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THE NEUROGENIC CONTROL OF THE CIRCULATION OF DOGS UNDER ETHER ANESTHESIA * †

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WHEN ether is administered to normal animals, various circulatory adjustments occur. In the dog, these changes affect the cardiac rate and output, the arterial blood pressure, the peripheral vascular tone, the circulating plasma volume, the volume of the spleen and probably the volume of various other organs. A knowledge of these responses is of interest in practical anesthesia and is of importance in investigations in which the nature and the magnitude of the variations imposed by the anesthetic must be considered. In the following discussion, the circulatory changes will be described in so far as the available data permit, and an attempt will be made to analyze the role of the autonomic nervous system in the production or regulation of these changes. Only two stages of anesthesia will be considered, that of excitement and that of surgical anesthesia, for the depth of anesthesia shifts so quickly in the dog that it is impractical to distinguish the first and fourth stages.

I. THE CIRCULATORY CHANGES

Cardiac Rate and Output.—Figures 1 and 2 show that the induction of ether anesthesia in dogs causes a temporary bradycardia. This cardiac slowing has been studied by Harris (1) and ourselves (2) and resembles that occasioned by the inhalation of other irritating vapors (3, 4, 5). The brief duration of excitement and the technical difficulties

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involved in the measurement of gaseous exchange through the lungs have prevented the study of cardiac output in this stage.

As the stage of excitement is passed and the stage of surgical anesthesia is entered (blood ether concentration 100 to 150 mg. per cent) the heart rate increases in the dog to between 160 and 230 beats per minute and remains high throughout the period of narcosis (2, 6, 7, 8, 9, 10). Under such conditions Blalock (11) measured an increase in cardiac output (Fick method) which varied between 7 and 180 per cent in different

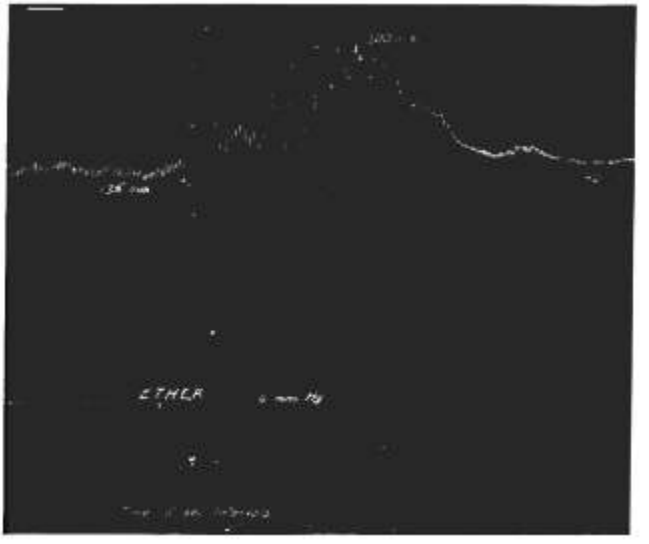

FIG. 1. The effect of ether inhalation upon the heart rate and blood pressure of a normal dog. Note the initial bradycardia and the rise in blood pressure followed by a tachycardia and a return of the blood pressure toward the preanesthetic level. Reprinted by courtesy of the American Journal of Physiology 133: 70, 1941.

animals. In a second series of 8 dogs this author reported that ether anesthesia increased the cardiac output between 4 and 44 per cent, the average being 20 per cent (12).

Blood Pressure.—In our experiments, the average mean arterial pressure in the femoral arteries of normal resting dogs is 120 mm. Hg (2). With the induction of ether anesthesia there is an immediate rise in pressure to between 180 and 220 mm. Hg (figs. 1 and 2). As the stage of surgical anesthesia is entered, the blood pressure returns toward the preanesthetic level and eventually is maintained during

one hour of anesthesia at 10 to 15 mm. Hg below the control value (2). Thus, the arterial pressure ordinarily ranges between 100 and 120 mm. Hg although there are wide variations both above and below the preanesthetic level. Blalock (11, 12) and Parkins (9) have reported that the arterial pressure is somewhat elevated under surgical ether, but in their studies the anesthesia was usually of short duration, and it has

FIG. 2. The effect of ether inhalation upon the heart rate and blood pressure of a dog six days after its spinal cord had been transected between T–11 and T–12. The response is about the same as that of the normal dog shown in figure 1 except that a longer time was required for the blood pressure to return to the control level. Reprinted by courtesy of the American Journal of Physiology 134: 65, 1941.

been our experience that the pressure is frequently slightly elevated during the first fifteen or twenty minutes of anesthesia.

The Peripheral Vascular Tone.—Very little data are available concerning the state of the peripheral vessels during the excitement stage. It is probable that in the splanchnic area the tone of the vessels is increased, as it is during fear, rage and pain (13). Support for this statement is furnished by the fact that the rise in arterial pressure in the excitement stage is abolished by interrupting the nerve supply to the

vessels of the viscera (14). The condition of vessels in other body areas during this period is not known.

Under surgical ether anesthesia the vascular tone in the visceral bed is either maintained or increased (2, 14, 15). Of the abdominal organs, the kidney and the spleen have been most intensively studied. The many reports concerning renal denervation hyperemia must be regarded as evidence of renal vasoconstriction induced by anesthesia (16), for it is known that, in the absence of general anesthesia, denervation of the dog's kidney is without effect upon the renal blood flow (17). Contraction of the spleen in dogs under ether has been observed in exteriorized spleens (18, 19) and under the fluoroscope after the organ has been outlined with radio-opaque clips (20). Indirect evidence of splenic contraction is furnished by the fact that ether causes a rise in the hematocrit value (21, 22, 23) which is much in excess of that which can be accounted for by a loss of fluid from the circulating blood. After splenectomy, the rise in the red cell volume is of a degree consistent with the decrease in plasma volume.

In contrast with the visceral vessels, it is generally agreed by physiologists and neurosurgeons that the cerebral vessels are dilated and that the brain substance is excessively vascular under ether (24, 25, 26, 27). This dilatation of cerebral vessels has been observed directly through a pial window in cats by Forbes, Wolff, and Cobb (25) and by Finesinger and Cobb (26). That dilatation also occurs in the superficial vessels is suggested by the flushing of the skin and the rise in surface temperature (28, 29, 30). The fact that the skin temperature of the hind legs of etherized dogs does not drop abruptly when these animals are exposed to cold indicates that under ether vasoconstriction is absent in the superficial vessels of the lower extremities (31). A similar dilatation occurs in the deep vessels of the lower extremity during ether anesthesia as evidenced by an increase in limb volume (15), and by an increase in the blood flow in the femoral artery (32).

The Circulating Plasma Volume.—The work of a number of investigators indicates that the administration of ether decreases the circulating plasma volume. In 1905 Burton-Opitz (33) measured an increased blood viscosity under ether anesthesia and concluded that this anesthetic produces hemoconcentration. A similar conclusion was reached by Carlson and Luckhard (34) who found an increase in the blood osmotic pressure. On the basis of exsanguination experiments, Mann (35) reported that less than the estimated normal quantity of blood was recovered from etherized dogs. Other investigators have noted an increase in cell volume (36) and in blood solids (37). Measurements of plasma volume by Gregersen's blue dye (T-1824) technic (38) show that ether generally causes a reduction of 10 to 12 per cent in the plasma volume of dogs (23, 39, fig. 3). A decreased plasma volume in 16 etherized patients was reported by Stewart (40) who used the same dye technic. Conley's (41) failure to find that ether anesthesia

decreases the plasma volume of cats suggests that the effect of this anesthetic upon the volume of the circulating plasma may vary in different species.

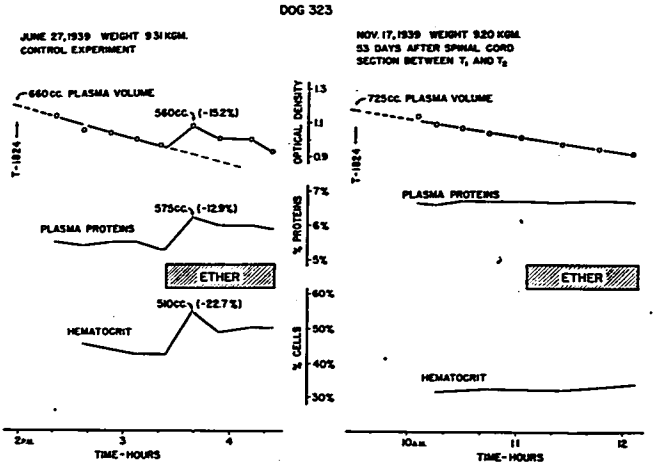

FIG. 3. The effect of one hour of ether anesthesia on a dog before and after spinal cord section between T–1 and T–2. In each case the upper curve represents the disappearance from the plasma of the blue dye T–1824 used for the measurement of the plasma volume. Concentration (in terms of optical density) has been plotted against time. In the middle curve the plasma protein level has been plotted against time, and in the lower curve the hematocrit value. In the control experiment, there is a rise in concentration of serum dye and plasma protein during ether. This has been taken as evidence of a loss of fluid from the circulation. The rise in hematocrit is proportionately greater than the rise of the other substances (spleen intact). Fifty-three days after high spinal cord transection ether causes no change in the level of either dye, plasma protein or hematocrit.

II. THE ROLE OF THE AUTONOMIC NERVOUS SYSTEM IN THE PRODUCTION OF THE CIRCULATORY CHANGES

Cardiac Rate and Output.—The bradycardia shown during the excitement stage is a vagal effect, for it is absent in vagotomized dogs and in animals which have received a dose of atropine sufficient to paralyze the vagi (1, 2). This vagal slowing is not the result of a carotid sinus reflex secondary to the rise in arterial pressure since it occurs in high spinal and sympathectomized dogs in which the arterial pressure falls during this stage. The slow heart rate can be accounted for as a reflex vagal stimulation produced by the irritant action of the anesthetic upon the respiratory mucosa (1, 2, 3, 4, 5).

The increased heart rate which is present during the surgical stage of ether anesthesia is attributed by Samaan (10) to three factors: (*a*)

paresis of the vagal inhibitory mechanism, (*b*) augmentation of the cardio-accelerator mechanism, and (*c*) liberation of certain sympathicomimetic substances.

That central vagal activity is inhibited by surgical ether anesthesia is shown by the fact that the heart rate increases to about 135 beats per minute (the denervated heart rate) in sympathectomized dogs (2). In such animals the mechanisms controlling the sympathetic cardio-accelerators and the liberation of adrenalin have been eliminated. Under these conditions the anesthesia, by removing vagal restraint, effects a functional cardiac denervation so that the heart rate stabilizes at the rate shown by a chronically sympathectomized, vagotomized animal. Further evidence of the inhibition of vagal restraint is furnished by the observation that the injection of adrenalin intravenously in normal dogs under surgical ether fails to produce the classical bradycardia which occurs in unanesthetized animals (10). Likewise, the finding that cervical vagal section under ether is not accompanied by any appreciable change in cardiac rate indicates that the heart is not inhibited by the vagi (10).

Ether may also produce a paralyzing effect on the vagal endings (42). According to Kobacker and Rigler (43) the cat's heart fails to respond to peripheral vagus stimulation when the animal is completely anesthetized with ether. Shafer, Underwood and Gaynor (44) found that peripheral vagus stimulation produced less effect upon the heart of etherized dogs than it did in the same animals in the absence of ether (decerebrated).

Augmentation of the sympathetic cardioaccelerator impulses is revealed by the finding that the usual degree of cardiac acceleration does not occur after complete sympathectomy (2, 10). The experiments in which the spinal cord of dogs was transected at different levels (fig. 4) show that the magnitude of the cardiac acceleration is dependent upon the quantity of intact sympathetic outflow to the heart. These sympathetic cardio-accelerator impulses arise in suprasegmental centers, for the isolated spinal cord centers cannot produce the customary tachycardia. Thus, in dogs with cervical cord transection (as in sympathectomized dogs) the heart rate increases under ether only to the extent that can be accounted for by the removal of vagal restraint (14). These results also indicate that the cardio-accelerator fibers which are reported to be present in cat and dog vagi (4, 45, 46, 47) are not excited by ether anesthesia (2).

It is doubtful that the adrenal secretion which is said to occur during ether anesthesia (48) plays an important role in the changes in heart rate. The administration of ether to 2 dogs with inactivated adrenals (2) caused the usual cardiac acceleration in the surgical stage indicating that neural impulses alone can account for the increase in heart rate. That the denervated adrenal gland is not affected directly by ether is

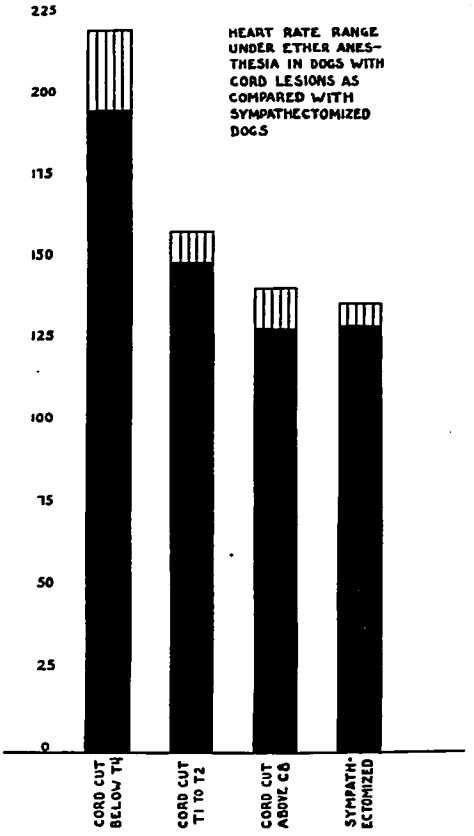

FIG. 4. The heart rate range (lined areas) during ether anesthesia in dogs with spinal cord transection at different levels as compared with sympathectomized dogs. The figures at the left are beats per minute. With cord transections below T–4 the heart rate range under ether is the same as that of normal dogs. With the cord cut above the eighth cervical segment it may be seen that the heart rate under ether is practically the same as that of a sympathectomized dog. This latter rate approximates that of a chronically sympathectomized, vagotomized dog (i.e., a completely denervated heart).

demonstrated by the observation that the inhalation of ether by vagotomized, sympathectomized dogs produces no change in heart rate (2).

Unfortunately, we have no measurements of cardiac output in etherized sympathectomized and etherized spinal cord dogs, so it is impossible to evaluate the role of the autonomic nervous system in the regulation of this important function. Nevertheless, the fact that the blood pressure variations are independent of cardiac rate and are directly related to the amount of intact sympathetic nervous outflow to the periphery suggests that the observed effects result from alterations in peripheral resistance rather than from primary changes in cardiac output.

The Blood Pressure and Peripheral Vascular Tone.—In sympathectomized dogs (fig. 5) and in dogs with the spinal cord sectioned above

FIG. 5. The effect of ether inhalation upon the heart rate and blood pressure in a totally sympathectomized dog. The immediate fall in arterial pressure is drastic, and, if the ether cone is not removed during the critical periods of asystole (as at the points marked "off"), the animal may die. Throughout surgical anesthesia, the blood pressure remains low. Reprinted by courtesy of the American Journal of Physiology 133: 70, 1941.

T6 the arterial blood pressure falls during the induction of ether anesthesia and remains low throughout the period of narcosis (2, 14). These responses are present whether the vagi are intact or whether they have been previously sectioned to eliminate changes in cardiac rate (figs. 6 and 7). Under surgical anesthesia the blood pressure of these animals ranges from 25 to 60 mm. Hg and is directly related to the blood ether concentration. With spinal cord sections below the tenth thoracic segment, however, the normal blood pressure responses are obtained (fig. 2). From these data, it is apparent that central sympathetic connections must be intact as low as the tenth thoracic segment for the normal blood pressure response to occur in both the excitement and the surgical

.stages of ether anesthesia. The sympathetic outflow from the sixth to the eleventh thoracic segments appears to be of especial importance. Hence, one might infer that during ether anesthesia the vascular tone in the area supplied by this outflow, i.e., the viscera, must be maintained or increased in the normal dog. Yet the splanchnics are not the sole mediators of the constrictor impulses, for dogs with bilateral subdiaphragmatic splanchnic nerve section show the same type of response as normal dogs.

FIG. 6. The effect of ether inhalation upon the heart rate and blood pressure of a vagotomized, sympathectomized dog. The blood pressure falls without any significant cardiac arrhythmia. The high heart rate shown by this animal is related to the acute section of the left vagus nerve. Twenty-four hours after completing the cardiac denervation, the heart rate of these dogs ranged between 128 and 135 beats per minute. Reprinted by courtesy of the American Journal of Physiology 133: 70, 1941.

The inability of the isolated spinal cord to maintain the blood pressure during ether anesthesia indicates that under this anesthetic the sympathetic nervous impulses are not developed in the spinal cord centers, but originate in the suprasegmental region. The same mechanism appears to control splenic contraction. Evidence for this statement is furnished by the finding that the administration of ether to normal dogs produces an increase in the hematocrit value which can be accounted for in part by splenic contraction (23, 39), whereas under the same conditions the hematocrit readings of high spinal dogs undergo little or no change (fig. 3).

The central sympathetic activity is not the result of reflex stimulation mediated by way of the buffer nerves, for the normal responses occur in dogs in which both carotid sinuses are completely denervated and both vagi (aortic depressor fibers) are sectioned (fig. 8). This sympathetic activity is probably produced by the direct action of ether upon the vasomotor center, although Pilcher and Sollman (49) state that any stimulation of the vasomotor center during ether anesthesia is secondary to anoxemia. We should like to point out that the marked differences between sympathectomized and spinal dogs on the one hand

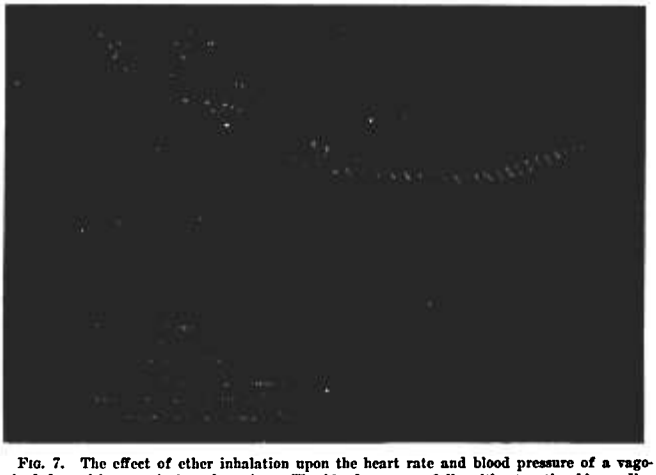

FIG. 7. The effect of ether inhalation upon the heart rate and blood pressure of a vagotomized dog with a cervical cord section. The blood pressure falls without noticeable cardiac slowing and remains low. Compare with figures 4 and 7. Reprinted by courtesy of the American Journal of Physiology 134: 65, 1941.

and normal dogs on the other hand exist in the presence of adequate arterial oxygen saturation.

That the sympathetic vasoconstrictor mechanisms are not maximally discharging during ether anesthesia is shown by the fact that a substantial rise in arterial pressure may be obtained when the pressure in the carotid sinuses is lowered by occlusion of the carotid arteries (fig. 9).

The cause of the fall in arterial pressure which occurs when ether is administered to high spinal or to sympathectomized dogs is uncertain. We believe that this effect of ether is the result of changes in the peripheral vessels rather than primary changes in cardiac output. Evidence supporting this view is furnished by the finding that the blood pressure falls in those etherized spinal dogs (T6) in which the nervous

FIG. 8. The effect of ether inhalation upon the blood pressure of a dog with the circulatory buffer nerves eliminated by denervation of the carotid sinuses and section of the vagi (to eliminate the aortic depressor fibers). Such preparations show a chronic hypertension which, in our experiments, has ranged from 150 to 200 mm. Hg. The response to ether resembles that observed in the normal dog, but the pressure variations are both extensive and erratic.

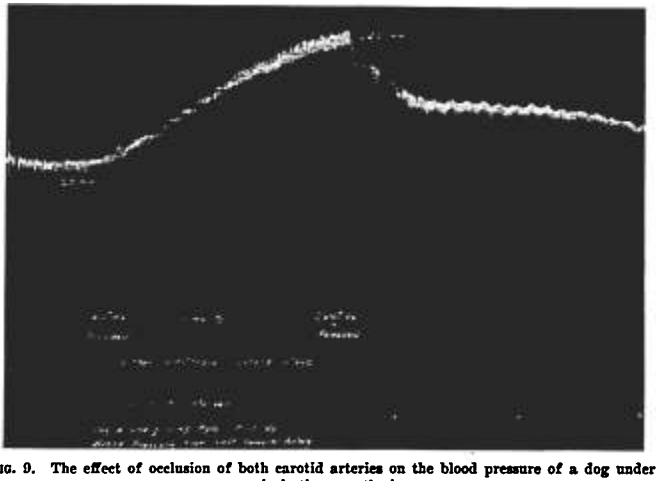

FIG. 9. The effect of occlusion of both carotid arteries on the blood pressure of a dog under surgical ether anesthesia.

supply to the heart has not been disturbed. Moreover, the blood pressure falls when ether is administered to animals in which the changes in heart rate have been eliminated by double vagotomy and complete sympathectomy. Since any sympathetic or vagal dilator pathways are interrupted by sympathectomy and vagotomy, it is probable that the fall in pressure results either from dorsal root dilator activity or from a direct dilating action of ether on the arterioles. In the normal dog such responses would be masked by the action of sympathetic impulses originating rostral to the thoracic spinal cord.

The cause of the vasodilatation occurring under ether in the vessels of the lower extremity and of the brain has not been studied.

The Plasma Volume.—It is difficult to explain the decrease in plasma volume which usually occurs in etherized normal dogs. The fluid loss is not the result of an increased salivary flow, for the decrease in plasma volume occurs in dogs which have received a dose of atropine sufficient to prevent salivary secretion (52). The plasma deficit cannot be accounted for by fluid loss from the lungs, since Barbour and Bourne (37) report that water loss through this channel is not excessive during ether anesthesia. The diminished urinary output under ether (50) indicates that renal excretion cannot account for the plasma depletion. Electrolyte studies combined with measurement of the extracellular fluid compartment show that no general decrease in interstitial fluid takes place during ether anesthesia (23, 51). Neither the degree of anoxia nor the magnitude of the reduction in alkali reserve bears any relation to the degree of plasma volume decrease (52). It is possible that fluid escapes into the extracellular extravascular spaces as a result of certain hemodynamic adjustments. Some support for this view is furnished by the finding that a few normal dogs in which ether anesthesia produces little or no change in plasma volume also show a level of mean pressure below 80 mm. Hg during the period of narcosis. Further indication that mechanical factors are involved in the fluid shift is revealed by the observation that in sympathectomized and high spinal dogs, which exhibit a marked fall in pressure, ether causes no change in plasma volume (53; also see figs. 3, 10). A possible explanation of these findings is that in the normal dog fluid escapes from the capillaries in certain areas of vasodilatation where capillary hydrostatic pressure is increased. Under conditions in which the general level of arterial pressure is maintained, Landis (54) has measured such a local increase in capillary hydrostatic pressure as a result of arteriolar dilatation and has even observed a transudation of fluid into the tissue spaces. In sympathectomized and high spinal dogs and in the normal dogs exhibiting a pressure below 80 mm. Hg the head of pressure in the capillaries in any areas of dilatation which may exist would be lower and the force promoting fluid transudation would be diminished. It must be stressed that this explanation does not consider other important factors such as capillary and interstitial osmotic pressure and capillary permeability. Likewise,

it must not be inferred that changes in plasma volume are directly dependent upon changes in mean arterial pressure.

SUMMARY

1. The available data concerning various circulatory changes in etherized dogs are summarized.

2. The induction of ether anesthesia in dogs causes a temporary bradycardia and a rise in arterial pressure. The cardiac slowing is a vagal effect, abolished by vagotomy and produced reflexly by the irri-

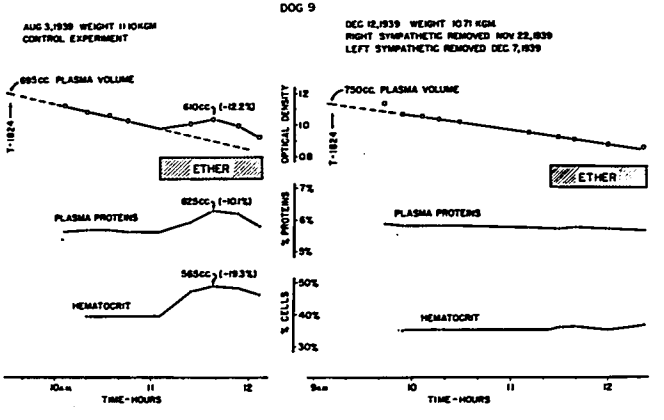

FIG. 10. The effect of ether inhalation for one hour upon a dog before and after complete sympathectomy. For explanation of the method of charting, see figure 3. The control experiment shows the customary decrease in plasma volume under ether (as measured by the rise in serum concentration of injected dye), the rise in plasma proteins and the excessive rise in the hematocrit value. After complete sympathectomy, ether causes little or no change in any of these values.

tant action of ether on the respiratory mucosa. The rise in pressure is caused by visceral vasoconstriction resulting from central sympathetic impulses which can be eliminated by spinal cord transection above the sixth thoracic segment.

3. In the surgical stage there is a marked increase in heart rate due to an inhibition of vagal restraint and to central activation of sympathetic cardioaccelerators. The cardiac output is increased. The blood pressure is maintained at or slightly below the preanesthetic value by the activity of the sympathetic nervous system, for in the absence of the sympathetics, ether reduces the blood pressure to dangerously low levels. Although the cardiac output is increased, the maintenance of the

blood pressure depends more upon the tone of the peripheral vascular circuit. The nervous impulses responsible for maintaining the vascular tone in the visceral bed are suprasegmental in origin, for their effects are absent in dogs with high spinal cord section. These impulses likewise are not dependent on buffer nerve reflexes because they persist when the carotid sinus and aortic depressor nerves have been cut. Like the blood vessels of the viscera, the contraction of the spleen is dependent on central sympathetic stimuli, for contraction fails to occur in dogs with a high cord section. Other evidence indicates that vasodilatation occurs in the brain, the superficial vessels and the deep vessels of the lower extremity.

4. Ether ordinarily causes a reduction of 10 to 12 per cent in the plasma volume of normal dogs. This loss of plasma is not seen in sympathectomized dogs or in dogs with a high spinal cord section in which arterial pressure falls. It is suggested that the transudation of fluid is associated with a local increase in capillary hydrostatic pressure secondary to arteriolar dilatation.

REFERENCES

1. Harris, A. S.: Cardio-inhibitory and Vasomotor Reflexes from Nose and Throat, Ann. Otol., Rhin. & Laryng. 48: 311–323 (June) 1939.
2. McAllister, F. F., and Root, W. S.: Circulatory Responses of Normal and Sympathectomized Dogs to Ether Anesthesia, Am. J. Physiol. 133: 70–78 (May) 1941.
3. Kretschmer, F.: Über Reflexes von den Nasenschleimhaut auf Athmung und Kreislauf, Sitz. d. k. Akad. d.Wiss. Wien. 62: 147, 1870.
4. Francois-Franck, C.: Effets des excitations des nerfs sensibles sur le coeur, la respiration et la circulation arterielle, Trav. de Laborat. de Marey 2: 221, 1876.
5. Brodie, T. G., and Russell, A. E.: On Reflex Cardiac Inhibition, Am. J. Physiol. 26: 92, 1900.
6. Cluzet, J., and Petzetakis: Étude électrocardiographique expérimentale sur les principaux modes d'anesthesie générale, Compt. rend. Soc. de biol. 76: 86–88 (Jan.) 1914.
7. Cannon, W. B., and Lewis, J. T.: Physiological Maximum Heart Rate as an Artefact, Am. J. Physiol. 82: 67–74 (Sept.) 1927.
8. Wachsmuth, W., and Eismayer, G.: Über den Einfluss operativer Eingriffe auf die Herztätigkeit, Deutsche Ztschr. f. Chir. 209: 145–156, 1928.
9. Parkins, W. M.: Observations on Direct Intra-arterial Determination of Blood Pressure in Trained Unanesthetized Dogs, Am. J. Physiol. 107: 518–525 (Feb.) 1934.
10. Samaan, A.: Au sujet du méchanisme de l'action de l'adrénaline, de l'atropine et de l'ether sur la fréquence cardiaque, Compt. rend. Soc. de biol. 115: 1751–1756, 1934; Effect of Adrenaline, Atropine and Ether on Heart Rate of Normal Dogs and of Animals Deprived of Different Parts of Autonomic Nervous System, Arch. internat. de pharmacodyn. et de thérap. 50: 101–127 (Mar. 15) 1935.
11. Blalock, A.: Cardiac Output in Dog during Ether Anesthesia; Effect of Ether Anesthesia on Cardiac Output, Arch. Surg. 14: 732–751 (Mar.) 1927.
12. Blalock, A.: Effects of Ether, Chloroform, and Ethyl Chloride Anaesthetics on Minute Cardiac Output and Blood Pressure; Experimental Study, Surg., Gynec. & Obst. 46: 72–78 (Jan.) 1928.
13. Cannon, W. B.: Bodily Changes in Pain, Hunger, Fear and Rage, 2nd Ed., New York, D. Appleton Co., 1929.
14. Root, W. S., and McAllister, F. F.: Circulatory Responses of Chronic Spinal Dogs to Ether Anesthesia, Am. J. Physiol. 134: 65–70 (Aug.) 1941.
15. Derouaux, J.: Nouvelles recherches sur l'action physiologique de l'éther sulfurique, Arch. internat. de pharmacodyn. et de therap. 19: 63–95, 1909.
16. Smith, H.: Physiology of the Renal Circulation, The Harvey Lectures 35: 166, 1939–40.

17. Rhoads, C. P.; Alving, A. S.; Hiller, A., and Van Slyke, D. D.: Functional Effect of Explanting One Kidney and Removing Other, Am. J. Physiol. 109: 329–335 (Aug.) 1934.
18. Barcroft, J., and Florey, H.: Effects of Exercise on Vascular Conditions in Spleen and Colon, J. Physiol. 68: 181–189 (Oct.) 1929.
19. Barcroft, J., and Rothschild, P.: Effect of Certain Anaesthetics on Volume of Exteriorized Spleen, Arch. internat. de pharmacodyn. et de thérap. 38: 569–576, 1930.
20. Hausner, E.; Essex, H. E., and Mann, F. C.: Roentgenologic Observations of Spleen of Dog under Ether, Sodium Amytal, Pentobarbital Sodium and Pentothal Sodium Anesthesia, Am. J. Physiol. 121: 387–391 (Feb.) 1938.
21. Searles, P. W., and Essex, H. E.: Changes in Blood in Course of Ether Anesthesia and Sodium Amytal Anesthesia, Proc. Staff Meet., Mayo Clin. 11: 481–483 (July 29) 1936.
22. Essex, H. E.; Seeley, S. F.; Higgins, G. M., and Mann, F. C.: Effect of Ether Anesthesia and Amytal Anesthesia on Erythrocytic Findings in Control and Splenectomized Dogs, Proc. Soc. Exper. Biol. & Med. 35: 154–156 (Oct.) 1936.
23. McAllister, F. F.: Effect of Ether Anesthesia on Volume of Plasma and Extracellular Fluid, Am. J. Physiol. 124: 391–397 (Nov.) 1938.
24. Roy, C. S., and Sherrington, C. S.: On the Regulation of the Blood-Supply of the Brain, J. Physiol. 11: 85–108, 1890.
25. Forbes, H. S.; Wolff, H. G., and Cobb, S.: Cerebral Circulation; Action of Histamine, Am. J. Physiol. 89: 266–272 (July) 1929.
26. Finesinger, J. E., and Cobb, S.: Cerebral Circulation; Action of Narcotic Drugs on Pial Vessels, J. Pharmacol. & Exper. Therap. 53: 1–33 (Jan.) 1935.
27. Schmidt, C.: The Circulation of the Brain and Spinal Cord, Assoc. Res. in Nerv. & Ment. Dis. 18: 1938.
28. Ipsen, J.: Les artères et l'anesthésie, Acta Chir. Scandinav. 65: 487–536, 1929.
29. Scott, W. J. M., and Morton, J. J.: Obliteration of Vasoconstrictor Gradient in Extremities under Nitrous Oxide-Oxygen, Ether, and Tribromethyl Alcohol Anesthesias, Proc. Soc. Exper. Biol. & Med. 27: 945–949 (June) 1930.
30. White; J. C.: The Autonomic Nervous System, 1st Ed., New York, The Macmillan Co., 1935.
31. Sheard, C.; Rynearson, E. H., and Craig, W. M.: Effects of Environmental Temperature, Anesthesia and Lumbar Sympathetic Ganglionectomy on Temperatures of Extremities of Animals, J. Clin. Investigation 11: 183–193 (Jan.) 1932.
32. Baldes, E. J.; Herrick, J. F., and Essex, H. E.: Measurement of Flow of Blood and Effects of Anesthesia and Lumbar Sympathectomy, Proc. Staff Meet., Mayo Clin. 7: 535–536 (Sept. 14) 1932.
33. Burton-Opitz, R.: The Changes in the Viscosity of the Blood during Narcosis, J. Physiol. 32: 385–389, 1905.
34. Carlson, A. J., and Luckhard, A. B.: The Increase in the Osmotic Concentration of the Blood during Ether and Chloroform Anesthesia, Am. J. Physiol. 21: 162–168, 1908.
35. Mann, F. C.: Some Bodily Changes during Anesthesia; an Experimental Study, J. A. M. A. 67: 172–175, 1916.
36. Epstein, A. A.: Concerning the Effects of Anesthesia on the Blood Volume and Its Relation to the Production of Shock, Am. J. Surg., Q. Suppl. Anesth. 31: 115–120, 1917.
37. Barbour, H. G., and Bourne, W.: Heat Regulation and Water Exchange; Influence of Ether in Dogs, Am. J. Physiol. 67: 399–410 (Jan.) 1923.
38. Gregersen, M. I.: Analysis of Colorimetric Methods in Relation to Plasma Volume Determinations, J. Lab. & Clin. Med. 23: 423, 1938; Macleod's Physiology in Modern Medicine, Bard, 18th Ed., St. Louis, C. V. Mosby Co., 1938, p. 911.
39. Bollman, J. L.; Svirbely, J. L., and Mann, F. C.: Blood Concentration Influenced by Ether and Amytal Anesthesia, Surgery 4: 881–886 (Dec.) 1938.
40. Stewart, J. D., and Rourke, G. M.: Changes in Blood and Interstitial Fluid Resulting from Surgical Operation and Ether Anesthesia, J. Clin. Investigation 17: 413–416 (July) 1938.
41. Conley, C. L.: Effect of Ether Anesthesia on Plasma Volume of Cats, Am. J. Physiol. 132: 796–800 (Apr.) 1941.
42. Ruttgers, P.: Über selektive Wirkung von Giften, insobesondere von Narkotika, auf die erregenden und hemmenden Mechanismen des Herzens, und über die Funktion der Scheidewandnerven, Ztschr. f. Biol. 67: 1–34, 1916.
43. Kobacker, J. L., and Rigler, R.: Behavior of Extracardiac Nerves of Cat under Ether; Potential Source of Error, J. Pharmacol. & Exper. Therap. 37: 161–175 (Oct.) 1929.
44. Shafer, G. D.; Underwood, F. J., and Gaynor, E. P.: Action of Amytal in Impairing Vagus Cardiac Inhibitory Effects, and of Ether in Increasing the Respiratory Rate after Its Depression by Amytal, Am. J. Physiol. 91: 461–466 (Jan.) 1930.

45. Moore, R. M., and Cannon, W. B.: Heart Rate of Unanesthetized Normal, Vagotomized, and Sympathectomized Cats as Affected by Atropine and Ergotoxine, Am. J. Physiol. 94: 201–208 (July) 1930.
46. Jourdan, F., and Nowak, S. J. G.: Les fibres cardio-accélératrices dans le nerf pneumogastrique du chien; leur origine et leur trajet, Compt. rend. Soc. de biol. 117: 234–238, 1934.
47. Brouha, L., and Nowak, S. J. G.: Role of Vagus in Cardio-accelerator Action of Atropine in Sympathectomized Dogs, J. Physiol. 95: 439–453 (May 15) 1939.
48. Elliott, T. R.: The Control of the Suprarenal Glands by the Splanchnic Nerves, J. Physiol. 44: 374–409, 1912.
49. Pilcher, J. D., and Sollmann, T.: Studies on the Vasomotor Center. XV. The Action of Ether, J. Pharmacol. & Exper. Therap. 6: 401–404, 1915.
50. Hawk, P. B.: Urine Formation during Ether Anesthesia, Arch. Int. Med. 8: 177–182, 1911.
51. Root, W. S.; McAllister, F. F.; Oster, R. H., and Solarz, S. D.: Effect of Ether Anesthesia upon Certain Blood Electrolytes, Am. J. Physiol. 131: 449–454 (Dec.) 1940.
52. Root, W. S., and McAllister, F. F.: Unpublished observations.
53. Root, W. S., and McAllister, F. F.: Some Effects of Ether Anesthesia on Spinal Dogs, Am. J. Physiol. 126: 613, 1939; The Importance of the Sympathetic Nervous System in Maintaining the Circulation during Ether Anesthesia, Am. J. Physiol. 129: 449, 1940.
54. Landis, E. M.: Micro-injection Studies of Capillary Blood Pressure in Human Skin, Heart 15: 209–228 (May) 1930.

MEETING OF THE AMERICAN SOCIETY OF ANESTHETISTS, INC.

SQUIBB BUILDING, 745 FIFTH AVENUE, NEW YORK CITY

October 8, 1942—7:30 P.M.

1. Anesthesia in Esophageal Surgery. 40 minutes.
   By A. Ralph Wilkins, Phm.B., M.B., Assistant Anesthetist, Toronto General Hospital and the University of Toronto, Toronto, Canada.
   Discussion to be opened by Milton C. Peterson, M.D., Director of Anesthesia, New York Post-Graduate Medical School and Hospital, New York, N. Y.
2. Aberrant Thymic Tissue in the Lung with Bronchial Compression and Sudden Death During Anesthesia. 20 minutes.
   By Robert L. Patterson, M.D., Director of Anesthesia, Allegheny General Hospital, Pittsburgh, Pa., and Elwyn L. Heller, M.D., Department of Pathology, Allegheny General Hospital, Pittsburgh, Pa.
   Discussion to be opened by Karl S. Russell, M.D., Chief of the Anesthesia Department, West Jersey Homeopathic Hospital, Camden, N. J.
3. Continuous Caudal Anesthesia During Labor and Delivery. 55 minutes.
   By Robert A. Hingson, M.D., Past Assistant Surgeon, U. S. P. H. S., Chief of the Department of Anesthesia, U. S. Marine Hospital, Staten Island, New York.
   Discussion to be opened by Waldo B. Edwards, M.D., Past Assistant Surgeon, U. S. P. H. S., Staten Island, New York.