EFFECT OF INTRAVENOUSLY ADMINISTERED SUCCINYLCHOLINE UPON CARDIAC RATE AND RHYTHM

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CHANGES of cardiac rate and rhythm following intravenously administered succinylcholine chloride have been reported.[1-3] Bullough [8] states: "It is not generally known that the practice of using intermittent intravenous injection of the short acting relaxant suxamethonium (succinylcholine) during anesthesia is potentially dangerous. The phenomenon of intermittent intravenous injection is not to be confused with the bradycardia and/or hypertension which may follow a single injection . . . the former being common in children."

Leigh and associates,[6] reported a series of 23 patients studied by electrocardiographic tracings. Detailed results were presented from the records of four of them. There were three showing bradycardia (A-V rhythm) and one in whom no change of rate occurred. It was stated, "The four cases described are representative of the series." The implication was that bradycardia was the usual result in the pediatric group.

In this study the cardiac effect of a single dose of succinylcholine, given intravenously, is evaluated. The patients were of diverse physical status and in whom the only prophylactic or therapeutic treatment for arrhythmias was the use of oxygen.

METHOD

Succinylcholine was administered on 30 occasions to patients ranging in age from four weeks to 16 years. They were anesthetized with either ether or thiobarbiturate followed by nitrous oxide and oxygen. The patients for this study were selected at random. Infants who were to undergo angiocardiography were given succinylcholine as the only agent. The premedication generally used was either atropine or scopolamine, sometimes in combination with an opiate. In some patients the preanesthetic medication was omitted intentionally.

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The amount of succinylcholine given ranged between 0.5 mg. to 2 mg. per pound of body weight. In almost all of the cases, immediately before the administration of the relaxant or simultaneously with it, 100 per cent oxygen was carefully administered with bag and mask. Oxygen was continued for two minutes or more following the administration of succinylcholine. The primary consideration was to provide the patient with good pulmonary ventilation. In all cases electrocardiographic tracings were obtained either with the Sanborn or the Gilson apparatus.

RESULTS

The results obtained are shown in table 1. It can be observed that the predominant response was sinus tachycardia, demonstrated in 21 of the 30 administrations. In six, no changes were observed either in pulse rate or rhythm. In three of the patients the presence of A-V rhythm with slowing and irregularity of the pulse was noted. In one of these the effect was marked.

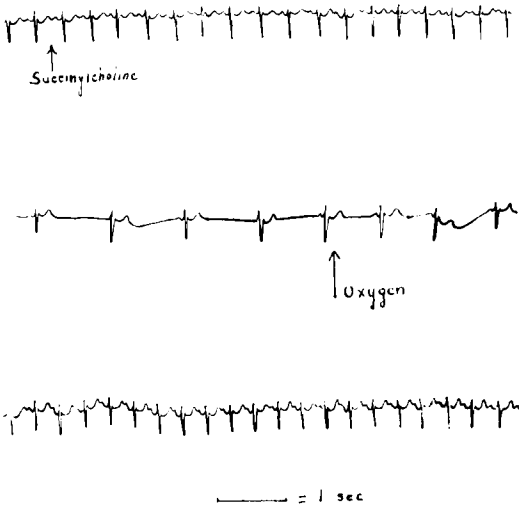

FIG. 1. Bradycardia and irregularity with occasional A-V rhythm occurring following administration of succinylcholine. Returned to normal rate and rhythm after 100 per cent O_2 was administered.

TABLE 1

CARDIAC EFFECTS FOLLOWING THE INTRAVENOUS INJECTION OF SUCCINYLCHOLINE

Pt. No.	Age	Weight, Lbs.	Succinyl-Choline, mg.	Before Drug Administration		After Drug Administration		Premedication, mg.
				Rhythm	Rate, Min.	Rhythm	Rate, Min.	
1	18 mo.	16	32	NSR*	132	NSR	160	None
2	10 mo.	20	30	NSR	150	NSR	150	None
3	5 mo.	11½	20	NSR	130	NSR	140	None
4	14 mo.	20	30	NSR	110	NSR	130	None
5	4 yr.	26	30	NSR	110	NSR	130	None
6	13 mo.	25	25	NSR	130	NSR	150	None
7	14 mo.	23	36	NSR	140	NSR	140	None
8	10 mo.	21	34	NSR	140	NSR	140	None
9	6 yr.	48	40	NSR	110	NSR	140	None
10	10 mo.	21	20	NSR	140	NSR	160	Atropine, 0.1
11	5 mo.	16	24	NSR	150	NSR	170	Atropine, 0.2
12	2.8 yr.	31	40	NSR	100	NSR	120	Atropine, 0.2
13	15 mo.	21	32	NSR	140	NSR	140	Atropine, 0.2
14	4 wk.	8	6	NSR	160	AVR**	60	Atropine, 0.2
15	9 mo.	18	20	NSR	140	NSR	150	Atropine, 0.2
16	7 yr.	38	40	NSR	90	NSR	110	Atropine, 0.4
17	10 mo.	18	20	NSR	160	NSR	180	Atropine, 0.3
18	9 yr.	58	50	NSR	90	NSR	100	Scopola., 0.3
19	16 yr.	127	100	NSR	100	NSR	120	Scopola., 0.4
20	3 yr.	35	40	NSR	150	NSR	160	Morph., 1; Atrop., 0.3
21	15 yr.	134	80	NSR	120	NSR	140	Morph., 10; Scop., 0.4
22	9 mo.	21	30	NSR	130	AVR	10	Morph., 2; Scop., 0.2
23	15 yr.	125	100	NSR	100	NSR	110	Pant., 20; Scop., 0.4 Pentobarb., 50
24	3.9 yr.	35	40	NSR	110	NSR	140	Meper., 25; Scop., 0.2
25	3 yr.	36	34	NSR	100	NSR	100	Meper., 30; Atrop., 0.3
26	2 yr.	26	40	NSR	130	NSR	150	Meper., 25; Atrop., 0.3
27	3 yr.	33	40	NSR	110	NSR	130	Meper., 25; Atrop., 0.3
28	16 yr.	130	60	NSR	110	NSR	110	Meper., 75; Scop., 0.3
29	11 yr.	68	50	NSR	70	NSR	90	Morph., 6; Scop., 0.15
30	5 yr.	40	20	NSR	98	AVR	50	Morph., 4; Scop., 0.2

* NSR = Normal Sinus Rhythm.
** AVR = A-V Nodal Rhythm.

Patient 14 (fig. 1), a four-week old infant weighing eight pounds who was to have an angiocardiogram, was given 0.2 mg. of atropine as preanesthetic medication. Six mg. of succinylcholine was given intravenously and 100 per cent oxygen was administered by bag and mask 18 seconds later. The electrocardiographic tracing shows a decrease of pulse rate with the presence of occasional periods of A-V rhythm. With the administration of oxygen under positive pressure, the pulse rate and rhythm returned to normal in six seconds, followed by a transient rise in pulse to 180.

Patient 22 (fig. 2), a nine-month-old infant weighing 21 pounds, was to undergo craniotomy for craniostenosis. Morphine 2 mg. and scopolamine 0.2 mg. were used as premedication. The induction of anesthesia was started with ten per cent thiopental rectally, 10 mg. per pound of body weight, and followed by nitrous oxide and oxygen. For tracheal intubation, 30 mg. of succinylcholine was administered intravenously. Even though oxygen was given by bag and mask, the patient's airway became obstructed by the tongue. A marked bradycardia and A-V rhythm appeared on the tracing and disappeared a few seconds after the insertion of an airway and adequate ventilation of the lungs. When this same patient was placed in the prone position for the opera-

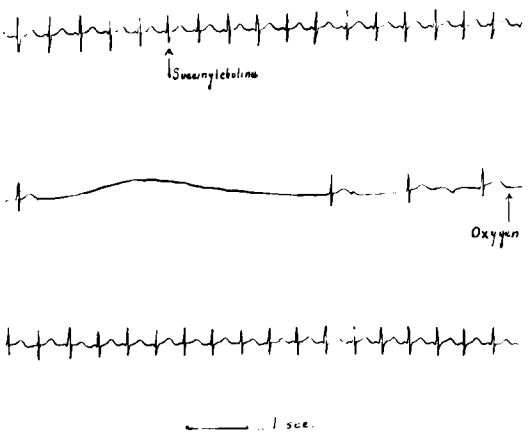

FIG. 2A. Short period of asystole followed by A-V rhythm ten to 12 seconds after administration of succinylcholine.

tion, his trachea was accidentally extubated. He was turned supine and the same dose of succinylcholine repeated, this time assuring patency of the airway. On the second occasion, there were no changes in the electrocardiogram. In both circumstances apnea persisted for ten minutes.

The third patient in whom A-V rhythm occurred was one in whom intentionally repeated doses of succinylcholine during endotracheal ether anesthesia caused changes in both pulse rate and rhythm. These changes were bradycardia and irregular A-V rhythm. This was an infant undergoing operation for ligation of patent ductus arteriosus.

DISCUSSION

Despite the theories of ganglionic blockade to explain tachycardia [4] or the cholinergic effect for the bradycardia,[5, 6] this study showed there was lack of a constant response to the intravenous injection of succinylcholine. It can be observed in the group without premedication (table 1) that the heart rhythm did not alter. In this same group, six of the nine patients showed an increase in heart rate. In all but one of the cases in the study, the anesthetic agents used in conjunction with succinylcholine were thiobarbiturate and nitrous oxide. Current investigation being conducted with other agents suggests that the effect of the succinylcholine upon the heart might be influenced by the anesthetic agent.

No single explanation or correlation can be made for the variation of behavior of the cardiac rate and rhythm. The relationship of hypoxia to the production of cardiac arrhythmias cannot be accurately evaluated unless samples of arterial blood are removed for measurement of oxygen saturation. In two of the patients, the decrease in pulse rate and change of rhythm were related to delay in the administration of oxygen.

SUMMARY

A series of 30 administrations of succinylcsholine intravenously to patients whose ages ranged from four weeks to 16 years and in whom

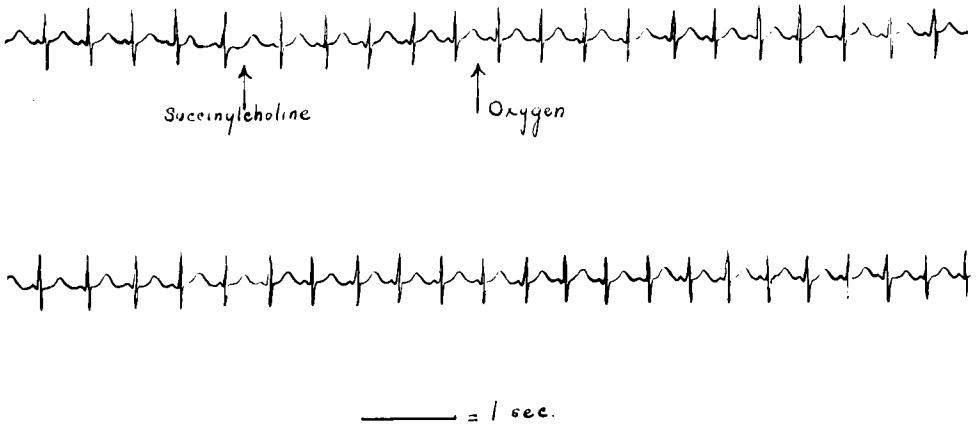

FIG. 2B. Same patient after second dose of succinylcholine. Cardiac rhythm and rate remained essentially without changes.

cardiac rate and rhythm were monitored electrocardiographically, is presented. Seventy per cent showed an increase in heart rate with unchanged sinus rhythm. Twenty per cent showed no changes in either rate or rhythm. Ten per cent showed a slowing in the heart rate with irregularity and the presence of A-V rhythm. In the latter group the change in rate and rhythm was transient and in all three patients there was reversion to S-A rhythm with the administration of oxygen.

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HYPNOTIC ANESTHESIA The first recorded case of hypnotic anesthesia in the United States was given on 28 June 1836. Most of the early operations performed under hypnosis (or mesmerism) were dental procedures. Further references are made to the use of hypnosis in surgical and obstetrical cases during the years 1837–1845. (*Carlson, E. T: Notes and Events–Addenda to the Early History of Hypnotic Anesthesia, J. Hist. Med. & Allied Sc. 15: 81 (Jan.) 1960.*)

TOXICITY The addition of epinephrine to tetracaine produced a highly significant reduction in systemic toxicity in mice. The toxicity was not altered appreciably when epinephrine was added to solutions of procaine, piperocaine, lidocaine and dibucaine. Procaine containing 1:50,000 epinephrine seemed more toxic than procaine alone. (*Avant, W. E., and Weatherby, J. H.: Effects of Epinephrine on Toxicities of Several Local Anesthetic Agents, Proc. Soc. Exp. Biol. & Med. 103: 353 (Feb.) 1960.*)