EFFECTS OF INTRAVENOUSLY ADMINISTERED SUCCINYLDICHOLINE ON CARDIAC RATE, RHYTHM, AND ARTERIAL BLOOD PRESSURE IN ANESTHETIZED MAN

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ALTERATIONS in blood pressure and cardiac rate following injection of succinyldicholine in man have been noted frequently,[1-6] but the response has not been systematically analyzed. In this paper the influence of various general anesthetic agents, of various doses of the relaxant, of repeated injections, of raised airway pressure, of autonomic blockade, of decamethonium, of succinylmonocholine, and of raised arterial pressure on circulatory responses to succinyldicholine will be reported. Observations on plasma potassium concentration will also be presented.

METHODS

Thirty-four patients ranging in age from 13 to 63 years were studied. All but 6 were female. Diagnoses of early cirrhosis of the liver and grade II essential hypertension, respectively, were made in patients 10 and 14. The others were considered physically normal. With the exception of 2 patients, all measurements were made prior to elective surgical operations. Patients 3 and 9 were studied during minor operations.

Preanesthetic medication and anesthetic agents used in 27 of these patients are listed in table 1. The 7 subjects not included in the table received either succinylmonocholine or decamethonium before the first dose of succinyldicholine. Three of these subjects received no preanesthetic medication, the remainder secobarbital 100 mg. and atropine or scopolamine 0.4 mg. All were anesthetized with cyclopropane.

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Cyclopropane and halothane were administered using a closed circle absorber system, the latter via a "copper kettle." Thiopental 2.5 per cent solution was given by intermittent intravenous injection in 2 subjects, and in 0.5 per cent concentration as a continuous intravenous drip in the others. Nitrous oxide-oxygen was given in 5/2 liter flow rates with a semiclosed circle system. Trichlorethylene 0.7 per cent and nitrous oxide and oxygen were administered in a nonrebreathing system. Ether was administered via an E.M.O. vaporizer with a nonrebreathing valve and bellows attachment.

Concentrations of ether in arterial blood were estimated using the method of Price and Price.[7] End-tidal air was collected in a syringe and analyzed for cyclopropane as described by Linde and Price.[8]

In 14 subjects end-expired P_{CO_2} was measured continuously with a Liston-Becker infrared carbon dioxide analyzer. In these subjects P_{CO_2} was maintained below 45 mm. of mercury except as noted in table 1. In the others, it is believed that P_{CO_2} also was maintained at or below normal levels, since assisted or controlled respirations were used whenever voluntary efforts appeared inadequate.

Continuous ECG (lead 2) monitoring was carried out during the entire procedure. Heart rate was measured from the ECG, either by counting for a 15 second interval, or by measuring the longest R-R interval following the injection.

Arterial blood pressure was measured by a strain gauge through an indwelling needle inserted in a brachial or femoral artery in most subjects. The output of the carbon dioxide analyzer, strain gauge manometer and electrocardiogram were recorded on a polygraph.

Succinyldicholine, 2 per cent solution, was used for injection. A one per cent succinyl-

monocholine solution was prepared from crystals and sterilized in an autoclave at two pounds pressure for thirty minutes. Compensation for hydrolysis was attained by adding 10 per cent to the desired final dose before sterilization.

Succinyldicholine and succinylmonocholine were injected in single doses through a continuous infusion of 5 per cent dextrose in water. When the thiopental drip was placed in the same tubing, the needle was flushed prior to the injection of other materials.

Trimethaphan (0.1 per cent solution) was administered by continuous intravenous drip to produce ganglionic blockade. Atropine sulfate was given by intermittent injection.

Neo-synephrine (0.002 per cent) was infused at a rate sufficient to increase mean arterial pressure approximately 20 mm. of mercury in subjects 33 and 34, who were anesthetized with cyclopropane. Initial doses of 40 mg. of succinyldicholine and subsequent doses of 40 or 20 mg. were then administered intravenously.

Decamethonium in doses of 2.5 to 5.0 mg. was administered to subjects 24, 26, 27, 30, and 31 prior to the initial dose of succinyldicholine. Subject 32 received 2 mg. of decamethonium after having received eight doses of succinyldicholine, and 3 mg. of decamethonium after receiving a ninth dose of succinyldicholine.

Arterial blood samples were taken before and after administration of succinyldicholine and serum potassium was determined in an internal standard flame photometer in 4 patients.

Results

The principal results are summarized in table 1, where they are grouped according to the anesthetic administered.

Influence of Anesthetic Agents

Thiopental. The 6 subjects anesthetized with thiopental each received two or more injections of succinyldicholine. The first dose was followed by a slight to moderate (5–25 mm. of mercury) increase in systolic and diastolic pressure in three cases, and by insignificant changes in pressure (less than 5 mm. of mercury) in the others. Heart rate increased moderately (about 20 beats/minute) in 3 and little in the remainder of the subjects. Cardiac rhythm was unaltered. Following the second injection arterial pressure increased in 4 instances and was unchanged in one. Heart rate behaved inconsistently; the extreme changes were a decrease of 10 beats per minute in subject 12 and an increase of 20 per minute in 15. Cardiac rhythm was unaltered in 4 individuals, but bigeminal rhythm occurred in one and persisted for three minutes. A statistically significant difference between responses to the first and second injections of succinyldicholine could not be shown.

Ether. In the 3 subjects anesthetized with diethyl ether the first injection of succinyldicholine was followed by minor and inconsistent changes in heart rate and arterial pressure. Cardiac rhythm was unaltered. Following the second injection arterial pressure increased transiently in two and was unchanged in the third case. Heart rate was inconsistently affected and cardiac rhythm remained normal.

Halothane. Three subjects received halothane. In each instance the first injection of succinyldicholine was followed by moderate hypertension and tachycardia which developed rapidly and reached maximal levels within 1–4 minutes. In contrast, a second injection of succinyldicholine given five minutes later was followed by cardiac slowing in 2 cases and asystole for 7 seconds in the third. Nodal rhythm occurred in two instances and bigeminal rhythm in the other. Changes in arterial pressure during the period of bradycardia were slight, except in the subject who developed asystole. Following these initial changes, cardiac rate and rhythm returned toward normal, while arterial pressure rose rapidly and transiently exceeded normal by 10–20 mm. of mercury.

Cyclopropane. In 8 of 14 subjects given cyclopropane, the first dose of succinyldicholine was followed within 45 seconds by slight cardiac slowing. Cardiac rhythm remained normal. Arterial pressure was inconsistently affected during this interval but immediately after this heart rate increased in all and arterial pressure increased in 11 of 12 subjects. Maximum levels were attained 3 to 5 minutes following the first injection.

A second injection, given 4 to 10 minutes after the first, was followed within thirty sec-

TABLE 1

Effects Produced by Succinyldicholine on Arterial Pressure, Heart Rate and Rhythm During Anesthesia*

Subject No. (Age-Years)	Preanes. Med. (mg.)	Anes. Conc.	Control 1		Dose 1 (mg.)	After Injection						Minutes Between Doses	Control 2		Dose 2 (mg.)	After Injection						Comments
						Lowest			Highest							Lowest			Highest			
			AP	HR		AP	HR	T	AP	HR	T		AP	HR		AP	HR	T	AP	HR	T	
Thiopental																						
12 (42)	Scopol. 0.4 Secobarb. 100	—	102/74	90	200	106/82	85	33	112/85	90	120	2	106/82	90	200	104/76	80	100	106/78	80	120	N_2O-O_2. Thiopental intermittent inj. (total 600 mg). 7 minutes.
13 (27)	Atropine 0.4 Secobarb. 100	—	85/48	96	40	83/48	96	17	87/50	104	120	8	82/46	92	40	82/48	92	15	102/68	92	60	As above. Total dose of thiopental 700 mg. Very light anesthesia. 59 minutes.
14 (49)	None	—	195/100	100	40	208/115	100	30	225/125	120	300	6	224/120	119	40	222/120	115	30	230/135	115	300	N_2O-O_2. Thiopental by drip (0.5%). Moderately deep anesthesia. 20 minutes
15 (34)	None	—	92/62	86	40	92/60	90	20	100/75	105	60	5	85/55	85	40	80/50	90	15	148/85	105	120	As above. Total thiopental 1.5 gm. Moderately deep anes. 78 minutes. Bigeminy in 45 seconds for 3 minutes following second injection.
16 (32)	Atropine 0.4 Secobarb. 100	—	94/75	95	40	88/70	95	30	92/77	105	60	6	97/77	96	40	96/75	95	20	103/80	110	300	As above. Total thiopental 1.6 gm. Moderately deep anes. 104 minutes.
22 (68)	Chloral Hydrate 500 Atropine 0.4	—	—	83	50	—	86	45	—	94	90	5	—	79	10	—	81	60	—	86	90	N_2O-O_2 intermittent thiopental (425 mg.) 18 minutes.
23 (22)	Scopol. 0.4	—	120/70	66	40	—	65	20	—	70	48	5	130/90	62	10	—	60	33	—	70	300	N_2O-O_2 0.7%. Trichloroethylene intermittent thiopental (325 mg.) 58 minutes. Nodal rhythm.
Ether																						
9 (56)	Atropine 0.4 Secobarb. 100	—	125/85	120	40	120/70	120	15	106/82	120	120	7	115/78	110	40	125/65	120	15	138/82	120	180	
10 (34)	None	95	105/72	100	40	103/66	100	15	105/72	112	60	5	103/65	112	40	107/58	80	20	147/85	108	180	Slowing in 15 seconds.
11 (34)	None	114	97/43	88	40	95/43	88	30	105/50	96	300	5	105/50	96	40	103/50	96	30	107/53	96	180	

* Abbreviations: Anes. Conc. = concentration of anesthetic agents: cyclopropane, volumes per cent end-expired; halothane, volume per cent inspired; ether, mg. per cent in arterial blood. AP = arterial pressure, mm. of mercury. HR = heart rate, beats/minute. T = time, seconds.

TABLE 1—*Continued*

Subject No. (Age-Years)	Preanes. Med. (mg.)	Anes. Conc.	Control 1		Dose 1 (mg.)	After Injection						Minutes Between Doses	Control 2		Dose 2 (mg.)	After Injection						Comments
						Lowest			Highest							Lowest			Highest			
			AP	HR		AP	HR	T	AP	HR	T		AP	HR		AP	HR	T	AP	HR	T	
Halothane																						
7 (32)	Atropine 0.4 Secobarb. 100	1%	85 / 62	60	40	98 / 72	60	15	102 / 77	100	60	7	100 / 67	65	40	102 / 67	60	20	126 / 84	85	180	Slowing in 16 seconds. Nodal rhythm in 105 seconds of 12 seconds duration.
8 (17)	Atropine 0.4 Secobarb. 100	1%	100 / 73	80	40	95 / 70	80	15	133 / 90	115	120	5	118 / 80	100	40	Asystole		15	130 / 82	84	300	Slowing in 11 seconds. 2 periods of asystole of 4.5 and 3 seconds. Nodal rhythm followed of 3 minutes duration.
18 (27)	None	1%	136 / 85	65	40	140 / 80	60	30	177 / 110	90	240	4	177 / 110	90	20	170 / 100	65	20	193 / 107	105	60	Slowing in 18 seconds. Rapid increase in heart rate followed. Bigeminy in 52 seconds, lasting 1 minute.
Cyclopropane																						
1 (47)	Atropine 0.5 Secobarb. 100	—	140 / 80	70	40	165 / 100	60	45	176 / 100	80	180	10	145 / 90	68	40	125 / 70	40	20	195 / 110	60	180	Slowing in 15 seconds. Nodal rhythm in 30 seconds, lasting 24 seconds.
2 (27)	Atropine 0.4 Secobarb. 100	12 to 21	126 / 62	94	40	133 / 63	84	45	137 / 74	100	300	5.5	125 / 62	95	10	103 / 38	60	15	147 / 73	92	180	Slowing in 15 seconds. Sinus depression lasting 26 seconds.
3 (63)	Atropine 0.4 Secobarb. 100	—	134 / 62	60	60	140 / 72	60	13	160 / 72	75	120	5.5	160 / 72	75	60	132 / 60	40	15	195 / 100	95	300	Slowing in 15 seconds. Nodal rhythm in 15 seconds, lasting 48 seconds.
4 (46)	None	13 to 18	153 / 75	60	40	137 / 73	50	45	190 / 98	64	180	7.5	174 / 87	60	40	Asystole		20	202 / 98	60	300	Slowing in 15 seconds. Nodal rhytym in 15 seconds, lasting 48 seconds.
5 (13)	None	3 to 26	132 / 76	68	40	126 / 72	64	15	124 / 82	72	240	6	142 / 80	70	40	Asystole		30	126 / 84	85	180	Slowing in 18 seconds. Nodal rhythm in 21 seconds. 2 periods of asystole of 5 and 6 seconds 3 seconds apart. Nodal rhythm followed of 45 seconds.
6 (40)	Atropine 0.4 Secobarb. 100	—	122 / 78	56	40	115 / 60	40	45	132 / 80	62	390	6.5	132 / 80	62	40	90 / 50	24	30	138 / 84	60	300	Slowing in 15 seconds. Nodal rhythm in 33 seconds of 30 seconds duration.
19 (30)	Atropine 0.4 Secobarb. 100	6 to 18	150 / 85	88	40	150 / 85	76	15	165 / 93	94	300	4	150 / 80	84	40	137 / 77	64	45	177 / 100	82	300	Slowing in 17 seconds. Nodal rhythm in 19 seconds with several irregular nodal escapes lasting 30 seconds.
21 (52)	Atropine 0.4	19	127 / 57	94	40	127 / 63	100	30	180 / 88	116	180	5	165 / 86	96	10	178 / 83	66	60	177 / 80	84	240	Slowing in 15 seconds. Nodal extrasystoles in 21 seconds of 75 seconds duration. P_{CO_2} 50–60 mm.

20 (28)	Scopol. 0.4	10	146 / 80	60	127 / 87	40	54	15	165 / 90	60	120	6	134 / 70	55	125 / 87	10	45	48	170 / 90	78	120	Slowing in 42 seconds.
25 (37)	None	14	135 / 75	80	127 / 77	40	80	40	135 / 80	90	300	5	127 / 78	80	125 / 72	10	56	40	140 / 85	93	120	Slowing in 30 seconds. P_{CO_2} 53 mm.
29 (38)	Morphine Atropine	—	—	70	—	40	79	25	—	89	45	5	—	83	—	10	79	30	—	—	—	Slowing in 30 seconds.
32 (21)	None	19	—	88	—	40	56	300	—	84	30	5	—	56	—	20	46	30	—	52	240	Slowing in 30 seconds. P_{CO_2} 54 mm.
33 (30)	None	15	157 / 85	56	120 / 80	40	64	60	160 / 90	86	45	5	158 / 93	64	133 / 78	20	27	75	175 / 110	60	240	Neo-synephrine infusion. Mean AP increased 22 mm. before dose *1*. Slowing in 70 seconds.
34 (26)	None	21	98 / 53	68	80 / 48	40	68	70	128 / 70	82	45	10	93 / 53	68	63 / 33	40	48	60	125 / 65	72	180	Neo-synephrine infusion. Mean AP increased 20 mm. before dose *1*. Slowing in 45 seconds. Nodal extrasystoles in 45 seconds lasting 50 seconds.

Cyclopropane

onds by dramatic changes. Heart rate and arterial pressure were markedly reduced in every case. Subjects 4 and 5 developed asystole. In subject 4 with an asystole of 30 seconds duration, the EGC tracing showed P waves occurring at 3–4 second intervals during this period, but the first ventricular complex following arrest was not immediately preceded by a P wave and presumably was nodal in origin. Nodal rhythm persisted for ninety seconds before cardiac rhythm returned to normal. Nodal extrasystoles or nodal rhythm occurred in 8 of the remaining cases.

These changes were rapidly reversed. Cardiac rate and rhythm returned to normal within 3-5 minutes following the injection, at which time arterial pressure exceeded the control level in most cases. Hypertension persisted for various periods ranging from 5 to 15 minutes; tachycardia was more brief in duration.

Trichlorethylene, Thiopental and Nitrous Oxide. Bradycardia was not observed after any of three successive doses of 10 mg. succinyldicholine after the initial dose in subject 23, who received trichloroethylene, thiopental, nitrous oxide and oxygen. Succinyldicholine in larger doses reduced heart rates 10 to 23 beats per minute in this patient.

Influence of Interval Between Injections and Numbers of Injections

Conditions necessary to elicit bradycardia following succinyldicholine injection were further studied in subjects anesthetized with halothane or cyclopropane. It was found (subjects 19 and 20) that bradycardia followed each of eight consecutive 10 or 40 mg. doses of succinyldicholine given 5 minutes apart. The most pronounced effect, however, occurred after the second injection. In subjects 6 and 18 the time interval between injections was decreased until bradycardia no longer occurred. When injections were given 1½ to 2 minutes apart bradycardia did not occur, although tachycardia and hypertension were sometimes noted. A minimum of three minutes between injections was generally necessary for the production of bradycardia. In a single case bradycardia was not elicited when the interval between injections was thirty minutes.

Effective Dose. Doses of succinyldicholine as small as 10 mg. elicited marked bradycardia on a third or fourth injection, provided they were not given sooner than three minutes following the previous injection. Yet initial injections as large as 60 mg. often did not produce bradycardia.

Effect of Atropine. Three subjects (1, 3, 18) anesthetized with cyclopropane or halothane who had manifested bradycardia after succinyldicholine injection were subsequently given atropine sulfate intravenously in doses ranging from 0.4 to 0.6 mg. Five to ten minutes after the administration of atropine, neither bradycardia nor nodal rhythm could be elicited by injection of succinyldicholine. Instead, heart rate was unaltered or increased. Bigeminal rhythm followed the injection of relaxant in two cases (subjects 1 and 18) and arterial hypertension in all. Atropine and scopolamine given intramuscularly as preanesthetic medication did not block bradycardia produced by successive doses of succinyldicholine (table 1).

Effects of Ganglionic Blockade. One subject (18) who received halothane and two given cyclopropane (21 and 22) were given trimethaphan (0.1 per cent) by continuous intravenous drip until sympathetic blockade was considered complete (judged from absence of arterial pressure "rebound" following release of positive airway pressure [9]). Neither tachycardia nor bradycardia occurred in response to succinyldicholine in any subject during blockade, nor were there changes in arterial pressure attributable to succinyldicholine injection. As the effects of trimethaphan were wearing off, tachycardia and hypertension, but not bradycardia, were observed following injection. When they were completely worn off, bradycardia could again be elicited.

Succinylmonocholine. One subject (17) anesthetized with cyclopropane was given 40 mg. succinylmonocholine intravenously, followed one and a half minutes after by 40 mg. succinyldicholine. No change in heart rate or arterial pressure followed the first injection, while after the second both increased. Six minutes after the second injection another 40 mg. dose of succinyldicholine was given. This was followed promptly by a reduction in heart rate from 85 to 59 beats per minute. Five minutes later an additional 40 mg. succinylmonocholine were injected without obvious effect; six minutes after this, administration of 20 mg. succinyldicholine resulted in bradycardia, the cardiac rate slowing from 85 to 65 per minute.

Serum Potassium. Serum potassium changes following succinyldicholine were inconsistent. No significant changes were observed with initial doses of 40 or 50 mg. of succinyldicholine. Subsequent doses of 10 mg. resulted in an increase of 0.55 mEq./liter in two subjects and a decrease of 0.07 mEq./liter in a third subject, with the second dose, and decreases of 0.7 and 0.2 mEq./liter with the third dose. An increase of 0.15 mEq./liter was observed with a fourth dose in one patient. There was no correlation between bradycardia and change in serum potassium.

Phenylephrine. Increasing mean arterial pressure 22 and 20 mm. of mercury by intravenous infusion of Neo-synephrine (subjects 33 and 34) had no effect on the cardiovascular response to succinyldicholine. Initial doses of 40 mg. of succinyldicholine produced increases in heart rate in both subjects; subsequent doses of 20 and 40 mg. of succinyldicholine resulted in reductions of heart rate ranging from 20 to 34 beats per minute.

Raised Airway Pressure. Positive pressure ventilation did not affect the bradycardia observed with succinyldicholine. In subject 7, positive pressure was maintained from the moment succinyldicholine was adminstered until after the bradycardia had disappeared. No lessening of the degree of bradycardia or incidence of nodal rhythm was observed. Also the majority of the cyclopropane group was maintained with intermittent positive pressure ventilation throughout the entire study. These observations suggest that positive pressure lung ventilation following the administration of succinyldicholine was not the cause of bradycardia. Neither do they support the suggestion that lung inflation will reverse bradycardia occurring after succinyldicholine administration.

Other Relaxants. Four of the 5 patients breathing cyclopropane who received decamethonium prior to succinyldicholine showed a reduction in heart rate ranging from 3 to 28 beats per minute with initial doses of 30 mg.

of succinyldicholine. Four of the 5 showed decreases in heart rate with subsequent doses of succinyldicholine, although the responses were not as consistent as in the group of patients given cylclopropane who had not received decamethonium. One patient was given 2 mg. of decamethonium after exhibiting bradycardia following each of seven doses of succinyldicholine. Subsequent administration of 20 mg. of succinyldicholine resulted in a reduction in heart rate from 60 to 45 beats per minute. Twenty mg. of succinyldicholine following a second dose of 3 mg. of decamethonium failed to reduce the heart rate of this patient. However, 40 mg. of succinyldicholine five minutes after this dose reduced the heart rate from 64 to 48 beats per minute.

Effect of Fasciculations. The presence of fasciculations bore no apparent relation to the bradycardia produced by subsequent doses of succinyldicholine. Fasciculations were usually observed following the first and sometimes the second and third doses of succinyldicholine. None were seen following other doses, yet bradycardia was observed in subjects 19 and 20 after each of eight successive doses of the relaxant.

Discussion

Succinyldicholine, administered in doses similar to those used in this study, is a drug commonly used to produce muscle relaxation for tracheal intubation. When there is difficulty with intubation successive doses may be administered. Under these circumstances cardiac arrest might occur in patients receiving cyclopropane or halothane. Instances of cardiac arrest requiring resuscitation have not, however, been reported to our knowledge, although we have shown that the usual doses of atropine and scoplamine given for preanesthetic medication do not prevent the bradycardia caused by successive doses of succinyldicholine.

The degree of hypotension produced was usually related to the severity of the bradycardia. The latter was inversely related to the length of time between injections and directly with the amount of drug administered. Our studies suggest that a minimum time must elapse between injections of succinyldicholine for the production of bradycardia. In subject 6 bradycardia could only be produced if the drug was administered at a time interval greater than two minutes. This may be related to the duration of sympathetic action. As long as this is present the profound vagal effects cannot be produced.

The influence of the speed of injection of succinyldicholine was not studied. However, since Craythorne[4] was unable to produce bradycardia with intramuscular injections in children, we might assume the rate of injection to be a factor. We do not have sufficient data to state whether greater depth of anesthesia affords any protection against bradycardia.

The ability of trimethaphan to suppress circulatory responses to succinyldicholine indicates that they are mediated via sympathetic and parasympathetic efferent nerves, and are not the result of direct actions of succinydicholine on vascular smooth muscle or on the heart. According to Beretervide,[10] succinyldicholine competes with acetylcholine for true cholinesterase, with the result that newly synthesized acetylcholine accumulates instead of being destroyed by the enzyme. He concluded that bradycardia and hypotension following administration of succinyldicholine resulted from central nervous parasympathetic actions, while hypertension and tachycardia were caused by stimulation of sympathetic ganglia.

Hypertension and tachycardia, occurring after the period of cardiac slowing, are independent of the earlier parasympathetic effect as indicated by our experiments with trimethaphan, atropine, thiopental and ether.

This distinct and separate response which follows the initial bradycardia may be the result of slower penetration of the drug into sympathetic ganglia than into the central nervous system. Its eventual subsidence may be caused both by destruction of the drug and by "buffering out" as the result of barostatic reflexes. Similar findings were reported by Craythorne in children.[4]

The most important unanswered question posed by our studies is why bradycardia was so much more marked following later doses of succinyldicholine than it was after the first one. We regard our findings with succinylmonocholine as indicating that accumulation of this metabolite of succinyldicholine is not responsible for the effect. The results of

plasma potassium analyses are difficult to evaluate since plasma values may not reflect physiologically significant changes on organs. In any event plasma values were inconsistent and quantitatively unimpressive. Moreover our experience, both with succinyldicholine and with decamethonium, indicates that the occurrence of muscular fasciulation (which is presumably the major source of increased potassium concentration in plasma) is not essential for the occurrence of conspicuous bradycardia following succeeding injections of succinyldicholine. It is also unlikely that bradycardia caused by later succinyldicholine injections is caused by the enhancement of reflexly increased vagal activity following the first dose, and resulting in turn from a barostatic response to arterial hypertension caused by the first dose. The experiments with Neosynephrine indicate this, for bradycardia was not observed following administration of the first dose of succinyldicholine in either of the subjects whose mean arterial pressure was maintained 20 mm. of mercury above normal by infusion of the pressor drug. Observations following repeated doses of succinyldicholine and decamethonium make it unlikely that the circulatory responses depend upon actions, including fasciculation, exerted at the myoneural junction. Finally, we did not find any effect of lung inflation on the response. The cause of the enhanced effect of doses subsequent to the first one therefore remains unknown to us.

Summary

Bradycardia and prolonged asystole have been produced by the administration of successive doses of succinyldicholine in patients receiving cyclopropane or halothane, but not if thiopental or ether were being used. The effect was most marked if an interval of two to five minutes separated the doses of succinyldicholine. It could be blocked by atropine.

Hypertension and tachycardia following injections of succinyldicholine occurred in patients anesthetized with any of the anesthetics studied. This effect was independent of the parasympathomimetic actions of the relaxant; it probably resulted from sympathetic nervous stimulation.

It is probable that succinyldicholine therefore stimulates both the parasympathetic and the sympathetic nervous systems.

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