Central Nervous Actions of Halothane Affecting the Systemic Circulation

Henry L. Price, M.D., Harry W. Linde, Ph.D., Howard T. Morse, M.D.

PREVIOUS studies of mechanisms underlying the hemodynamic actions of halothane are difficult to interpret, either because the drug was not confined to the area studied or because of failure to show that the local actions which were observed could produce systemic circulatory changes. In the experiments to be reported halothane was confined to the cephalic circulation while hemodynamic functions in the remainder of the body were measured. The results obtained are interpreted to mean that halothane has central actions which can inhibit efferent sympathetic nervous activity, weaken barostatic reflexes, and reduce cardiac rate, contractile force and arterial blood pressure.

Methods

Adult mongrel dogs weighing 15–20 kg. were studied. Anesthesia was produced by administering chloralose intravenously (50–80 mg./kg.), following which the trachea was intubated and artificial respiration instituted using a Bird respirator supplied with oxygen. The rate of respiration and tidal volume remained constant throughout each study. Arterial blood P_{CO_2} was measured repeatedly in six animals by means of an electrode (Instrumentation Laboratories, Model No. 113) and found to average 38 mm. of mercury (range 33–42) during the period of study.

In eleven dogs the chest was incised in the midline and the sternum split. Umbilical tape was passed around the brachiocephalic and left subclavian arteries. The pericardium was opened over the right ventricle, and tape was passed around the superior vena cava below the point of entry of the azygos vein. A strain gauge arch was attached to the right ventricle with silk sutures and its feet extended 50 per cent from their initial length. The right axillary artery was prepared for retrograde perfusion by isolation, cannulation, and ligation of the omocervical artery. The carotid sheath was opened below the thyroid cartilage and the vagosympathetic trunk isolated. Tape was placed around the common carotid arteries. Arterial pressure was measured in the cephalic portion of the preparation through a catheter passed into the superior thyroid artery and advanced until its tip lay within 1 cm. of the carotid sinus. Heparin (75 mg.) was injected intravenously. The right atrial appendage was incised and a 1 cm. bore glass cannula was passed into the superior vena cava and held by a purse string suture in the atrium. An incision was made in the right chest at the level of the vena cava, through which a plastic cannula was passed and connected to the vena caval cannula. Figure 1 illustrates schematically the perfusion system and the relevant vascular anatomy.

Cephalic bypass was begun with retrograde perfusion through an axillary artery at the rate of 160–180 ml./minute* utilizing a bubble oxygenator supplied with oxygen (2 liters/minute) and a Sigmamotor pump. The

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* The perfusion rate chosen was arrived at by process of elimination. Rates below 140 ml./minute frequently resulted in hyperventilation, suggesting cerebral anoxia, while rates above 200 ml./minute caused relative arterial hypertension in the cephalic circulation, leading to troublesome leakage of blood out of the perfusion system into the systemic circulation, and occasionally, to cerebral edema.

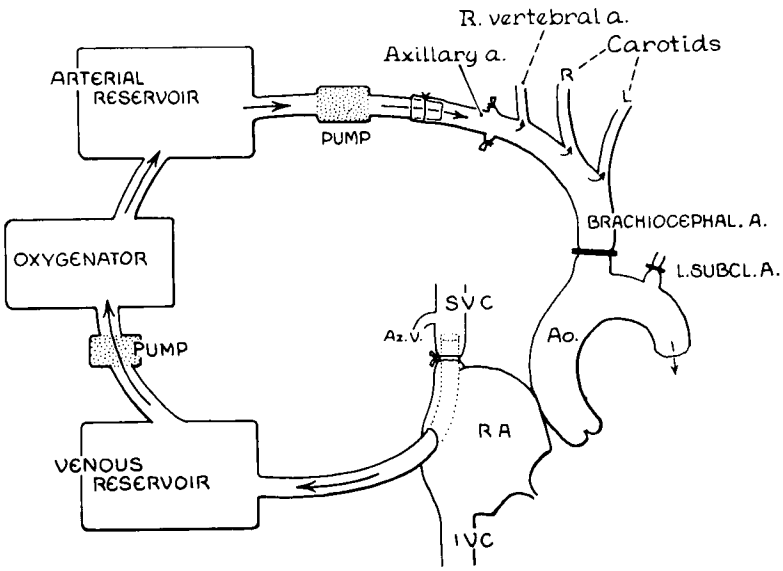

FIG. 1. Schematic diagram of head perfusion technique. A., a. = artery; V. = vein; Ao. = aorta; Az. = azygos; I.V.C. = inferior vena cava; R.A. = right atrium; S.V.C. = superior vena cava; R., L. = right, left; — = clamp. Direction of flow is shown by arrows.

pump was filled with blood drawn from a heparinized donor. After leaving the pump, blood passed through coils of polyethylene tubing immersed in a water bath maintained at 37° C. Venous blood returned by gravity from an accurately controlled level above the vena cava. After blood balance was established the brachiocephalic and left subclavian arteries were clamped. Total cephalic bypass was then accomplished by tightening the tape around the vena cava and cannula.

Halothane was introduced via the oxygenator when its actions were to be studied. Initially the concentration given was 5 per cent; over the course of three minutes it was reduced in steps to the desired concentration (either 1 or 2 per cent). Expired air was withdrawn continuously at a rate of 0.6 liter/minute from the upper third of the endotracheal tube and analyzed for halothane by an infrared analyzer. End-expired concentrations as small as 0.05 per cent (v/v) could be detected with confidence, and in none of the head perfusion studies reported were end-expired concentrations of halothane as great as this present.

Femoral arterial pressure was measured in all, and central venous pressure in three cases. All pressures were transduced using Statham P23D or P23B strain gauges, and were recorded on a Grass polygraph.

In two animals isolation of the carotid sinus was performed by ligating the superior thyroid, external carotid, internal carotid, ascending pharyngeal, lingual, occipital and several smaller arterial branches. The common carotid artery was tied and the occlusion bypassed by the polyethylene tubing. The bypass contained a T-tube leading to a 250 ml. reservoir pressure bottle containing 100 ml. of heparinized blood. Pressure within the reservoir could be measured and altered as desired.

The carotid arteries were occluded in the neck by means of umbilical tapes or by bulldog clamps applied without obvious traction upon either the carotid sinus or its adjacent structures. The occlusion was maintained until the reflex response was maximal (20–30 seconds). When the cephalic circulation was perfused with blood containing halothane, both mean and pulse pressures in the common carotid artery were reduced, presumably by pharmacologic actions of the drug on vascular smooth muscle. For this reason it was necessary, in order to test the reflex, to increase flow during the administration of halothane until the mean carotid pressure equalled that present during the control observations. No effective method of controlling the pulse pressure was found. As an alternative, the cephalic arterial pressure was damped mechani-

TABLE 1. Systemic Circulatory Effects of Administering Halothane to the Head

Number	AP_0	Δ	HR_0	Δ	CF_0	Δ	Time	% Halo.
62-23 (B)	73	−23	166	−14	20	−7	14	1.0
62-25 (B)	92	−19	159	−55	25	−10	20	1.0
62-26 (B)	75	−15	182	−26	23	−4	11	1.0
62-27 (B)	60	−20	144	−20	19	−4	10	1.0
62-05	110	0	200	−4	19	−5	19	1.0
62-30	90	−25	146	−16	27	−7	10	1.0
62-34	65	−10	184	−56	28	−10	10	1.0
Means	80.7	−16.0	169	−27.3	23.0	−6.7	13.4	1.0
% Mean Change		−19.8		−16.1		−29.1		
62-10	70	−25	180	−40	23	−5	15	2.0
62-12	92	−52	160	−52	30	−8	8	2.0
62-13	105	−61	164	−50	29	−9	15	2.0
Means	89.0	−46.0	168	−47.3	27.3	−7.3	12.7	2.0
% Mean Change		−51.7		−28.2		−26.8		

AP = systemic arterial pressure in mm. Hg; HR = cardiac rate in beats per minute; CF = contractile force in arbitrary units; 0 = control observations; Δ = change from control; Time = duration of exposure to halothane in minutes; % Halo. = final concentration (v/v) of halothane delivered to the oxygenator in volumes per cent; (B) = animals blocked with mepivacaine.

cally in all carotid occlusion studies until the pulse pressure in the absence of halothane was less than 5 mm. of mercury.

In four animals the right carotid sinus was perfused from the common carotid artery by means of the pump-oxygenator. The external carotid artery was cannulated well above the carotid sinus and connected to a Starling resistance. All other visible branches of the artery were ligated. By this method halothane could be delivered to the carotid sinus without entering the systemic circulation, and the pressure within the sinus could be set at any desired level by adjusting the resistance.

Electrical activity in the cervical vagus nerves was blocked reversibly with cold in three cases by contact with notched silver bars whose distal ends were immersed in ice and brine.

In four animals the first eight intercostal nerves were blocked bilaterally under direct vision just after the animal was put on bypass, 0.5 ml. of 1.5 per cent mepivacaine being deposited adjacent to each nerve. In addition, the skin edges of the neck incision were infiltrated with 0.5 per cent mepivacaine and the exposed surface in the neck was bathed with the local anesthetic applied on gauze sponges. An additional animal was given cyclopropane via the pump-oxygenator during complete bypass.

Results

Central Hemodynamic Actions of Halothane. Typical results are shown in table 1. Administration of one per cent halothane to the oxygenator was followed shortly by decreases in mean arterial blood pressure, heart rate and contractile force averaging, respectively, 20, 16 and 29 per cent. Pulse pressure (not shown) was also reduced. Hemodynamic alterations closely paralleled, or soon followed, loss of the corneal reflex.

Administration of 2 per cent halothane resulted in more pronounced decreases in arterial pressure and heart rate, although with the limited number of observations available only the decrease in blood pressure could be shown statistically to be larger than at the lower concentration of halothane. Central venous pressure was measured in two animals, and increased 1 mm. of mercury in both cases during the action of halothane.

Analgesic (in contrast to autonomic) ac-

tions of halothane could conceivably have accounted for the results observed. Consequently, local anesthesia and intercostal nerve block were produced in four animals as described in the methods. No direct hemodynamic effect of this procedure was evident, and blocked animals responded to halothane in the same manner as did the others. Five per cent cyclopropane was administered (via the pump-oxygenator), to another animal; neither the measured hemodynamic variables nor their responses to carotid occlusion were affected. In two additional animals doses of chloralose approximating 0.3 g., or 20 per cent of the initial dose, failed to affect either the hemodynamic variables which were measured or their responses to carotid occlusion. Apparently analgesic actions of halothane do not explain its hemodynamic effects under the conditions of this study.

Most of the blood exposed to halothane entered the cerebral circulation by way of the common carotid arteries; therefore it was necessary to determine whether actions on carotid sinus baroreceptors or chemoreceptors could have caused the hemodynamic actions which were observed. For this purpose one common carotid artery was isolated from the systemic circulation and perfused with blood containing halothane as described in the methods; the opposite side was denervated. These perfusions had no measurable hemodynamic action nor any consistent effect upon carotid sinus reflexes. Hence, the hemodynamic actions of halothane which were observed apparently did not result either from chemo-

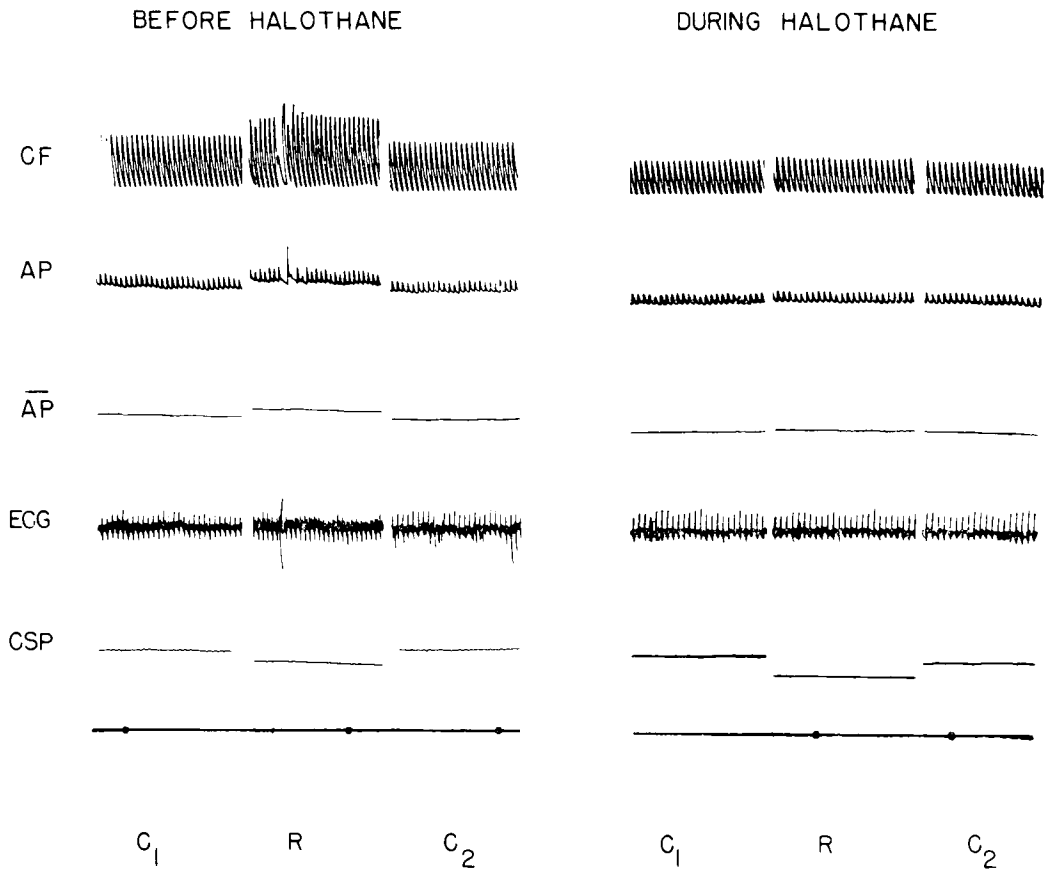

FIG. 2. Hemodynamic effects of carotid occlusion before and during the administration of halothane. CF = contractile force; AP = systemic arterial pressure; $\overline{AP}$ = mean systemic arterial pressure; ECG = electrocardiogram; CSP = carotid sinus pressure; Lowest line = base line; C_1 and C_2 controls before and after carotid occlusion; R = response during carotid occlusion.

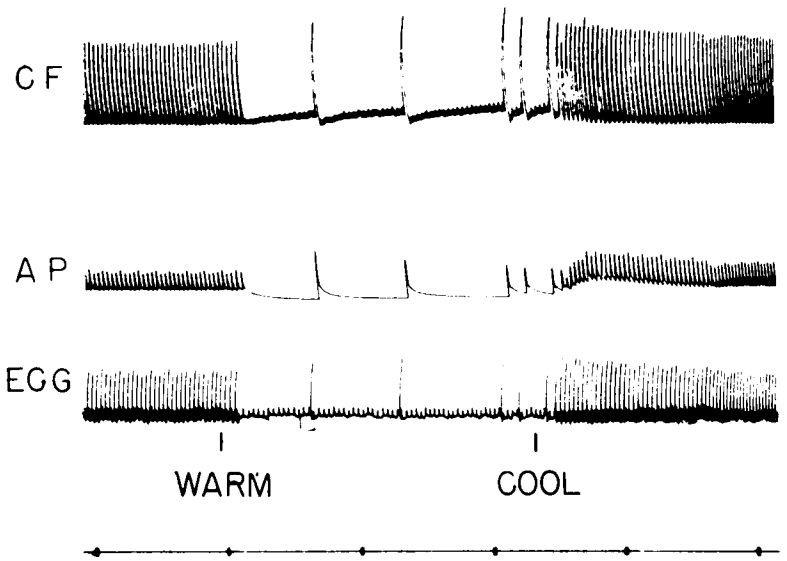

FIG. 3. Hemodynamic effects of vagal cooling and warming during the administration of halothane. CF = contractile force; AP = systemic arterial pressure; ECG = electrocardiogram; Warm = stop vagal cooling; Cool = start vagal cooling; Time marks 15 seconds.

receptor desensitization or from an increase in baroceptor sensitivity.

Central Actions Affecting Barostatic Reflexes. Administration of 1 per cent halothane to the head in ten experiments was promptly followed by a reduction in the response of systemic arterial pressure to changes in intrasinusal pressure, whether produced by carotid occlusion or by sinus distention. On average, the response was decreased by 59 per cent, with a standard deviation (S.D.) of 19 per cent. Responses of contractile force to the same stimulus were more greatly reduced (average 75 per cent), but because of large individual differences in the response (S.D. = 31 per cent) it was not possible to determine whether there was any quantitative distinction between the behavior of pressure and force. Reduced intrasinusal pressure had no consistent effect on heart rate either before or during halothane administration, but in animals which initially exhibited tachycardia during carotid occlusion the administration of halothane reduced or abolished the response. A typical result is shown in figure 2.

Since only three of the ten animals studied recovered its initial responsiveness following the administration of halothane, it was reasonable to suspect that spontaneous deterioration of the preparation contributed to the results obtained. Attempts to assess the importance of spontaneous changes were of three kinds. First, the trends of arterial pressure responses to carotid occlusion just prior to the administration of halothane were, on average, time-independent. Second, it was noted that reduction in the carotid sinus response closely paralleled that in the corneal reflex. Third, when only the results of experiments in which the initial response was fully regained following the discontinuation of halothane were considered, the mean decrease in the response (52 per cent) during halothane administration was statistically the same as in the group as a whole. It should be mentioned that failure to return the preparation to its initial condition following the administration of halothane resulted at least in part from inefficient removal of the drug by the pump-oxygenator system; in preparations that did recover completely one or more hours were required for recovery following removal of halothane from the gas mixture supplied. Many preparations deteriorated spontaneously in an equal period.

Blockade of the Vagus Nerves. In two animals blockade of the vagus nerves had no consistent effect on blood pressure or contractile force, either before or during the administration of halothane, but the *sinus*

rate increased an average of 40/minute when the vagii were cooled during halothane administration. This increase, although statistically significant ($P < 0.05$), was only 40 per cent of the decrease in heart rate which attended the administration of the drug. Consequently the heart rate was still reduced during halothane breathing even in the presence of vagal blockade. These data are summarized in table 2. In one animal the *ventricular* rate was markedly reduced during the administration of halothane in consequence of a high degree of A-V blockade. In this animal vagal cooling abolished the block completely and reversibly on each of three occasions, while warming re-instituted it (fig. 3). Discontinuing halothane terminated the blockade. In a fourth animal (D62-30) the vagii were sectioned in the neck before halothane was added to the blood perfusing the head. This procedure did not appreciably modify either the hemodynamic actions of halothane or its effect on the response to changes in intrasinusal pressures, but the degree of bradycardia observed was less than the average.

Administration of Halothane to the Entire Body. Four animals inhaled halothane in oxygen at the tension required to increase the end-expired concentration to one per cent (v/v). One carotid sinus was prepared for inflation with static pressure and the other side denervated. During the administration of halothane the average response of systemic arterial pressure to standard changes in intrasinusal pressure was reduced to one-half that observed in the same animals during the control period, and the change in the response was indistinguishable statistically from that observed when halothane was confined to the head.

Discussion

In the absence of actions demonstrable by perfusing the carotid sinus with blood containing halothane, the findings suggest that this anesthetic produces hemodynamic effects by acting within the central nervous system. Since all of the animals studied had surgical wounds, these actions could have been analgesic in nature. However, despite repeated

TABLE 2. Systemic Circulatory Effects of Vagal Blockade Before and During Administration of Halothane to the Head

Number	AP_0	Δ	HR_0	Δ	CF_0	Δ
			Control [On Bypass]			
62-12-I	91	0	182	0	33	0
62-12-II	92	0	166	0	33	0
62-13-I	105	+10	164	+17	31	0
62-13-II	105	0	168	+24	30	+1
Means	98	+3	170	+10	32	0
			During Halothane [2%]			
62-12-I	35	+4	107	+24	26	+1
62-12-II	32	+3	54	+64	27	−5
62-13-I	42	0	100	+27	24	+1
62-13-II	40	+4	96	+44	22	+2
Means	37	+3	89	+40*	24	0
Mean Differences (From Control)	−61	0	−81	+30	−8	0

AP = mean systemic arterial pressure in millimeters of mercury; HR = cardiac rate in beats per minute; CF = contractile force in arbitrary units; 0 = control observations; Δ = changes observed during vagal cooling; I, II = experiment number. Data with the same number are directly comparable; * = significant effect of vagal blockade.

efforts, clear evidence for analgesic hemodynamic actions of halothane could not be shown.

The four principal results obtained in the present study by means of head perfusion deserve individual consideration.

Reduced Contractile Force. The significance of this measurement is not clear. Even though attempts are routinely made (by stretching the myocardium between the feet of the recording gauge) to eliminate the influence of varying amounts of ventricular filling, it can be shown[1,2] that the results obtained do in fact depend upon filling, and upon other extraneous factors as well. A moderate reduction in blood volume, in our experience, depresses "contractile force," although it ought (reflexly) to increase it. The converse is also true. Ventricular extrasystoles can cause monumental increases in measured "contractile force," although these changes seem more likely to result from large excursions near the gauge than from any general change in contractility. For these reasons we regard measurements obtained with strain gauge arches as indicating the performance of ventricular tissue adjacent to their feet under the existing conditions (including extracardiac ones), rather than the functional ability of the heart as a whole.

As the result of these observations a minimum of two interpretations of the reduction in contractile force caused by halothane are possible: reduced contractility *per se*, or reduced venous return. The present experiments cannot distinguish between them, but neither can earlier studies which were considered by their authors to provide indisputable evidence for direct myocardial actions of halothane.[3,4] It would seem that, if direct myocardial depression by halothane is as important hemodynamically as has been urged, conspicuous venous hypertension ought to appear during the action of the drug. Yet, venous pressure increases either not at all[5,6] or by only a few millimeters of mercury, and the increases reported are often within the range which can be expected on the basis of cardiac slowing (*e.g.*, by vagal stimulation) alone. This could mean either that direct myocardial depression by halothane is absolutely unimportant, or that it is not the only important action of the drug which can affect blood pressure. The second alternative is considered far more likely.

In addition, the present study shows that it is incorrect to view acute experiments with strain gauge arches as adequate for measuring, or even establishing the existence of, direct myocardial actions of halothane. In our animals contractile force was diminished by nearly 30 per cent when halothane was admitted to the central nervous system but denied access to the heart.

Reduced Arterial Blood Pressure. Cardiac output could not, for technical reasons, be accurately measured in these studies; hence it is not known whether reduced blood flow or diminished vascular resistance was primarily responsible for arterial hypotension. However, cardiac output could be estimated roughly from the pulse pressure by the method of Hamilton and Remington.[7] These estimates indicated that blood flow was reduced slightly less than blood pressure, which suggests in turn that the principal cause of diminished arterial pressure was a reduction in cardiac output. Total peripheral resistance apparently decreased to a lesser extent.

Since halothane reached only controlling (as opposed to "working") circulatory elements in these studies, it is apparent that central actions caused hypotension. Furthermore the separation of cephalic and systemic circulations means that systemic circulatory actions of halothane must have been neuronally rather than humorally mediated. Since vagal blockade had no typical effect either on arterial blood pressure or contractile force it is unlikely that increased vagal activity caused the hemodynamic actions which were observed. Therefore they indicate a reduction in sympathetic nervous activity.

Reduced Cardiac Rate. Cardiac slowing occurred consistently and was conspicuous in one case. In this animal halothane caused a high degree of atrioventricular block which was relieved by vagal cooling. Vagal cooling consistently elevated cardiac rate during the administration of halothane, but not in the absence of the drug. This indicates a central nervous action of halothane which elevates vagal "tone." However, in no case did vagal cooling increase the heart rate to the

level observed prior to the administration of halothane. Apparently † central vagal actions of halothane do not entirely explain the typical response of heart rate to the administration of the drug in this study. This again suggests a reduction in sympathetic nervous outflow.

Reduced Reflex Response to Changes in Carotid Sinus Pressure. Of all the results obtained, this one most clearly points toward a direct action of halothane on central nervous cardiovascular representations. Unlike the effects of halothane on contractile force, blood pressure and heart rate, the ability of this drug to suppress both pressor and depressor reflexes appears impossible to attribute to analgesic actions.

The principal importance of halothane's ability to suppress barostatic reflexes is that by so doing it permits direct actions on cardiac and vascular smooth muscle to exert their full hemodynamic consequences, unhindered by compensatory reactions. Probably it is this central nervous action of halothane which permits authors to conclude, for example, that: "The reduction in cardiac output and blood pressure is attributed primarily to the direct depressant effect of halothane on the myocardium."[9] Surely, the resemblance between patients in heart failure and patients anesthetized with halothane is anything but striking, and differences in the response to cardiac incompetence in the two populations undoubtedly result more from autonomic actions of halothane than from other causes. This is not to say that direct peripheral circulatory actions of halothane are unimportant. They are important, but only because paralysis of normal homeostatic mechanisms by halothane permits them to occur.

The findings of an equal reduction of the reflex in intact and head-perfused animals was unexpected. Since halothane reduces the responsiveness both of cardiac and of vascular smooth muscle to the sympathetic mediator,[7, 8] it was expected that the reflex response would be affected more in intact than in head-perfused animals. Since it was not, it appears either that these actions are not limiting in the overall response, or that the head-perfused animals were more sensitive to the actions of halothane than were the others.

As the result of these experiments, we view the central nervous action of halothane on the circulation as consisting of two main parts: sympatholytic; and "permissive," or due to weakening or abolition of homeostatic reflexes. It is not presently known how these actions are related, although undoubtedly they are. Studies which explicitly deny such actions[10, 11] argue either illogically or from indirect evidence. In addition, there is evidence for a central action which increases vagal "tone."

The effect of halothane on the circulation now appears principally as a mixture of central actions on nervous tissue and peripheral actions on cardiac and smooth muscle. We believe that it is the first of these which permits the others to attain hemodynamic significance.

Summary and Conclusions

In 10 mongrel dogs halothane was administered to the head alone, while hemodynamic changes in the remainder of the animal were observed. Administration of one per cent halothane was followed by loss of the corneal reflex, decreased arterial mean and pulse pressures, reduced heart rate, diminished cardiac contractile force, and a reduced hemodynamic response to carotid sinus occlusion. None of these changes resulted from direct actions of halothane on the heart, on vascular smooth muscle, or on ganglia below the level of circulatory isolation. Since the only effective connections between head and trunk consisted of somatic and autonomic nerves, the central autonomic actions of halothane must have consisted of increased parasympathetic nervous activity, reduced sympathetic nervous activity, or both. In the present study, the circulatory changes observed were attributable more to decreased sympathetic than to increased parasympathetic discharge. The central actions of halothane appear important hemodynamically in two ways: Directly, by reducing sympathetic and increasing parasympathetic "tone"; indirectly by weakening homeostatic mechanisms, thereby permitting direct actions of the drug on cardiac and vas-

† Some caution is necessary in the interpretation of these results since the canine cervical vagus contains a relatively small number of sympathetic fibers which, of course, would also be blocked by vagal cooling.

cular smooth muscle to occur unhindered by normal compensatory reactions.

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STEROID ANESTHESIA Hydroxydion anesthesia was used for 213 patients undergoing caesarean section. Thrombophlebitis was seen in one patient only and was believed to be due to faulty technique. Maternal circulation and respiration remained constant. There seemed to be minimal transmission through the placenta. Even in patients with preoperative fetal distress, spontaneous respiration occurred in the baby immdiately following delivery. (*Dimpfl, J.: Experiences with Steroid Anesthesia in 213 Caesarean Sections, Der Anaesthesist 12: 182 (June) 1963.*)

OBSTETRIC CARDIAC ARREST Over a recent five-year period, 10 patients on a large obstetrics-gynecology service developed cardiac arrest. This was an incidence of 1 : 7270 in obstetric patients, and 1 : 840 in gynecologic patients. Hypoxia during induction of anesthesia, high spinal anesthesia, and blood loss were frequently a factor in these cases. Necessity of early diagnosis and rapid effective therapy of this catastrophe is emphasized. A training program in the management of cardiac arrest should be mandatory for every obstetrician-gynecologist. (*Cavanaugh, D., Decenzo, J. A., and Ferguson, J. H.: Cardiac Arrest and the Obstetrician-Gynecologist, Obstet. Gynec. 22: 56 (July) 1963.*)

HALOTHANE Halothane has been used for almost three years in deliveries with favorable results. Oxytocin caused prompt uterine contractions. Tocometric measurements were made in the immediate post-partum period comparing halothane and ether anesthesia. During halothane anesthesia, slight uterine contractions were seen but none was observed with ether anesthesia of the same depth. Oxytocin caused a more marked effect with halothane than with ether. (*Uter, F.: Halothane Anesthesia in Obstetrics; Letter to the Editor, Der Anaesthesist 12: 161 (May) 1963.*)