CYCLOPROPANE ARRHYTHMIAS IN THE CAT: THEIR CAUSE, PREVENTION AND CORRECTION

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THERE is continued interest in the cardiac irregularities observed during cyclopropane anesthesia in both animal and man. Numerous papers have been written to elucidate the mechanism by which these arrhythmias are prevented, produced or terminated once they have developed. We have studied the cat since it is most susceptible to the development of spontaneous arrhythmias during surgical stages of cyclopropane anesthesia. The arterial blood concentrations of oxygen, carbon dioxide and cyclopropane were correlated with the appearance of cardiac arrhythmias. We have also observed the effect of cardio-sympathectomy on the arrhythmias and have investigated the use of barbiturates in terminating arrhythmias.

Lucas and Henderson,[1] reporting the anesthetic activity of cyclopropane, noted changes in blood pressure which they interpreted as due to dropped beats or premature ventricular contractions. Stutzman and Pettinga[2] observed the case of production of arrhythmias in cats. They believed the cause of these arrhythmias to be abnormal afferent impulses arising in the upper gastrointestinal tract and mediated to the heart via the sympathetic outflow. Later, Allen and his associates[3] observed the spontaneously occurring arrhythmias during cyclopropane anesthesia in the cat, and finally concluded[4] that an intact sympathetic nervous system to the heart was essential for the production of these arrhythmias. When the chest was opened and the sympathetic chain between the stellate and fifth thoracic ganglion removed, the arrhythmias disappeared. Kurtz, Bennett, and Shapiro[5] reported their study of cardiac arrhythmias during cyclopropane anesthesia in man in 1936. In 1958, Lurie and co-workers[6] investigated the effects of cyclopropane and excess carbon dioxide upon cardiac arrhythmias in man.

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METHODS

Cats, usually weighing 3 to 4 kg., were gently restrained on an animal board. A control electrocardiogram was recorded from lead II in a Sanborn viso-cardiette. Anesthesia was then induced using cyclopropane of 200 ml. per minute and oxygen 400 ml. per minute in a to-and-fro system with a 90-Gm. absorber. As soon as surgical anesthesia was established, an endotracheal tube, approximately 6 mm. in external diameter, was inserted and connected directly to the to-and-fro system. An indwelling needle was placed into the femoral artery. The animal continued to breathe the cyclopropane and oxygen mixture until arrhythmias developed. If they did not appear in ten to fifteen minutes, the concentration of cyclopropane was increased to approximately 40 per cent. Arrhythmias were observed soon after this. When arrhythmias were noted, a sample of arterial blood was withdrawn and analyzed for oxygen, carbon dioxide, and cyclopropane in the Van Slyke-Neill apparatus with the Orcutt-Waters[7] technique for cyclopropane. Samples of blood from some animals were analyzed for hydrogen ion concentration by the use of the Beckman H2 hydrogen ion meter. The respiration of the animal was supplemented by intermittent pressure on the bag until the rhythm returned to normal and a second sample of arterial blood was removed for analysis. The cat was then permitted to breathe spontaneously until arrhythmias developed a second time, at which a third sample of arterial blood was withdrawn; supplemental respiration was again started, and as soon as the rhythm returned to normal, a fourth sample of blood was removed for analysis.

Bilateral thoracic sympathectomy was done on 7 cats by removing the sympathetic chain from the stellate to the sixth or seventh thoracic ganglion. The chest wall was closed in layers and the animals were given 300,000 units of benzathine-penicillin G; they healed readily without gross evidence of infection. These cats were anesthetized from one day to 144 days following the sympathectomy, and if arrhythmias developed, blood samples were taken and analyzed for oxygen, carbon dioxide and cyclopropane. Supplemental respiration was started, and as soon as the rhythm became normal, a second sample of blood was withdrawn for analysis.

The effect of a barbiturate, amobarbital or secobarbital 5–8 mg. per kilogram, on the arrhythmia was observed in 13 control cats and in 7 cats following sympathectomy. The cats were permitted to breathe spontaneously until arrhythmias were observed, at which time samples of arterial blood were drawn for analysis. The cats were then given amobarbital or secobarbital intravenously. An ECG record was made at the time of, and for 30 seconds following, the injection. Respiration was assisted for 1–2 minutes and then the cat was permitted to breathe spontaneously until the arrhythmia was again noted and a second sample of arterial blood was collected for analysis.

TABLE 1

THE CONCENTRATION OF CARBON DIOXIDE, OXYGEN AND CYCLOPROPANE IN ARTERIAL BLOOD OF CONTROL CATS DURING PERIODS OF ARRHYTHMIA AND IMMEDIATELY AFTER THE RETURN OF REGULAR RHYTHM

Cat	Carbon Dioxide (vol. %)		Oxygen (vol. %)		Cyclopropane (vol. %)	
	Irr.	Reg.	Irr.	Reg.	Irr.	Reg.
1	44.5	35.3	12.1	11.6	7.7	9.5
4	41.8	24.3	19.3	18.1	17.5	14.5
6	48.2	34.0	15.7	15.7	12.4	11.5
6	51.6	45.6	11.6	11.7	13.9	13.1
7	37.7	20.1	15.2	14.4	12.7	11.5
8	56.1	35.4	12.9	13.4	11.4	12.6
9	42.4	34.3	13.3	12.3	10.4	8.6
15	53.1	25.8	16.2	14.2	9.9	9.7
17	49.3	25.9	14.9	14.4	9.4	11.2
18	47.0	27.1	19.8	18.8	10.9	11.1
Average	47.3	30.8	15.1	14.5	11.6	11.3

TABLE 2

THE CONCENTRATION OF CARBON DIOXIDE, OXYGEN AND CYCLOPROPANE IN ARTERIAL BLOOD OF CATS WITH AN OPEN THORAX DURING PERIODS OF ARRHYTHMIA AND IMMEDIATELY AFTER THE RETURN OF REGULAR RHYTHM

Cat	Carbon Dioxide (vol. %)		Oxygen (vol. %)		Cyclopropane (vol. %)	
	Irr.	Reg.	Irr.	Reg.	Irr.	Reg.
1	42.5	30.4	12.3	13.6	9.1	15.1
4	43.0	28.7	13.2	15.8	18.3	13.6

THE RESULTS

In table 1 are the data obtained from the 10 cats used as controls. When arrhythmias were noted, the average concentrations of carbon dioxide, oxygen and cyclopropane in the arterial blood were 47.3, 15.1, and 11.6 volumes per cent, respectively. The average concentrations observed immediately after the

TABLE 3

THE CONCENTRATION OF CARBON DIOXIDE, OXYGEN AND CYCLOPROPANE IN ARTERIAL BLOOD OF CARDIO-SYMPATHECTOMIZED CATS DURING PERIODS OF ARRHYTHMIA AND IMMEDIATELY AFTER THE RETURN OF REGULAR RHYTHM

Cat	Carbon Dioxide (vol. %)		Oxygen (vol. %)		Cyclopropane (vol. %)		Post Sympathectomy
	Irr.	Reg.	Irr.	Reg.	Irr.	Reg.	
9	33.0	27.7	10.9	11.2	10.0	10.0	24 Hours
16	54.8	36.8	13.0	15.7	14.8	12.8	24 Hours
16	54.7	39.0	12.4	15.6	14.9	15.1	72 Hours
16	54.3	43.5	12.6	13.5	13.3	12.3	7 Days
2	57.0	43.5	8.7	10.3	20.2	15.0	9 Days
6	44.8	41.5	13.7	13.8	14.2	13.9	17 Days
2	50.3	40.4	10.9	15.0	12.5	13.4	35 Days
8	50.0	29.3	18.3	16.4	14.7	16.1	70 Days
1	56.0	46.7	14.3	12.8	17.0	14.1	80 Days
8	59.2	24.5	16.3	16.7	14.3	16.4	90 Days
2	62.4	19.5	15.7	14.2	12.5	11.9	120 Days
2	55.5	26.4	18.6	18.2	15.3	15.2	140 Days
Average	52.7	37.4	13.8	14.4	14.5	13.8	

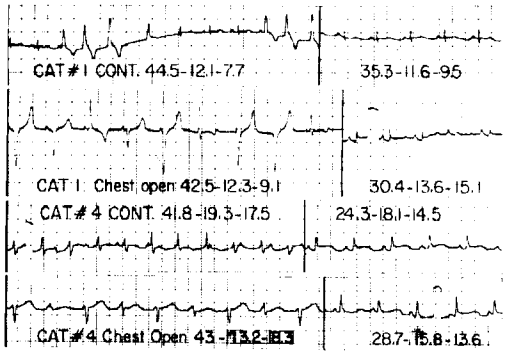

FIG. 1. Electrocardiographic records and blood gas data for 2 cats. The numbers on each strip of record are the volumes per cent of carbon dioxide, oxygen and cyclopropane in the arterial blood at the time the records were made. The top record shows the abnormal rhythm and the normal rhythm after supplemental respiration in the intact cat; the second record was obtained with the chest open.

return of normal rhythm as a result of supplemental respiration were 30.8 volumes per cent, carbon dioxide; 14.5 volumes per cent, oxygen, and 11.3 volumes per cent, cyclopropane. In table 2 are similar data from 2 cats which had arrhythmias during a period of inadequate respiration while both sides of the chest were open; adequate supplemental respiration returned the rhythm to normal. In table 3, are data from 12 cats which were obtained 24 hours to 140 days following bilateral sympathectomy. With high carbon

TABLE 4

CONCENTRATION OF GASES IN ARTERIAL BLOOD AT TIME OF DEVELOPMENT OF ARRHYTHMIAS ON SUCCESSIVE TRIALS DURING THE SAME EXPERIMENTAL PERIOD

Cat	Carbon Dioxide (vol. %)		Oxygen (vol. %)		Cyclopropane (vol. %)		Condition
4	41.8	43.0	19.3	13.8	17.5	18.2	Control
9	42.4	41.2	13.3	11.5	10.4	9.9	Control
15	53.1	50.0	16.2	14.9	9.9	11.0	Control
							Post Sympathectomy
2	57.1	55.0	8.7	8.0	20.3	19.0	9 Days
8	50.5	50.8	18.3	15.4	14.7	16.0	70 Days
8	59.2	60.2	16.3	14.2	14.2	13.5	90 Days
2	62.4	59.3	15.7	14.8	12.5	13.8	117 Days
2	55.5	56.2	18.6	15.9	15.3	12.7	140 Days
Average	52.7	51.9	15.8	13.5	14.3	14.2	

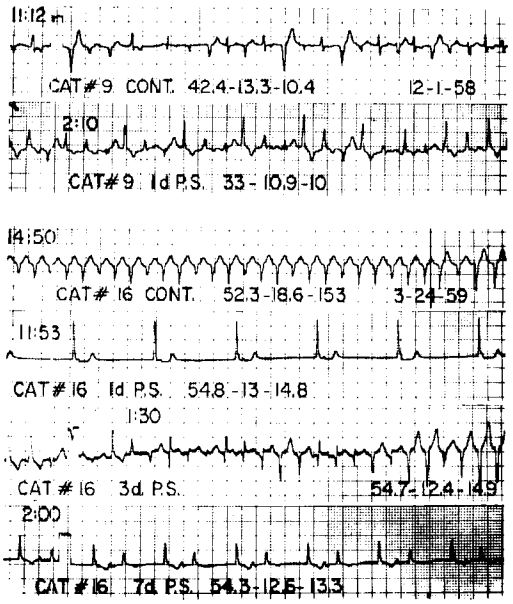

FIG. 2. Electrocardiographic records and blood gas data (volumes per cent carbon dioxide, oxygen and cyclopropane) for 2 cats before and from 1–7 days after sympathectomy.

dioxide content in arterial blood (average 52.7 volumes per cent) irregularities developed; and if the respiration was assisted, the rhythm returned to normal (average carbon dioxide content, 37.4 volumes per cent).

In figure 1 are electrocardiographic records for 2 cats, one from the control group and

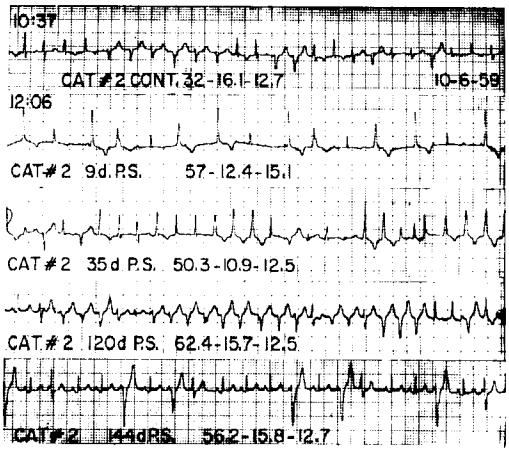

FIG. 3. Electrocardiograph records and blood gas data (volumes per cent carbon dioxide, oxygen and cyclopropane) for cat 2 before and from 9–144 days after sympathectomy.

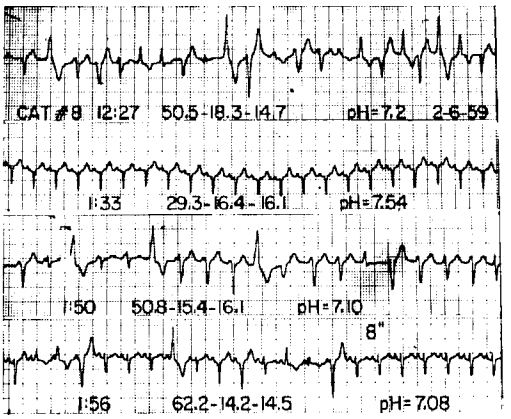

FIG. 4. Cat 8. Electrocardiographic records and blood gas data (volumes per cent carbon dioxide, oxygen and cyclopropane). Note the *p*H values in relation to the changes in rhythm. *12:27* Multifocal ventricular extrasystole during moderate depth of anesthesia with cat breathing on its own effort. *1:33* Return of normal rhythm under deep anesthesia but with supplemental respiration. *1:50* Return of arrhythmia with cat breathing on its own effort. *1:56* Return of normal rhythm 8 seconds following the intravenous injection of amobarbital 8 mg. per kilogram.

one with chest open, showing return of normal cardiac rhythm after supplemental respirations.

When the cat's heart was permitted to become arrhythmic on two successive occasions during the same experimental period and conditions, both periods of arrhythmias occurred with approximately the same concentration of carbon dioxide (average 52.7 and 51.9 volumes per cent—table 4).

Electrocardiographic records of cats, one to 144 days following sympathectomy, showed that arrhythmias were as severe after the sympathectomy as before (figs. 2 and 3). Figure 4 is an electrocardiographic record obtained during spontaneous respiration and supplemental respiration, showing cardiac arrhythmias when the carbon dioxide content and hydrogen ion concentration of the arterial blood were elevated. Supplemental respiration produced a reduction in the carbon dioxide content and hydrogen ion concentration and a return of normal rhythm. The intravenous injection of amobarbital, 8 mg. per kilogram, during a period of increased carbon dioxide

TABLE 5

THE CONCENTRATION OF BLOOD GASES AT THE ONSET OF ARRHYTHMIAS BEFORE AND AFTER BARBITURATES, AMOBARBITAL OR SECOBARBITAL, 8 MG. PER KILOGRAM

Cat	CO_2 Vol. %		O_2 Vol. %		C_3H_6 Vol. %	
	Before	After	Before	After	Before	After
1	50.0	56.0	13.6	14.3	10.4	17.0
2	59.3	67.5	14.8	10.8	13.8	14.8
2	55.5	63.7	18.6	13.6	15.3	12.7
8	52.1	69.3	13.3	9.2	12.5	17.5
8	50.8	62.2*	15.4	14.2	16.0	14.5
9	40.5	47.5	13.4	11.8	11.8	13.2
11	39.3	54.0	14.5	13.2	9.9	13.0
12	52.5	55.9	16.4	15.8	10.1	11.1
13	53.7	57.6	16.0	15.1	12.8	15.8
15	50.0	64.5*	14.9	11.6	11.0	14.0
Average	50.4	59.8	15.1	13.0	12.4	14.4

* No arrhythmias in these two cats after amobarbital.

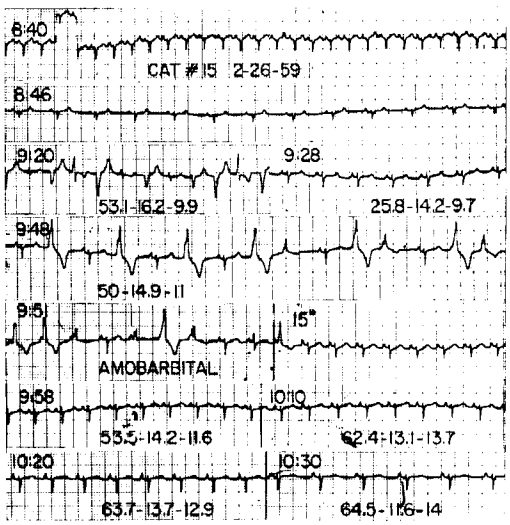

FIG. 5. Electrocardiographic records and blood gas data (volumes per cent carbon dioxide, oxygen and cyclopropane) for cat 15 showing the prolonged effect of amobarbital in maintaining a regular rhythm. *8:40* Control. *8:46* Light anesthesia. *9:20* Light anesthesia with moderate CO_2 content and cardiac arrhythmia. *9:28* Regular rhythm after supplemental respiration. *9:48* Bigeminy-trigeminy. *9:51* Return to normal rhythm in 15 seconds after amobarbital 8 mg. per kilogram. *9:58, 10:10, 10:20* and *10:30* rhythm remained regular with increasing carbon dioxide content. This cat was apneic from *9:51* to *10:30* but continued to receive oxygen-cyclopropane so that oxygen requirements were adequate. After *10:30* the cyclopropane was reduced, supplemental respiration started and bilateral sympathectomy done.

TABLE 6

THE EFFECT OF AMOBARBITAL WHEN GIVEN INTRAVENOUSLY IN CATS UNDER CYCLOPROPANE ANESTHESIA DURING A PERIOD OF SPONTANEOUS ARRHYTHMIA

Cat	Type Arrhythmia	Rate	Amobarbital mg./kg.	Time (seconds)	Rate	Duration (minutes)	Condition
1	MFVEx	230	8	12	200	4	control
	MFVEx	200	8	8	120	5	14 d p.s.
	MFVEx	200	8	7	90	10+	40 d p.s.
	MFVEx	200	8	8	120	15+	60 d p.s.
2	MFVEx	160	8	7	140	10	9 d p.s.
	Tot. irr.	140	6	5	130		9 d p.s.
	MFVEx	180	6	54	150	4	22 d p.s.
	AV Dis	150/V80	6	12	120		22 d p.s.
	MFVEx	200	4	25	150	10+	34 d p.s.
	MFVEx	240	8	8	185	2	120 d p.s.
3	MFVEx	140	8	9	120	10	control
	PVEx	150	8	6	150	5	control
	N.PVEx	180	8	12	110	4	control
	Tot. irr.	170	4	14	170	5+	control
4	MFVEx	200	8	40	180	5+	control
5	Nodal	140	8	30	140		control
6	Tri gem	160	8	6	165	3	7 d p.s.
	MFVEx	180	8	8	150	10+	17 d p.s.
	PVEX	120	8	6	140	10+	21 d p.s.
7	MFVEx	190	8	10	140	3	control
	Nodal	100	8	15	100	25+	1½ h p.s.
8	MFVEx	200	8		260 vt		control
	MFVEx	190	8	15	180	20+	18 d p.s.
	N.MFVEx	160	8	9	180		70 d p.s.
9	MFVEx	200	8	8	180	15+	control
	PVEx	200	8	18	180	4	control
	PVEx	220	8	10	180	6	control
	MFVEx	190	8	9	200	3	control
	PVEx	220	8	9	200	6	control
	MFVEx	180	8	9	120	14+	1 d p.s.
10	PVEx	160	8	6	180	4+	control
	AV Dis	180	8	6	190		control
11	MFVEx	180	8	15	170		control
12	N.PVEx	220	8	12	100	2+	control
13	PVEx	140	8	12	140	2	control
15	Bi gem	120	8	15	140	20	control
16	Nodal	120	8	9	100	11	1 d p.s.
	VT	180	8	9	160	14	3 d p.s.
	VT	220	8	9	165		7 d p.s.
17	MFVEx	170	8	15	180		control
	AVN	100	6	11	150		3 d p.s.
	MFVEx	240	8	30	300 vt		17 d p.s.
	MFVEx	220	8	12	220	12	17 d p.s.

Type = MFVEx = Multifocal ventricular extrasystole. Tot. irr. = Totally irregular. AV Dis = Auricular ventricular dissociation. PVEx = Premature ventricular extrasystole. Tri gem = Tri geminy. Nodal = A. V. nodal rhythm. VT = Ventricular tachycardia.

Time = Seconds after intravenous injection of amobarbital before rhythm returned to normal or to ventricular tachycardia.

Duration = Duration of regular rhythm before second arrhythmia developed or when time marked +, duration before further experimental procedure done.

Condition = Control animal or days post bilateral sympathectomy.

content and hydrogen ion concentration, resulted in a return of normal rhythm.

Data from the 10 cats given intravenous amobarbital or secobarbital (8 mg./kg.) are recorded in table 5, showing a marked increase in the concentration of carbon dioxide in the blood after administration of the barbiturate. Cardiac arrhythmias developed in the cats when the carbon dioxide concentration approximated 50 volumes per cent, whereas after the barbiturate, the average concentration of carbon dioxide at the time of the arrhythmias was 60 volumes per cent. The oxygen content was decreased and the concentrations of cyclopropane were increased after the barbiturate. An example of this effect is given in figure 5; in two separate trials the cardiac rhythm became irregular with 53.1 and 50. volumes per cent of carbon dioxide. After the administration of the barbiturate, the rhythm remained regular for 30 minutes with 62.4, 63.7 and 64.5 volumes per cent of carbon dioxide during which time there was a decrease in arterial oxygen from 14.9 to 11.6 per cent and an increase in the arterial cyclopropane from 11 to 14 per cent.

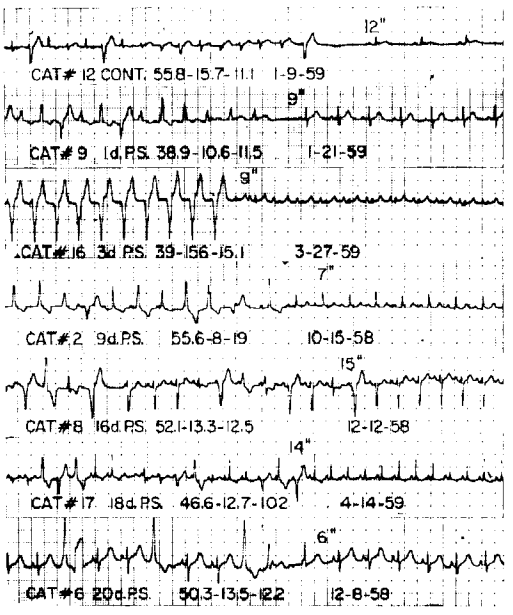

FIG. 6. Electrocardiographic records showing the return to normal rhythm in 6 to 15 seconds after the intravenous injection of amobarbital 8 mg. per kilogram in 7 cats, a control and from 1–20 days after sympathectomy.

In 43 attempts to correct arrhythmias by the intravenous injection of a barbiturate in the cat success was noted in 41 instances. These were made in control animals and in cats that had been sympathectomized from 1 hour to 144 days. In 26 of 43 instances, the heart rate decreased from 10 to 110 beats per minute, (average 42), following the injection of the barbiturate. In 9 of 43 trials the heart rate increased more than 5 beats per minute. In two cases of multiple focus ventricular extrasystoles, of 200 a minute, the barbiturate produced a single focus ventricular tachycardia, with a rate of 250 per minute. Following a single intravenous injection of barbiturate, the rhythm remained normal for an average of 10 minutes (table 6).

The effect of an intravenous injection of a barbiturate upon the cardiac rhythm in 7 cats, a control cat and in cats from 1 to 20 days following sympathectomy are shown in figure 6. From 6 to 54 seconds elapsed between the injection of the barbiturate and the return to normal rhythm.

DISCUSSION

From these observations three general statements may be made. (1) Cardiac irregularities are easily and regularly produced in cats respiring a mixture containing 30–40 per cent cyclopropane in oxygen. The rhythm can be returned to normal by supplemental respiration which produces a reduction in the carbon dioxide content of the arterial blood. (2) An intact sympathetic innervation of the heart is not necessary for the production of arrhythmia in the cat. (3) The irregular rhythm during cyclopropane and high carbon dioxide content can be returned to normal by the intravenous injection of amobarbital or secobarbital.

The data presented in table 1 show that during the periods of irregularities and the period of regular rhythm, the oxygen and cyclopropane contents of the arterial blood are relatively constant, whereas the carbon dioxide content is approximately one third lower during the period of regular rhythm than during the period of arrhythmia; thus the carbon dioxide content, or more likely the P_{CO_2}, must be a deciding factor in the production of arrhythmia during a fixed level of anesthesia with cyclopropane. This is in agreement with

the observations made by Lurie *et al.*,[6] who found that the cardiac irregularities could be produced routinely in man by elevating the P_{CO_2} during cyclopropane anesthesia, and that by reducing the P_{CO_2} the rhythm returned to normal. The mechanism by which the excess carbon dioxide acts to increase the irritability of the ventricular conducting system or myocardium in the cat is not known; Price and associates[8] believe that hypercarbia, in man, increases the rate of liberation of catechol amines from the sympathetic nerve endings in the myocardium and that this causes ventricular arrhythmias during cyclopropane anesthesia.

In contrast to the observations of Allen and co-workers[4] who found that bilateral cardiac sympathectomy, stellate to sixth ganglion, prevented the cardiac arrhythmia in cat, we were unable to prevent the arrhythmia by sympathectomy, but were able to prevent or terminate them by supplemental respiration. In two cats showing arrhythmia with the chest open, an increase in supplemental respiration with a reduction in carbon dioxide content terminated the arrhythmias; in 12 experiments on six cats from 1 to 140 days following bilateral sympathectomy cardiac irregularities were routinely observed and these were terminated by supplemental respiration. It is possible that the results obtained by Allen[4] could have been due to excess ventilation during the thoracotomy and sympathectomy and not a result of the sympathectomy, because theirs were acute experiments.

The use of amobarbital or secobarbital to terminate the arrhythmias in the cat was based upon the observation of Robbins, Baxter and Fitzhugh[9] who showed that amobarbital corrected premature ventricular extrasystoles in dogs under cyclopropane anesthesia, and that the barbiturates prevented the arrhythmias that occur with cyclopropane, excess carbon dioxide and oxygen lack in dogs. Robbins[10] also used secobarbital to terminate ventricular arrhythmias in man during cyclopropane anesthesia. The mode of action of the barbiturates in terminating the arrhythmia in the cat is not known, but it is not by way of blocking vagal effect, because as a rule the rate after the return to normal rhythm was less than during the arrhythmia.

Summary

Studies in the cat of the relationship of the concentration of carbon dioxide, oxygen and cyclopropane in the arterial blood to the cardiac rhythm indicate that an elevated carbon dioxide content is associated with ventricular arrhythmia.

Bilateral sympathectomy, stellate to the sixth thoracic ganglion in 7 cats did not prevent the occurrence of ventricular arrhythmias. An intact sympathetic system to the heart appears not necessary for the production of the arrhythmia in the cat.

The intravenous administration of a barbiturate, amobarbital or secobarbital (5–8 mg. per kilogram) terminated the ventricular arrhythmia in 41 or 43 trials.

We believe that carbon dioxide retention is the main cause of cardiac arrhythmia during cyclopropane anesthesia in the cat, that supplemental respiration will prevent the arrythmia, and that either supplemental respiration or intravenous administration of barbiturates wil correct the arrhythmia.

The authors acknowledge the technical assistance of Ruth Gay Robbins.

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ANTIBIOTIC PARALYSIS Not only does neomycin intraperitoneally produce neuromuscular blockade but also streptomycin may produce the same effect. The intravenous administration of antibiotic agents in rabbits further demonstrates this action. Polymyxin B sulphate and kanamycin sulfate in addition to the above mentioned drugs produce muscular blockade in rabbits. It is suggested that when intraperitoneal antibiotics seem indicated postoperative administration through catheters placed during surgery may be a solution to the problem. (*Pittinger, C. B., and Long, J. P.: Potential Dangers Associated with Antibiotic Administration During Anesthesia and Surgery, A. M. A. Arch. Surg. 79: 207 (Aug.) 1959.*)

OXYTOCIN–SUXAMETHONIUM In 5 patients who had received pitocin infusions for more than 8 hours during labor and in 6 others who received 2–5 units of pitocin in infusion in more than 4 hours, it was observed that the administration of succinylcholine in ordinary apneic doses produced no fasciculations, did not cause apnea, and had a slow onset, prolonged duration, and lessened intensity of action. Recovery was hastened by neostigmine. The most marked deviations from the usual action of succinylcholine occurred when the pitocin infusion ran over 24 hours. Pitocin causes changes in intracellular ionic fluxes, especially of potassium. The observed changes are attributed to this. (*Hamer Hodges, R. J., and others: Effects of Oxytocin on Response to Suxamethonium, Brit. M. J. 1: 413 (Feb. 14) 1959.*)

DIGITALIS–CALCIUM A chelating agent, sodium versenate (Na EDTA) is capable of binding serum calcium and effecting a transient hypocalcemia when given intravenously. This agent has proved useful in eliminating cardiac arrhythmias due to digitalis intoxication, by producing a transient reduction of serum calcium. Twenty-seven digitalized patients were studied, 11 having evidence of digitalis intoxication and 14 showing none. Control electrocardiograms were done and an infusion of 3 grams of Na EDTA in 400 cc. of 5 per cent glucose in water were given in 30 minutes with electrocardiographic tracings taken every five minutes. In all cases serum calcium was reduced and in all cases showing digitalis intoxication there was abolition or reduction of cardiac arrhythmias for periods of 15 minutes to two hours. Drug may also be useful as a test to see if a given arrhythmia or conduction disturbance may be due to toxic effects in digitalized patients. (*Jick, S., and Karsh, R.: Effect of Calcium Chelation on Cardiac Arrhythmias and Conduction Disturbances, Am. J. Cardiology 4: 287 (Sept.) 1959.*)

ISOPROPYLCHLORIDE Tachycardia and arrhythmia were observed in children under open drop isopropylchloride–oxygen anesthesia. The arrhythmias disappeared promptly after the drug had been stopped. The drug may be used for both long or short anesthesia, but not for intubation of the trachea. (*Tischer, W., and Bock, U.: Electrocardiographic Changes During Isopropylchloride Anesthesias in Children, Der Anaesthesist 8: 293 (Oct.) 1959.*)