

## AN EXPERIMENTAL RESEARCH INTO THE RESUSCITATION OF DOGS KILLED BY ANESTHETICS AND ASPHYXIA.

By GEORGE CRILE, M.D., AND DAVID H. DOLLEY, M.D.

(*From the Laboratory of Surgical Physiology, Western Reserve Medical School, Cleveland.*)

### PLATES XLII-XLIX.

In a previous communication by one of us (Crile) resuscitation was attempted by means of both direct and indirect cardiac massage, with and without artificial respiration, with and without intravenous saline infusion, and with and without the addition of adrenalin. The results of these experiments may be summarized as follows: By cardiac massage alone animals were rarely resuscitated at any time after quiescence of the circulation and respiration; by combining either direct or indirect cardiac massage with artificial respiration and the head-down posture a certain percentage of the animals were recovered after the lapse of from one to three minutes. The results, however, were quite uncertain, and in the case of death from chloroform recovery was the exception. By adding to cardiac massage artificial respiration and intravenous saline infusion, resuscitations were in a slightly greater proportion successful. The same procedures, with the addition of adrenalin to the intravenous saline infusion, were markedly more successful in the deaths from asphyxia than rarely so in the cases of death from chloroform. It required in almost every instance a vigorous compression of the thorax over the heart for a considerable time, varying from five to ten minutes, before resuscitation could be accomplished. Even then there were a considerable number of failures. In the majority of these failures autopsy showed the heart to be in a state of marked distension, indicating complete cardiac paralysis; not infrequently when results were delayed, the acute cardiac dilatation was partly relieved by venesection

and by placing the dog in the inclined head-up posture, thereby partly relieving the paralyzed heart of its over-distension.

In this research the problem was approached from the arterial side, with the following physiologic factors as a basis: The physiological researches of Ringer, Kulibio, Sollman, and others had shown that the excised heart could be made to beat again, even many hours after excision, when the coronary arteries were subjected to a considerable pressure from some circulating medium. The researches of Sollman showed that the *inauguration* of the beat was more dependent upon the physical factor of the increased pressure in the coronary arteries than upon the quality of fluid producing such pressure. He was able to inaugurate cardiac beats by perfusing the coronaries with metallic mercury. The basic problem, then, in resuscitation seemed to us to be that of securing by means of some infusion a coronary pressure approximately amounting to from thirty to forty millimeters of mercury. It was impossible to raise the coronary pressure in the intact animal to this height by means of cardiac massage alone; it was almost impossible to raise it by the mechanics of infusion alone, and generally impossible to raise it to this height by means of cardiac massage and plain infusion combined.

The value of adrenalin in raising the blood pressure, by its action upon the vascular walls in the state of suspended animation, has been heretofore thoroughly established. Introducing the adrenalin into the venous circulation, while easy and practical, had the following disadvantage: the adrenalin first came in contact with the vessels having the least power of influencing the blood pressure, and before a material rise could be effected by its action upon the arteries it was necessary that the solution should pass through the right heart, the lungs, then back to the left heart on its way to the aorta, thence affecting directly the coronary artery. In the previous research it was found that this too often caused an accumulation of solution and blood in the dilated, paralyzed chambers of the heart, defeating resuscitation.

It seemed reasonable to us to suppose that the most direct and effective way of producing a coronary pressure amounting to thirty or forty millimeters of mercury was by introducing a

solution of adrenalin into the arterial system toward the heart. In this way the moment the adrenalin was introduced it caused a contraction of the strong arterial walls, beginning to produce an arterial pressure which would communicate directly with the coronary artery without first passing through the already distended and paralyzed chambers of the heart and through the lungs. These considerations were particularly impressed upon us by a clinical case which has elsewhere been reported.

Sixty experiments were made in which the tracings were preserved and the records noted. In the recovery experiments the greatest care was taken to secure for the animals all the comfort possible, and as few of this class of experiments were made as was consistent with the collection of data sufficient for our conclusions.

Three groups of cases were investigated: (1) death from asphyxia, (2) from ether, (3) from chloroform. Inasmuch as the methods and results were quite similar, they may all be described together.

We first tested the method as to its usefulness in raising the blood pressure, and found that by tying a cannula into an artery toward the heart and introducing through it a solution of adrenalin the blood pressure was immediately raised, the latent period being distinctly less than when the solution was introduced intravenously. In fact, we may say that there was no latent period, the blood pressure rising instantly. It was found, too, that the most effective method of raising the blood pressure was by the injection of the full strength of 1-1000 solution of adrenalin by means of a hypodermic syringe into the rubber tubing near the cannula. The results in these experiments showed a distinctly more powerful action than in the experiments in which the solution of adrenalin was previously made and poured into the funnel.

The first experiment was made upon an animal killed by chloroform. A cannula was inserted in the femoral artery directed toward the heart. After two minutes artificial respirations were begun and the saline solution was allowed to flow for perhaps ten seconds; then a hypodermic injection of from one to two cubic centimeters of 1-1000 solution of adrenalin was given into.

the tube near the cannula. A few seconds later the blood pressure began to rise steadily; then a few firm pressures upon the thorax over the heart caused a leaping up of high pulse waves, and at the end of three quarters of a minute the heart beat vigorously, driving the blood up into the infusion bottle, which was raised to a height of five feet. The saline injection and cardiac massage were discontinued, and in a few minutes irregular respirations slowly began, decreasing in force and increasing in frequency until the normal was established. The animal was then killed.

Following this experiment others were made, each time a longer period being allowed to elapse from the moment of death until the beginning of resuscitation. In determining the moment of death from chloroform it was found that by administering an excessive dose by inhalation the blood pressure sank with remarkable rapidity to the abscissa line, but that after this in forty per cent. of such deaths feeble pulse waves reappeared after periods varying from a few seconds to over three minutes, the blood pressure rising to from ten to thirty millimeters, then gradually declining, the pulse wave progressively diminishing in length. After a period of resumption of beat varying from a few seconds to six minutes, and averaging two minutes, the pressure usually fell to the abscissa line and no further waves were recorded upon the drum. It was only after the pressure had reached the abscissa line, after all pulse waves had ceased, and no heart sound, however feeble, could be auscultated, that we recorded the moment of death. The time was then extended to five, ten, fifteen, twenty, and thirty minutes in adult dogs, and in puppies up to an hour.

The limitations of the method soon appeared. Among them were the following: (a) ante-mortem clotting, (b) over-distension of the heart, (c) later failure of the heart, circulation, and respiration, (d) the apparently imperfect recovery of the brain.

*Ante-mortem Clotting.*—When a considerable time had elapsed, say ten minutes or more after death, and particularly if there had been some delay in resuscitation of the heart, thereby inducing vigorous and protracted massage, clotting occurred more frequently. Direct cardiac massage was then made, which seemed

further to facilitate clotting. In other cases indirect massage was made with extreme vigor, showing like results. Finally, an animal was etherized to the point of respiratory failure, then sustained with a very low blood pressure by means of artificial respiration combined with vigorous cardiac massage, for a period of about seven minutes, after which the heart was opened before it had ceased beating and small clots were found. No infusion in this case had been given and no other operation was performed. This seemed to confirm our previous conclusion that either vigorous or prolonged massage of the heart predisposed to ante-mortem clotting and that direct massage of the heart favored clotting more than indirect massage by rhythmic pressure upon the intact thorax over the heart.

It was found that animals could be resuscitated after several compressions of the chest by waiting a little longer for the rise in the pressure from the adrenalin infusion alone. It was obvious that in our previous experiments we gave unnecessary massage as to force, frequency, and duration. This is the more reasonable since the object of the massage is not that it will materially aid the heart in *inaugurating* its rhythmic contractions, but that it will distribute the fluid through the circulation which has been injected by gravity alone. Pressing upon the thorax itself, would effect a certain amount of circulation, if no heart existed and a system of vessels, one having valves and the other not, replaced it, and in the dead animal pressure upon the thorax alone is capable of producing an artificial circulation. This is by no means accomplished by its action upon the heart solely, but by its action upon all the large vessels—arteries, veins, and capillaries together.

We also observed what on *a priori* grounds we supposed to be true, namely, that a tendency to clotting was increased in a direct ratio with the lapse of time. When clotting occurred resuscitation was practically always impossible. The heart would beat for a short time, but the beat would soon fade away.

*Over-distension of the Heart.*—In the unsuccessful cases, particularly those in which a considerable amount of infusion was given, the heart at autopsy was found over-distended. This

over-distension was due to two factors; the first was the inevitable accumulation of blood in the heart during its gradual failure either in asphyxia or following chloroform and ether; second, the infused fluid passed through the coronary arteries, through the coronary vein into the right side of the heart. The left heart was not generally so distended as the right heart. It is obvious that if for any reason resuscitation is not promptly made the accumulation of fluid in the right heart must not only be considerable but must occur under pressure; since the valves prevent the escape of the fluid toward the periphery of the veins the accumulation must be largely in the heart itself, and when this condition is reached a peculiar vicious circle has been established. The greater the efforts at resuscitation by increasing the infusion the greater the distension of the paralyzed ventricle and the more certain is the failure of the attempt. In some instances after the chest was opened and the heart on inspection was found distended, rhythmic contractions of the heart began; on opening the pericardium in one instance a splendid resuscitation (Fig. 6.) was made.

*Later Failure of the Heart, Circulation, and Respiration.*—In a number of instances after a temporary resuscitation the circulation and the respiration failed, after which a second attempt at resuscitation was useless. In these cases the failure was generally gradual and occurred after the lapse of a varying period of time, ranging from three and a half to twenty hours. In these cases for the most part ten minutes or more had elapsed before resuscitation was attempted. A few animals in which resuscitation was attempted in less than ten minutes also showed later failure. We are not prepared to state the exact cause of this later failure.

*The Apparently Imperfect Recovery of the Brain Proper.*—In resuscitations after ten minutes, particularly those after from fifteen to twenty minutes, it was noted that while the circulation and the respiration were satisfactory the animal did not regain consciousness; the reason for this we are not at present prepared to state, but it will be further investigated.

We also investigated the influence of prolonged chloroform

anesthesia prior to death. Animals were kept under chloroform anesthesia for an hour and then killed by an over-dose. They were resuscitated perhaps a little less readily than the animals killed immediately by an over-dose, though their resuscitation was, up to ten minutes, satisfactory.

*Hirudin.*—After encountering the difficulty with clotting in the delayed cases, we tested the value of hirudin in the prevention of clotting. In the cases in which hirudin was given prior to death no clotting occurred in a single instance in the heart or blood-vessels. In these cases, which were limited to puppies not over three months old, we are, however, inclined to believe that oedema of the lungs was distinctly more marked. The blood seemed to lose its viscosity and the oedematous fluid accumulated rapidly in the bronchial tubes, and finally flowed out of the trachea, presenting a reddish tinge. In the successful cases this always occurred before the inauguration of the heart-beat. There was in addition free blood-tinged fluid in the thorax. While hirudin, in the case of puppies at least, entirely obviates the clotting factor it introduces an equally fatal obstacle, *i. e.*, waterlogging of the thorax, which we were not able to prevent by reducing the infusion pressure to a minimum consistent with its efficacy.

*Puppies.*—The experiments on puppies showed at once that resuscitation was more readily done, and successful after a longer period after death, than in the adult dog. In three instances thirty-five minutes after death a fairly good circulation was obtained (Fig. 8).

*Solution Employed for Infusion.*—The observations in this series as well as in other researches within the past year have led us to conclude that Ringer's solution yields more favorable results than saline solution. The most satisfactory results were obtained with the infusion bottle at a height of five or six feet.

*Conclusions.*—Based on the data supplied by these researches and those of preceding researches on similar lines we arrive at the following conclusions:

- (1) Animals after death from chloroform, ether, or asphyxia up to five minutes were uniformly and readily resuscitated,

provided that the full technique which has been described was applied; up to ten minutes there was an occasional failure; beyond ten minutes consciousness was rarely restored, and the proportion of successes in the resuscitation of the circulation and the respiration diminished with the lapse of time; after twenty-three minutes in adult dogs and thirty-five minutes in puppies complete return of the circulation was not accomplished.

(2) After death from chloroform and ether animals were more readily resuscitated than after death from asphyxia.

(3) Resuscitation, if successful, occurred within one minute after the administration of adrenalin in the majority of instances; it rarely occurred after an interval greater than three minutes.

(4) The greatest difficulty to be overcome was clotting. The probability of clotting increased in direct ratio with the lapse of time after death. Cardiac trauma predisposed to clotting.

(5) The younger the animal, up to certain limits, the more readily it was resuscitated.

(6) Any artery may be used for the infusion, though the advantage is probably slightly in favor of the carotid. The infusion should be directed toward the heart.

(7) The probable success of resuscitation is greater in inverse relation to the lapse of time after death; a rapid rise in the arterial pressure is attained by arterial infusion with a therapeutic dose of adrenalin, together with good artificial respiration and the avoidance of unnecessary cardiac trauma by massage. All artificial aids should cease as soon as the functions are competent.

#### ILLUSTRATIVE PROTOCOLS.

EXPERIMENT IV. *The effect of time and of massage on the formation of clots.*  
Mongrel mastiff, 20 K. Chloroform administered until death.

10.09—Respiration stopped.

10.14—Pulse was not palpable at carotid.

10.15—Heart sounds ceased. The opened carotid bled for some time with a non-pulsating drop flow which stopped when the head was raised. A tracheal cannula was rapidly adjusted, and with a manometer cannula in the left carotid a small cannula in the right carotid was connected with the saline infusion bottle at a height of seven feet. A pin-hole cannula in the left femoral vein was connected to an infusion bottle containing 1-100000 solution of adrenalin chloride at a height of three feet.

10.47 (32 minutes)—Resuscitation attempted. Cardiac massage was vigorous. Two cubic centimeters of the solution of adrenalin chloride were injecte

into the infusion tubing. No results came from the first dose of adrenalin within two minutes. With a second dose there was a perceptible manometer pulse and measures were temporarily stopped, the blood pressure being 40 mm., but the level quickly declined to the abscissa. Further efforts, namely massage and a mass dose of adrenalin with a continuous 1-10000 infusion into the femoral vein, failed to improve the heart-beat and to raise the blood pressure. After some time, when no pulse could be felt, the carotid infusion was again started and a second time beats were evoked, but other measures failed to keep up the heart's activity. In the latter part of the experiment the dog's feet were lowered.

At autopsy the heart was full of bloody fluid, the pericardium was tense and the heart dilated. Clots were found in the heart.

**EXPERIMENT XXVIII. Resuscitation six minutes after death.** (Fig. 1.) Ether anesthesia. This experiment was performed with moderate aseptic precautions.

10.59—Control blood pressure 156 mm.

11.00—Began to force ether.

11.04—Respiration stopped, after which cardiac failure gradually ensued.

11.06—Heart sounds ceased and pulse not palpable.

11.12—Resuscitation was begun (6 minutes). Saline infusion plus 2 c.c. of adrenalin chloride (1-1000) was administered through the axillary artery together with artificial respiration and gentle heart massage. About twenty respirations were given. The heart began to beat in about one and a half minutes. The blood pressure rose sharply to 240 mm., which level was sustained for several minutes. It gradually fell to 80 mm., at 11.24.

11.18—Spontaneous respiration began.

11.20—Ether was required and given. The respiration was at first very irregular and spastic, gradually becoming quiet and regular.

11.29—Blood pressure 130 mm.

11.40—Blood pressure 160 mm.

11.50—Blood pressure 160 mm. The trachea was sewed up, the wounds in the neck and axilla were closed with large gauze drainage and were bandaged. The dog was removed from the table and allowed to recover. Late in the afternoon the dog had considerable respiratory difficulty, apparently due to obstruction in the trachea. The animal was etherized, a blood clot was removed from the trachea, a cannula inserted, and the neck bandaged.

The next morning his condition was good and he was walking about, but as considerable pus was coming from the tracheal cannula it was decided to kill him. He was etherized and the carotid artery was again connected with the manometer.

10.30—Blood pressure 106 mm.

11.00—Blood pressure 112 mm.: the difference being due to the depth of anesthesia. The dog was then killed.

Beyond the infection about the trachea, there were no macroscopic lesions found at autopsy.

**EXPERIMENT XIX. Resuscitation five minutes after apparent death.** (Fig. 2.) Mongrel in rather poor condition; weight 7.3 K. Anesthetized with ether.

10.35—Control blood pressure 140 mm.

10.42—Trachea clamped. Several control determinations of blood pressure showing stages of asphyxia were made.

10.44—Last respiration occurred.

10.47—Heart sounds had ceased, and pulse was not palpable.

10.54½—Resuscitation started: Infusion of saline through axillary artery, the infusion bottle being placed at a height of five feet, plus two c.c. solution of adrenalin; artificial respiration was begun at 10.55 without any heart massage. The heart began to beat in about one minute, the blood pressure rising quickly to 206 mm. from which it gradually declined to 130 mm. at 11.10.

11.07—Spontaneous respirations occurred. Artificial respiration was stopped.

11.18—Ether required.

11.50—Control blood pressure 128 mm.

12.05—Control blood pressure 122 mm.

11.15—Control blood pressure 122 mm. Ten mg. of morphine per kilo was administered. Ether was discontinued.

2.05—Control blood pressure 108 mm.

2.21—Control. The animal was killed as soon as recovery was apparent.

4.41—Control taken. Five hours and forty minutes after resuscitation the dog's condition was excellent, but on account of the septic condition of the operative wounds he was killed.

**EXPERIMENT XLVII. Temporary Resuscitation.** (Fig. 3.) Mongrel weighing about 10 K.

10.20—Anesthesia with ether started.

10.59—Control blood pressure 110 mm.

11.06—Trachea clamped.

11.09½—Control showing a stage of asphyxia.

11.11½—Control showing a stage of asphyxia.

11.12—Heart sounds ceased and pulse was not palpable.

11.22—Started resuscitation (10 minutes). Infusion of Ringer's solution, plus 2 c.c. of adrenalin (1—1000) was given through axillary artery, artificial respiration and firm but gentle heart massage were started simultaneously. The heart beat after two minutes, the blood pressure rising to 220 mm. with the typical adrenalin effect. This level was maintained for over three minutes and the decline was gradual.

11.31—Control blood pressure 180 mm.

11.35—Control blood pressure 80 mm. Pulse wave much smaller. Burning of paw evoked no reaction. There were no corneal reflexes.

11.40—Spontaneous respiration occurred. Control.

11.45—Control blood pressure 50 mm. Pulse wave improved.

11.55½—Control blood pressure 60 mm. Artificial respiration stopped. No corneal reflexes, feeble knee jerk in right hind leg.

12.00½—Control. Sharp reaction to paw burning, followed by a secondary reaction with the higher level maintained. Knee jerk appears in left hind leg.

12.07—Control blood pressure 80 mm. Dilating and burning the anus evokes a vasomotor rise, but no respiratory reflex. Corneal reflex appears in right eye. Pupils were extremely dilated after resuscitation, but now are narrowed to some extent. They do not react to light.

12.15—Control blood pressure 50 mm. Pulse wave weaker. Corneal reflex appears in left eye. Respirations have assumed a normal rhythm.

1.06½—Control blood pressure 50 mm. Corneal reflexes improved. In-

competence of sphincters. The animal is entirely anesthetized to pain and completely unconscious. Rigidity of the hind legs appears. Respirations are becoming more labored and quicker.

2.00—Pupils slowly dilating. Left front leg now resists passive motion slightly. Right front leg is entirely relaxed.

2.45—Front legs are also stiffened. Vasomotor reflex is disappearing. Knee jerks cannot be obtained.

3.00—Control blood pressure 50 mm.

3.15—Rapid cardiac failure.

The only finding at autopsy was oedema of the posterior parts of the lungs, which explained the change in the character of the respirations.

EXPERIMENT XXXVIII. *Failure to resuscitate the higher centres of the brain.* (Fig. 4.) Pug dog weighing 15.9 K.

10.45—Anesthetized with ether, changing at once to chloroform.

11.25—Control blood pressure 110 mm.

11.37 (52 minutes)—Chloroform forced.

11.37 $\frac{1}{2}$ —Control.

11.38 $\frac{1}{2}$ —Respiration stopped; cardiac failure followed quickly.

11.39 $\frac{1}{2}$ —No heart sounds.

11.40 $\frac{1}{2}$ —Control. Heart-beats recurred.

11.42—Final cardiac failure.

11.52 (10 minutes)—Started resuscitation. Infusion of Ringer's fluid through axillary artery, plus artificial respiration, was continued for one and a half minutes, and then 1 c.c. of adrenalin was administered. After waiting one and a half minutes, firm, slow massage of the chest was instituted. After a half minute the heart began to beat and the blood pressure rose to 250 mm. from which apex it quickly declined to 80 mm. in about three minutes, the pulse wave growing progressively larger. Drum stopped.

12.07—Control blood pressure 72 mm. This lowering of the pressure occurred synchronously with lowering the dog's feet.

12.11—First spontaneous respiration.

12.13—Control, showing respiratory curve of the blood pressure.

12.17—Control; clot.

12.21—Control blood pressure 90 mm. Artificial respiration stopped.

12.30—Control blood pressure 120 mm. Tracheal cannula out of position.

1.37—Control blood pressure 70 mm.

1.38—Control. Distinct reaction to burning of paw. Corneal reflexes are barely present. No anesthetic required.

2.00—Control blood pressure 84 mm. The dog was in such excellent shape that it was decided to allow him to recover. After the wounds were closed leaving the tracheal cannula in, he was removed from the table.

5.00 (5 hours)—Animal in good shape but unconscious. No ether had been required. Found dead the next morning.

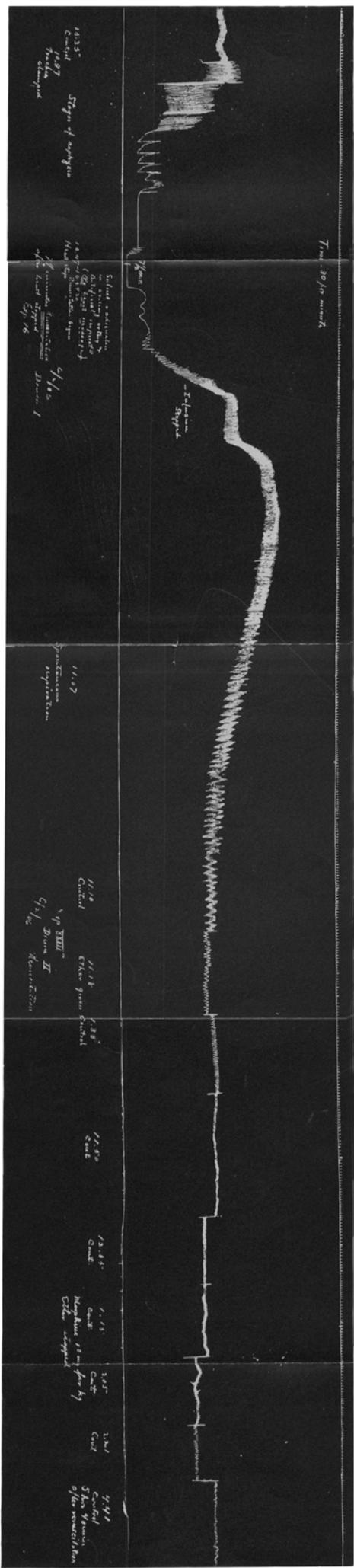
All the organs and tissues presented at autopsy evidence of infection with *B. aerogenes capsulatus*. There were no clots in the heart and no macroscopical lesions of the viscera other than the post mortem infection noted.

EXPERIMENT XLII. *Resuscitation of smaller part of brain after lapse of more time.* (Fig. 5.) Mongrel, 6.9 K., emaciated but healthy looking.

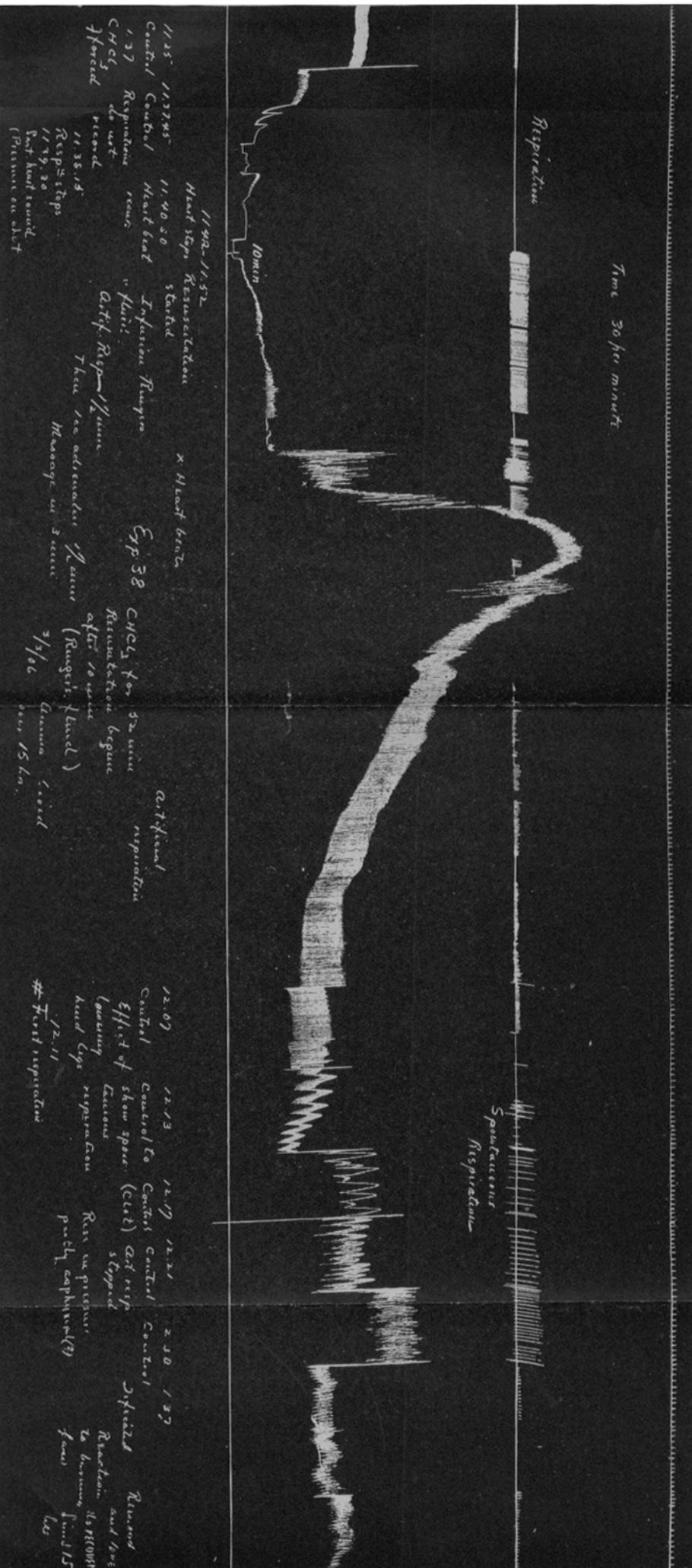
- 10.15—Anesthetized with chloroform.  
11.13—Chloroform forced (anesthesia during one hour).  
11.17½—Heart stopped before respiration according to manometer tracing.  
11.19—Heart-beats recurred for about ten seconds.  
11.20—Heart-beats recurred a second time, about twelve in all being recorded.  
11.20½—Heart sounds ceased and the pulse was not palpable.  
11.40½ (20 minutes)—Resuscitation started. Infusion of Ringer's fluid through axillary artery, plus 2 c.c. of solution of adrenalin chloride (1—1000), and artificial respiration were started simultaneously. Gentle pressure at intervals on the chest with the feet lowered was also used. After several minutes the axillary cannula pulled out and was refixed in the right carotid.  
11.43 (23 minutes)—Resuscitation was again attempted, artificial respiration having been kept up continuously. In about a minute the heart was noticed to be beating. The blood pressure curve rose rather slowly to a height of 110 mm. with a very irregular pulse wave.  
11.48—Respiration recurred.  
11.49—Control: blood pressure level has fallen to 50 mm.  
12.07—Control blood pressure 36 mm. Burning of paw evokes no reaction.  
12.20—Blood pressure 50 mm. Pulse wave much improved. Faint corneal reflex in right eye.  
2.00—Control blood pressure 54 mm. Respirations have become more normal in type, but the heart rate is distinctly lowered. Burning of paw evokes a very slight rising of blood pressure only. Knee jerks are present in both hind legs. Corneal reflex is present in the right eye, though feebler than normal; it is just perceptible and delayed in the left. Dog appears insensible to touch sensation, but burning anus causes spontaneous movements of the legs. The pupils do not react to light. The right pupil is more dilated than the left.  
4.00—Control, showing rise of pressure due to accidental asphyxia.  
4.30—Control blood pressure fallen to 28 mm.  
Ante-mortem examination was performed. No clot appeared in the heart and the viscera were apparently normal.
- EXPERIMENT XLVIII. *Partial resuscitation of a puppy thirty minutes after apparent death.* (Fig. 7.) Skye terrier puppy, three months old, weight 2 K.; anesthetized with ether. A full dose of hirudin—10 c.c. of a .4 of 1% solution—was given to obviate clotting.
- 2.55—Control blood pressure 104 mm. Chloroform exchanged for ether and forced.  
3.03½—Respiration stopped. Control.  
3.06—Heart sounds, then pulse, not perceptible.  
3.36 (30 minutes)—Began resuscitation. Infusion of Ringer's solution from the height of four feet, plus 2.5 c.c. of 1—1000 adrenalin through carotid artery, with artificial respiration and gentle heart massage, were started simultaneously. Heart began to beat in about two minutes. Infusion was stopped as soon as the blood pressure began to rise decidedly. In about three minutes the blood pressure reached a height of 94 mm., with a regular and rhythmic heartbeat of good strength.  
3.41—Drum stopped.  
3.50—Control blood pressure 102 mm.

## SUMMARY OF EXPERIMENTS

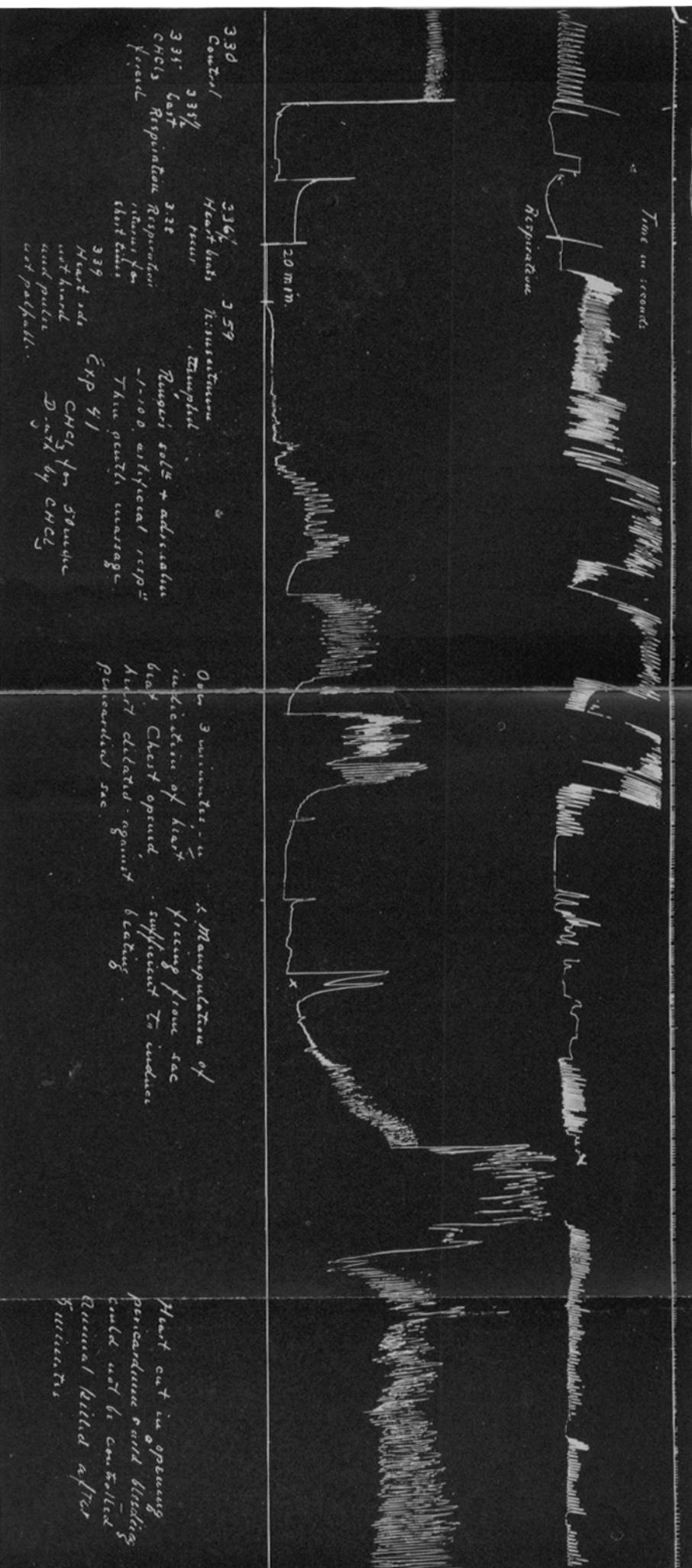
	Experiment		REMARKS	
	Mode of death	Heart Resp.	Duration of suspended animation (in minutes)	
1 Chloroform	2	—	1 hr.	Window in chest. Infusion very rapid.
2 Asphyxia.	5	—	7 min.	Grenhan; anesthesia.
3 Chloroform	51	—	10 min. 1 h. (killed)	Death due to accidental hemorrhage.
4 Chloroform	11	—	12 min.	Rapid infusion.
5 " "	21	—	12 min.	Repeated doses of adrenalin till ineffectual.
6 " "	25	—	2 min.	Infusion kept up too long.
7 " "	36	—	2 min.	Effort not uniform nor continuous.
8 " "	30	—	2 min.	Absolute failure.
9 Asphyxia.	32	—	Short	"
10 Asphyxia.	30	—	Short	"
11 " "	19	—	Short	"
12 " "	15	—	Delayed	"
13 Chloroform	113	—	Delayed	"
14 Chloroform	6	—	1 min.	Length of survival
15 Asphyxia.	17	—	1 min.	Complete return of circulation.
16 Asphyxia.	11	—	1 min.	Heart beat: Circulation not maintained.
17 Ether	10	—	1 min.	Respiratory recovery
18 Asphyxia.	17	—	1 min.	Vaso-motor recovery
19 Asphyxia.	7	—	1 min.	Approximate time of return of heart beat.
20 Ether	72	—	1 min.	Total duration of suspended respiration
21 " "	0	—	1 min.	Clot
22 " "	0	—	1 min.	Cardiac dilatation
23 Ether	5	—	1 min.	
24 Chloroform	9	—	1 min.	
25 Ether	10	—	1 min.	
26 Ether	6	—	1 min.	
27 Ether	15	—	1 min.	
28 " "	17	—	1 min.	
29 " "	24	—	1 min.	
30 Chloroform	15	—	1 min.	
31 Ether	64	—	1 min.	
32 " "	13	—	1 min.	
33 " "	5+	—	1 min.	
34 " "	52	—	1 min.	
35 " "	64	—	1 min.	
36 " "	10	—	1 min.	
37 " "	5	—	1 min.	
38 " "	74	—	1 min.	
39 " "	94	—	1 min.	
40 " at once	10	—	1 min.	
41 " once	124	—	1 min.	
42 " once	13	—	1 min.	
43 " once	20	—	1 min.	
44 " once	21	—	1 min.	
45 " once	20	—	1 min.	
46 Asphyxia	30	—	1 min.	
47 Ether	184	—	1 min.	
48 Asphyxia	214	—	1 min.	
49 Chloroform	10	—	1 min.	
50 " "	324	—	1 min.	
51 " "	61	—	1 min.	
52 " "	46	—	1 min.	
53 " "	35	—	1 min.	
54 " "	40	—	1 min.	
55 " "	362	—	1 min.	
56 " "	35	—	1 min.	
57 " "	304	—	1 min.	
58 " "	364	—	1 min.	
59 " "	37	—	1 min.	
60 " "	—	—	1 min.	

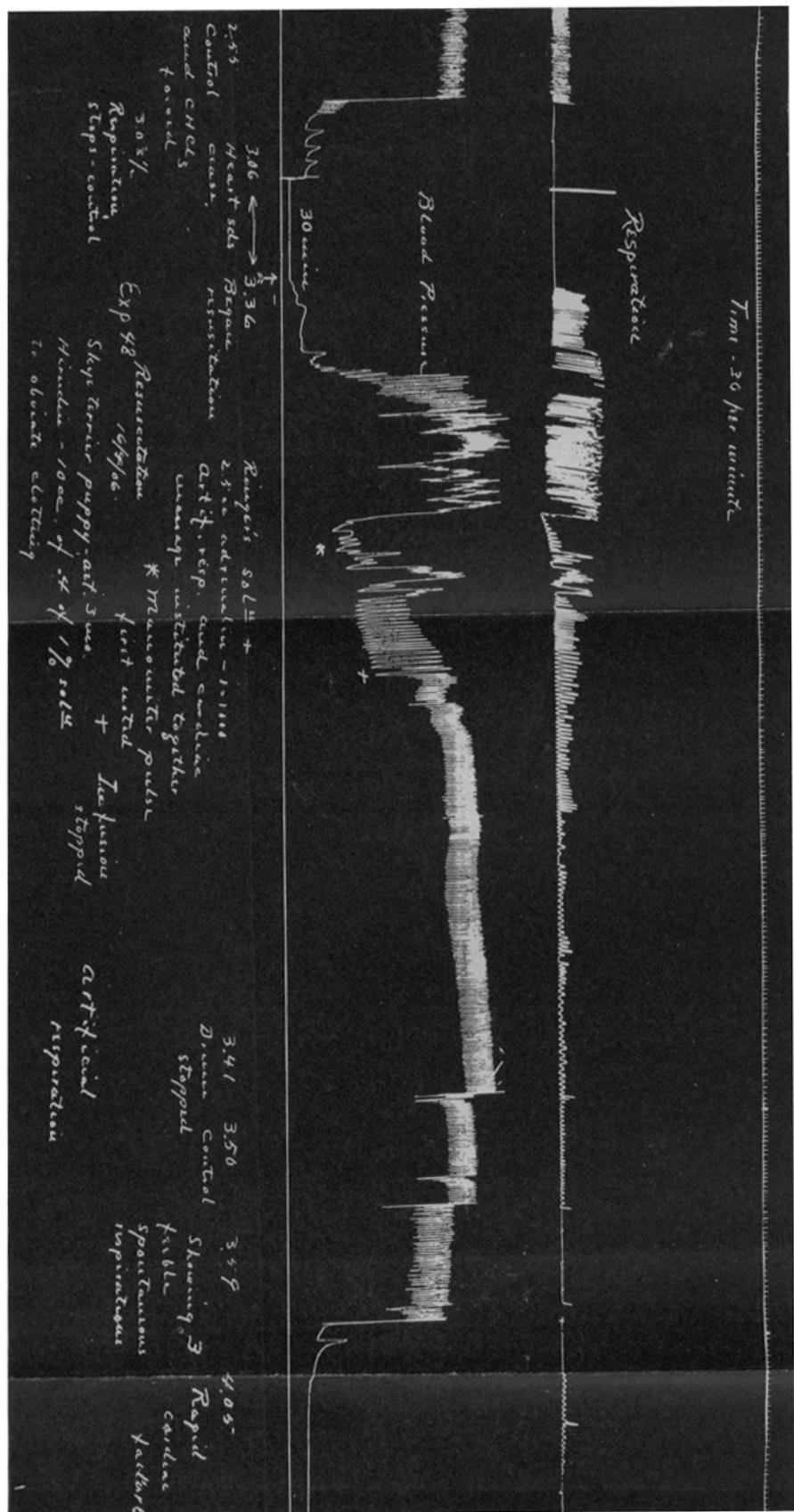


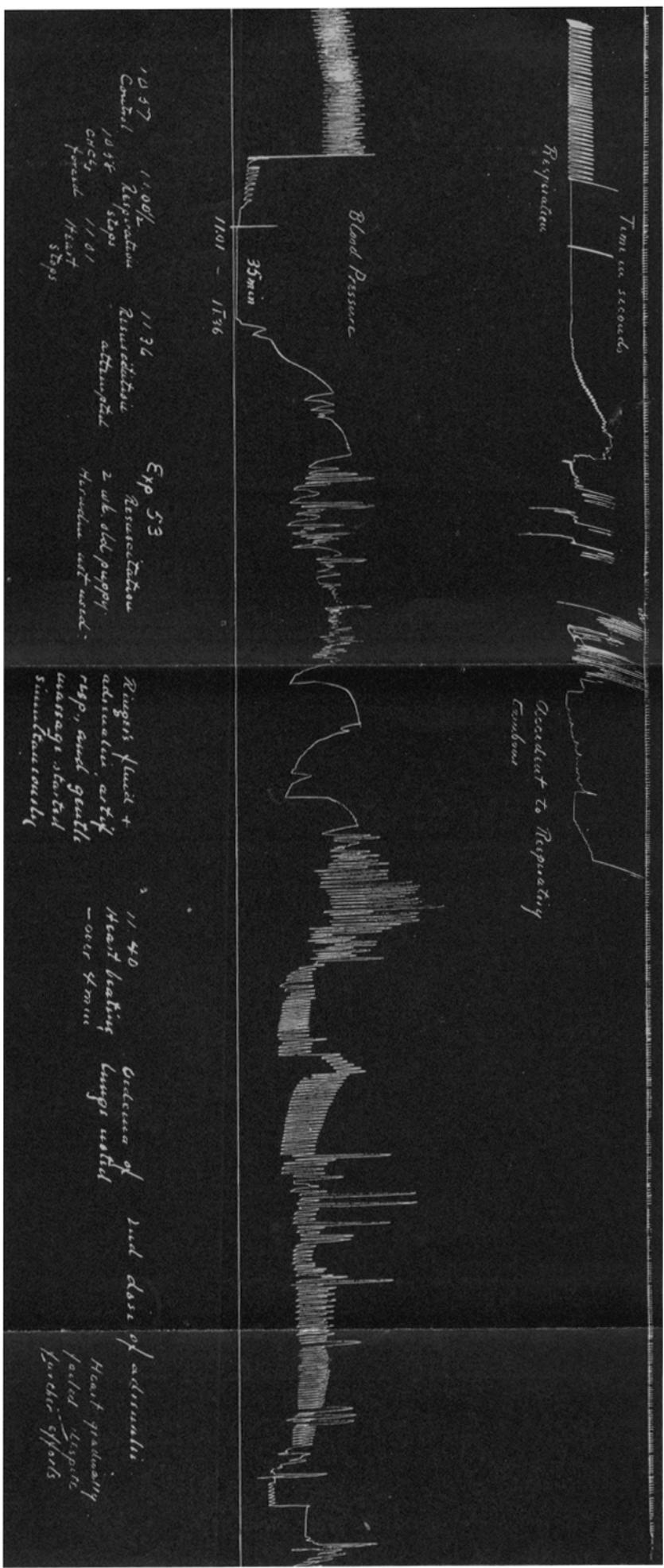












3.59—There are three feeble spontaneous respirations which the control shows. These occurred after several attempts to induce them by stopping artificial respirations, burning anus, and pulling on the tongue. Artificial respiration was continued, but the heart began at once to fail.

4.05—Heart stopped beating entirely.

Complete resuscitation within six minutes has been obtained in a number of experiments since the foregoing was sent to press. Later experiments have apparently established the unequal susceptibility for resuscitation of various parts of the brain.