lawin and associates (1966) that patients should be taken off antihypertensive and vasodilator drugs at least 4 days before induction of NLA. They reported three cases of cardiac arrest during induction of NLA in patients receiving vasodilator therapy. Cardiac arrest occurred within one circulation time of the rapid injection of large doses (10–22 mg) of droperidol, and although cardiac arrest was attributed to failure of cardiac filling as a result of excessive arteriolar dilatation, it is more likely to have been the result of a bolus of drug entering

the coronary circulation. In our patient the onset of heart block following severe sinus bradycardia occurred 3 minutes after the end of the injection, in a patient who immediately previously had an appearance time of less than 10 seconds for a dye-dilution curve.

Propanidid was particularly vicious in its hypotensive effect which was maximal during the first minute after its administration, and before maintenance anaesthesia had been commenced. Although the steady-state arterial pressures were no lower than those following other induction agents, in view of the sudden hypotension and the marked tachycardia we cannot recommend its use in the hypertensive patient.

Contrary to the established finding that methohexitone causes less arterial hypotension in normotensive subjects than thiopentone, when used in doses comparable to this present study (Dundee and Moore, 1961), we have found that methohexitone produced a significantly greater fall in systolic and mean arterial pressure in hypertensive patients, associated with a transiently higher heart rate during the first 1–3 minutes. Although we measured cardiac output in only one of the patients receiving methohexitone (in whom no change of systemic vascular resistance occurred), our findings were otherwise similar to those of Rowlands and associates (1967) in a series of patients with pre-existing cardiac disease, all of whom had supraventricular dysrhythmias beforehand. They concluded that the moderate hypotension which occurred in their patients was almost entirely the result of a fall in cardiac output and stroke volume. It has been widely established that thiopentone causes arterial hypotension as a result of reduced cardiac output (Fieldman, Ridley and Wood, 1955; Prys-Roberts, Kelman and Greenbaum, 1967), although in normotensive subjects the rate of injection may be a significant factor (Etsten and Li, 1955). We have also measured arterial pressures by sphygmomanometry in two hypertensive patients who were induced with 10 mg and 20 mg respectively of diazepam, and in view of the marked but transient hypotension (−40 mm Hg mean arterial pressure) and the excessive hypertension following laryngoscopy in both cases (+37 and +50 per cent of the awake control values of systolic arterial pressure) we did not proceed to more complete studies. We are unable to comment as yet on the possibilities of inhalational induction for hypertensive patients, though the use of cyclopropane or halothane/nitrous oxide must be considered.

APPENDIX

Cumulative Sum procedures (Cusums)

Cumulative sum procedures (Woodward and Goldsmith, 1964) may be used to detect alterations in mean levels between groups of values in a time series, and to identify the time of onset of such alterations. The procedures are performed by a digital computer which is programmed to read in a time series, and to calculate cumulative sums of the series using the grand mean of the series as a reference value. The computer also determines the occurrence of changes in the slope of the cusum/time relation, the assessment of which is based on Student's *t*-tests, nominally applied at the 0.1, 1 and 5 per cent significance levels.

This procedure is carried out by scanning the time series in a "forward" direction in such a way as to compare each cusum value with previous significant changes in the series, and to perform *t*-tests between groups of intervening cusum values. When the *t*-test values are not significant, the computer moves to the next cusum and repeats the process. When a significant change is detected, the computer is programmed to check that the change in slope is maintained beyond the test point, and that the onset of the change (the "corner") is identified on the time scale. This scanning procedure is repeated in the reverse direction, since certain real step changes may only appear to be significant in one direction. The results of both forward and reverse scanning are then amalgamated, and the computer is programmed to print out the results in graphical form (viz. fig. 4). The Manhattan diagram, although resembling a histogram in appearance, actually represents the "corners", or points where the slope of the cusum/time relation changes significantly.

Previous applications of this method of statistical analysis in the medical field have analyzed trend changes in the mean arterial pressure in patients in whom the systolic and diastolic pressures have been varying in a random fashion around a mean level (Corbett, 1968): for example, the labile hypertension of patients with severe tetanus.

The present application enabled us to determine whether changes in the incidence of dysrhythmia as a result of induction, laryngoscopy or intubation were statistically significant events, or whether they represented random changes in the background dysrhythmia level which were due to chance.

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ETUDES DE L'ANESTHESIE PAR RAPPORT A L'HYPERTENSION

II: CONSEQUENCES HEMODYNAMIQUES DE L'INDUCTION ET DE L'INTUBATION ENDOTRACHEALE

SOMMAIRE

Les réactions électrocardiographiques et hémodynamiques à l'induction d'anesthésie, suivie de laryngoscopie et intubation endotrachéale ont été étudiées chez un groupe de seize patients hypertendus non-traités, ainsi que chez un groupe de vingt patients sous traitement antihypertenseur jusqu'au jour de l'opération inclus. Les effects de cinq agents d'induction différents, thiopentone, methohexitone, propanidid, diazepam et la neuroleptanalgésie par une association de phenoperidol et droperidol ont été comparés. La neuroleptanalgésie causa moins d'hypotension artérielle que les autres agents, mais ne procura qu'une protection marginalement meilleure contre l'hypertension, la tachycardie et la dysrhythmie, associés à la laryngoscopie et l'intubation trachéale. Propanidid et diazepam causèrent une hypotension dramatique mais passagère chez un petit nombre de patients et leur étude ne fut plus poursuivie. Methohexitone, contrairement à ses effects chez des sujets normotendus, causa chez les patients hypertendus une plus forte hypotension que thiopentone. Les auteurs donnent la rationale du bloquage prophylactique des récepteurs beta-adrénergiques pour prévenir les crises hypertensives au cours de la laryngoscopie et l'intubation, aussi bien chez les hypertendus traités que non-traités.

UNTERSUCHUNGEN ZUR FRAGE ANAESTHESIE UND HYPERTENSION

II: HÄMODYNAMISCHE FOLGEERSCHEINUNGEN VON NARKOSE-EINLEITUNG UND ENDOTRACHEALER INTUBATION

ZUSAMMENFASSUNG

Elektrokardiographische und hämodynamische Reaktionen unter der 1rkoseeinleitung mit anschließender Laryngoskopie und endotrachealer Intubation wurden bei einer Gruppe von 16 unbehandelten Patienten mit erhöhtem Blutdruk und einer Gruppe von 20 Patienten unter einer antihypertensiven Therapie vor der Operation bis einschließlich dem Operationstag untersucht. Der Einfluß von 5 verschiedenen Medikamenten für die Narkoseeinleitung—Thiopenton, Methohexitan, Propanidid, Diazepam, sowie von Neuroleptanalgesie durch Kombination von Phenoperidin und Droperidol — wurde verglichen. Die Neuroleptanalgesie verursachte eine geringen arterielle Hypotesion als alle anderen Mittel, bot jedoch nur einen wenig besseren Schutz gegen Hypertension, Tachykardie und Dysrhythmie bei der Laryngoskopie und Trachealintubation als die anderen Mittel. Sowohl Propanidid als Diazepam verursachten eine dramatische, jedoch transitorische Hypotension bei einer kleinen Zahl von Patienten und wurden nicht weiter untersucht. Im Gegensatz zu ihren Wirkungen auf Personen mit normalen Blutdruck war die Blutdrucksenkung bei hypertensiven Patienten bei Methohexitan stärker als bei Thiopenton. Die theoretische Grundlage für eine prophylaktische Blockade der Beta-adrenergischen Rezeptoren zur Vermeidung von hypertensiven Krisen während der Laryngoskopie und Intubation bei behandelten und unbehandelten Hochdruckpatienten wird dargelegt.

ESTUDIOS SOBRE LA ANESTESIA EN RELACION CON LA HIPERTENSION

II: CONSECUENCIAS HEMODINAMICAS DE LA INDUCCION E INTUBACION ENDOTRAQUEAL

RESUMEN

Las respuestas electrocardiográficas y hemodinámicas a la inducción de la anestesia seguida por laringoscopia e intubación endotraqueal han sido estudiadas en un grupo de dieciséis pacientes hipertensos sin tratamiento y en un grupo de veinte pacientes bajo terapia antihipertensora hasta e incluyendo el día de la operación. Fue comparada la influencia de cinco agentes de inducción diferentes, tiopentona, metohexitona, propanidid, diacepam, y neuroleptanalgesia inducida por una combinación de fenoperidina y droperidol. La neuroleptanalgesia causó menos hipotensión arterial que cualquiera de los otros agentes, pero dio una protección sólo ligeramente superior a la de los otros agentes contra hipertensión, taquicardia y disritmia asociadas con la laringoscopia e intubación traqueal. El propanidid y diacepam causaron hipotensión dramática pero transitoria en un pequeño número de pacientes y no fueron investigados más. En oposición a sus efectos en sujetos normotensos, a methohexitona causó mayor hipotensión que la tiopentona en pacientes hipertensos. Se explica el fundamento para un bloqueo profiláctico de los receptores beta-adrenérgicos para impedir crisis hipertensivas durante la laringoscopia e intubación en pacientes hipertensos con y sin tratamiento.

NOTICE

It is proposed to establish a Zambian Society of Anaesthetists with the ultimate object of forming a Regional Society. The inaugural meeting took place on May 29, 1971, in the Medical School of the University of Zambia at Lusaka. Any anaesthetist in Central or East Africa who is interested in supporting this venture should contact Dr F. F. Casale, F.F.A.R.C.S., University Teaching Hospital, Private Bag R. W. 1, Lusaka, Zambia, for further information.