Intracranial Pressure and Internal Carotid Blood Flow During Halothane Anesthesia in the Dog

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THE study of cerebrovascular hemodynamics during anesthesia and surgery is of basic importance. However, the available information on the specific effects of the various anesthetic agents on cerebral blood flow, cerebrovascular resistance or cerebral metabolic rate is incomplete, or as in the case of halothane anesthesia, virtually nonexistent. Yet a recent report [1] describes an increase in intracranial pressure during its administration. Such an increase implies modifications in cerebral blood flow and vascular resistance which may have clinical significance, but are incompletely defined at this time.

Contemporary study of cerebral blood flow and vascular resistance is largely based on application of the Fick principle by the Kety and Schmidt technique of nitrous oxide administration and its various modifications.[2] Unfortunately, this technique is not practical for continuous measurements. As a practical compromise, continuous recording of blood flow and vascular resistance in one internal carotid artery was used as a guide to changes occurring in the cerebral circulation as a whole. Thus, the left internal carotid blood flow was studied with a square-wave electromagnetic flow meter.[3-5] Halothane was administered and its effect on the internal carotid blood flow and vascular resistance was correlated with simultaneous changes in intracranial pressure, arterial and central venous pressures. These results were considered indicative of cerebrovascular hemodynamics under halothane anesthesia.

Method

Sixteen mongrel dogs, with an average weight of 14 kg., were studied. Anesthesia was induced with intravenous thiopental sodium, 20 mg./kg. Endotracheal intubation was per-

formed and muscular paralysis maintained with 10 mg. of succinylcholine administered intravenously every 20–30 minutes. A Mark 8 Bird respirator, adjusted to deliver 100 per cent O_2, 200 ml./kg./minute with a frequency of 10 respirations per minute, was used for ventilation, and halothane was delivered through a noncalibrated vaporizer. (Arbitrarily, halothane anesthesia was considered "deep" when the frequency in the fronto-occipital leads [EEG] and the mean blood pressure were 30 per cent below the control value.)

Pressure transducers (Statham) were connected through plastic catheters to the following: abdominal aorta, cisterna magna, pleural cavity and superior vena cava. These transducers were calibrated with standard mercury and water manometers before each experiment. Electrodes for lead 2 of the ECG and fronto-occipital EEG leads were applied. Blood flow determinations were made with an Avionic Series 6,000 electromagnetic blood flow meter [2] and recorded with the other parameters in a Sanborn 150 recording system. Internal carotid vascular resistance was determined as the ratio of mean blood pressure in millimeters of mercury to internal carotid blood flow in milliliters/minute.

Arterial blood samples of 4–6 ml. were taken for P_{O_2}, pH and P_{CO_2} determinations. The total amount of blood withdrawn was close to 50 ml. for each experiment, and was not replaced. An Instrumenation Laboratory Series 105 polarograph was used for these studies, utilizing Clark and Severinghaus electrodes for P_{O_2} and P_{CO_2}, respectively.

Surgical Preparation. The left femoral artery was dissected free and cannulated with a plastic catheter, which was advanced to the abdominal aorta. Following left lateral dissection of the neck, the maxillary vein was cannulated and a catheter advanced downward to the superior vena cava. The left common carotid artery and the bifurcation were exposed before ligation of the external carotid and occipital arteries. Then a 2–3 mm. flow-meter

probe was placed in the internal carotid artery. Next, a 19 B-D spinal needle was inserted in the cisterna magna. Finally, an intercostal space was punctured with a 14 B-D needle and a plastic catheter placed to determine intrathoracic pressure. Calibration of the probe was done by actual bleeding after completion of the experiment. Values varied by ±4 per cent. *"Occluded zero"* was used throughout the experiment and only controls in which this "value" was steady (±5 per cent), are listed. Control determinations were those in which stabilizations of internal carotid blood flow, mean blood pressure and superior vena cava pressure were obtained for a minimum of 20 minutes. Halothane was administered for various lengths of time and the recovery period observed. Only experiments in which arterial P_{CO_2} was kept the same, or a maximum of minus 5 mm. of mercury from control to the end of halothane administration, were used in compilation of data (7 out of 16).

The following observations are described: (1) control determinations as a function of time, (2) halothane administration, and (3) the effects of variations in P_{CO_2}.

Results

The control values for the various parameters are listed in table 1. These values stabilized within ±8 per cent during periods up to 45 minutes for the same arterial P_{CO_2}.

Intravenous administration of succinylcholine, 10 mg. every 20–30 minutes, had a transient effect on most parameters lasting a maximum of three minutes.

Figures 1 and 2 and table 1 show the control values and their modifications by halothane. There was a significant 56.4 per cent ($P <$ .001) decrease in the internal carotid vascular resistance. There was a decrease of 40.7 per cent in mean blood pressure without significant increase in the internal carotid blood flow (26.8 per cent). Modifications in the vascular resistance were similar during both deep and very deep (greatest cardiovascular depression) halothane anesthesia. Internal carotid blood flow increased during light anesthesia (lesser cardiovascular depression) and was close to control value with a reduction of 40.7 per cent in mean blood pressure. With lower mean blood pressure there was an absolute decrease

TABLE 1. Data from Seven Experiments of Halothane Anesthesia in Dogs

	Average	Percentage Change	Standard Error	P
M.B.P.				
Control	135			
Halothane	80.1	−40.7	9.1	<.001
M.B.F.				
Control	25.8			
Halothane	32.6	+26.8	4.2	<.2
I.C.V.R.				
Control	5.67			
Halothane	2.47	−56.4	.5	<.001
S.V.C.P.				
Control	−0.72			
Halothane	−0.83	−15.3	.7	<.9
C.S.F.P.				
Control	4.8			
Halothane	8.6	+79.2	3.2	<.3
Pa$_{CO_2}$				
Control	40.5			
Halothane	38.5	−4.9	.8	<.05
H.R.				
Control	132.9			
Halothane	131	−1.4	10.5	<.9

M.B.P. = Mean blood pressure (mm./Hg). M.B.F. = Mean blood flow (ml./minute). I.C.V.R. = Internal carotid vascular resistance (units). S.V.C.P. = Superior vena cava pressure (mm./Hg). C.S.F.P. = Cerebrospinal fluid pressure (mm./Hg). Pa$_{CO_2}$ = Arterial Pco2 (mm./Hg). H.R. = Heart rate per minute.

in the internal carotid blood flow depending on the cardiovascular depression. The cerebrospinal fluid pressure increased sharply upon the beginning of inhalation of halothane, and slowly returned to normal values. Figures 3 and 4 graphically show these changes during induction and recovery. There were no significant changes in mean superior vena cava pressure. Figure 5 shows the control and halothane wave form from the internal carotid blood flow. The principal modification produced by halothane consists of a "dip" in the flow during the diastolic phase.

Table 2 and figure 6 show the corresponding values for different arterial P_{CO_2} values. A maximal decrease in the internal carotid vascular resistance was observed when arterial P_{CO_2} was between 50 and 60 mm. of mercury. At the same time, a small increase in mean blood pressure, a two-fold increase in the internal carotid blood flow and a sharp rise in cerebrospinal fluid pressure were observed. In some cases there was noted a threefold increase of
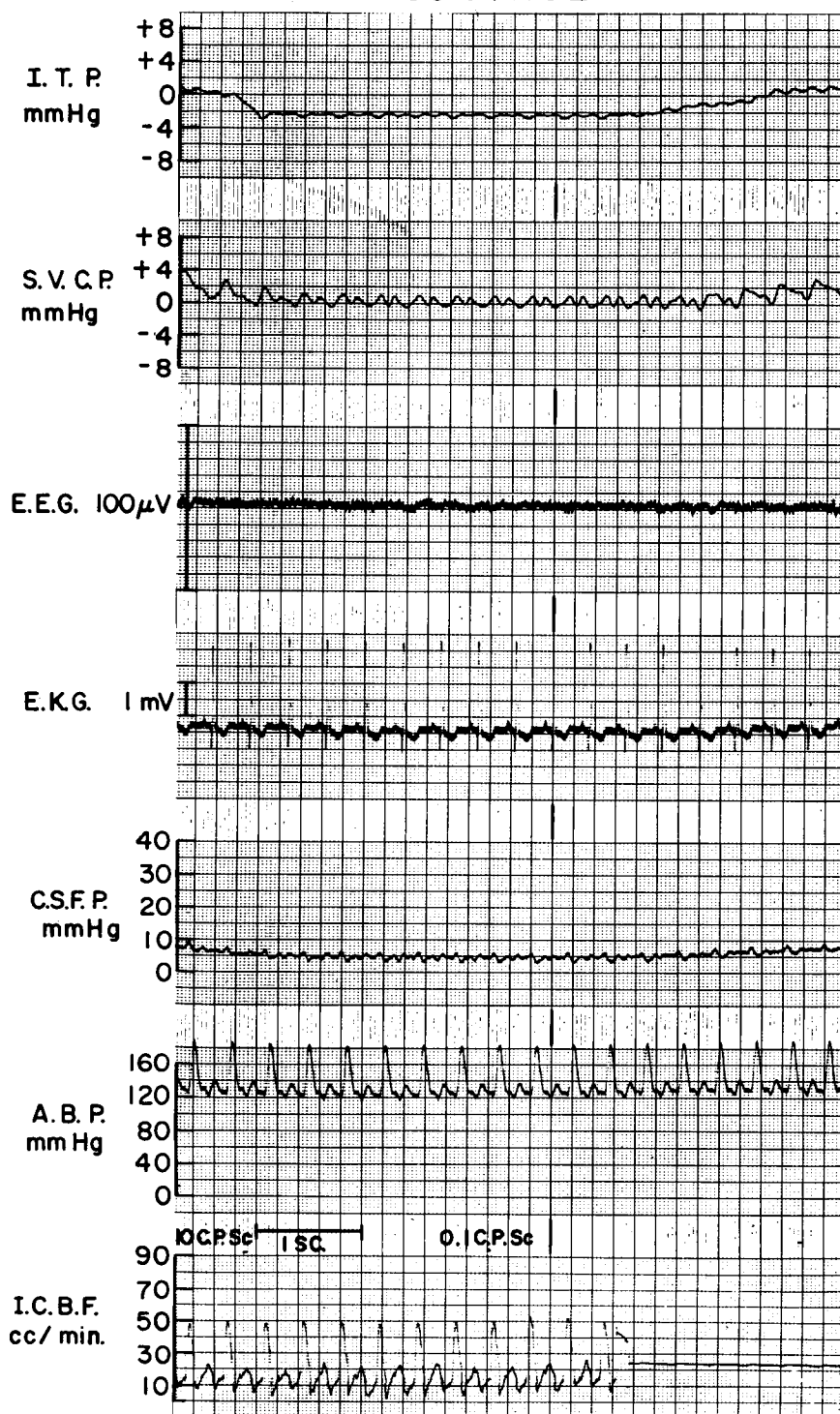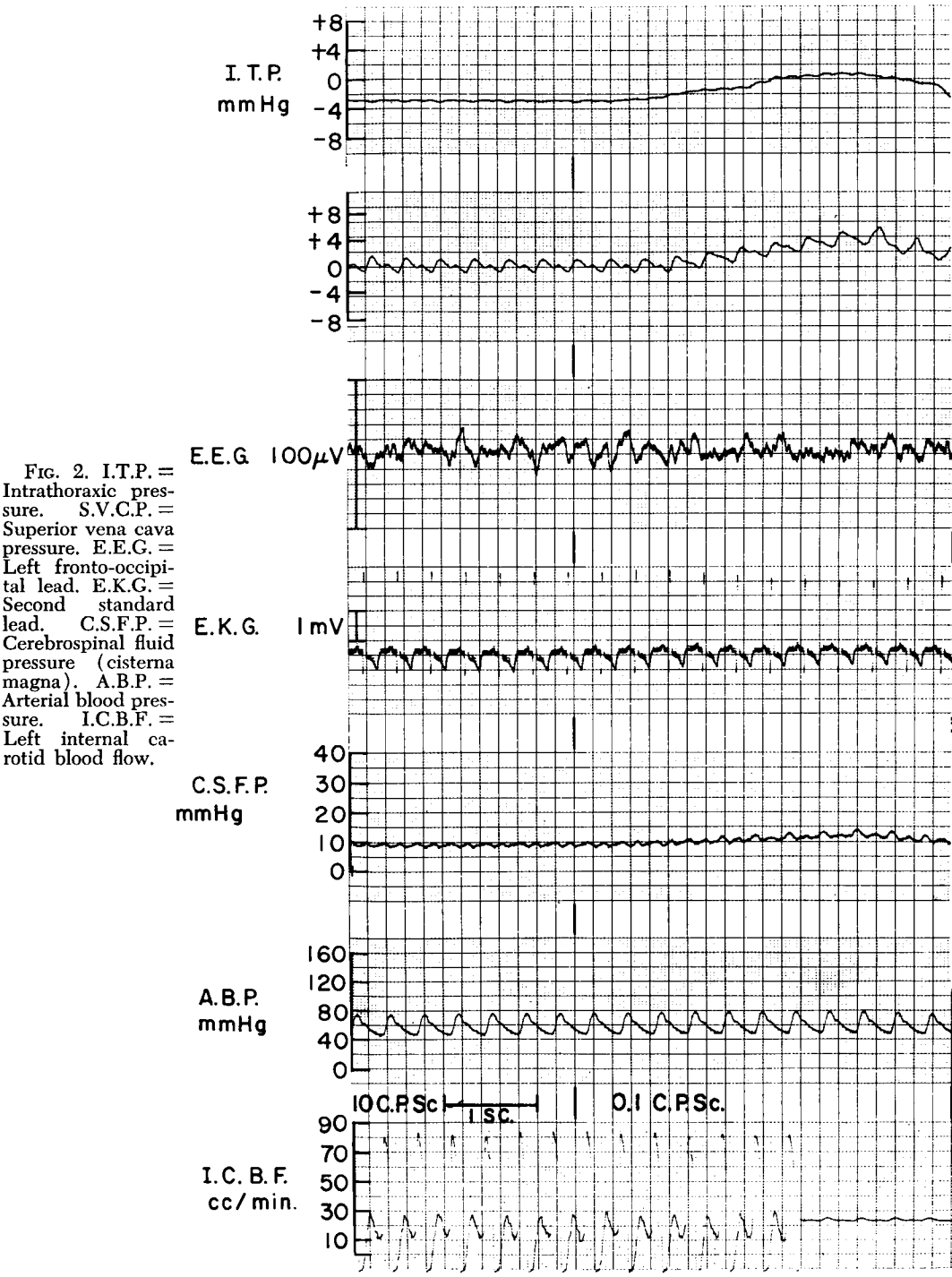
CONTROL

FIG. 1. I.T.P. = Intrathoraxic pressure. S.V.C.P. = Superior vena cava pressure. E.E.G. = Left fronto-occipital lead. E.K.G. = Second standard lead. C.S.F.P. = Cerebrospinal fluid pressure (cisterna magna). A.B.P. = Arterial blood pressure. I.C.B.F. = Left internal carotid blood flow.

HALOTHANE

I. T. P.
mmHg

+8
+4
0
-4
-8

+8
+4
0
-4
-8

E.E.G. 100μV

E.K.G. I mV

FIG. 2. I.T.P. =
Intrathoraxic pres-
sure.   S.V.C.P. =
Superior vena cava
pressure. E.E.G. =
Left fronto-occipi-
tal lead. E.K.G. =
Second   standard
lead.   C.S.F.P. =
Cerebrospinal fluid
pressure (cisterna
magna). A.B.P. =
Arterial blood pres-
sure.   I.C.B.F. =
Left internal ca-
rotid blood flow.

C.S.F.P.
mmHg

40
30
20
10
0

A.B.P.
mmHg

160
120
80
40
0

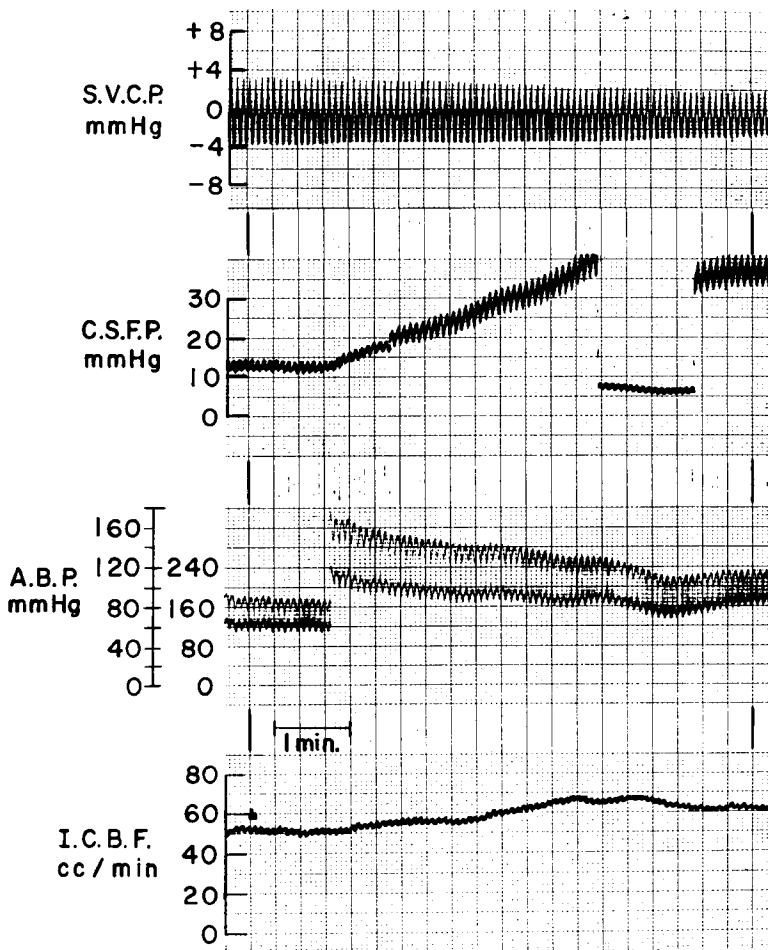10 C.P.Sc. 1 Sc. 0.1 C.P.Sc.

I.C.B.F.
cc/min.

90
70
50
30
10

FIG. 3. Beginning of the administration of halothane. S.V.C.P. = Superior vena cava pressure. C.S.F.P. = Cerebrospinal fluid pressure (cisterna magna). A.B.P. = Arterial blood pressure. I.C.B.F. = Left internal carotid blood flow.

the cerebrospinal fluid pressure value and a subsequent stabilization in accordance with the new cerebrovascular hemodynamics. This value was always higher than the control values. Figure 6 graphically shows these modifications.

Discussion

In an anatomic and angiographic study of the circulation to the dog's brain, De La Torre [6] describes the two internal carotid arteries and the basilar artery as the main blood supply. The internal carotid artery of the dog is smaller in diameter than the external carotid artery and carries an average of 15 to 20 per cent of the common carotid blood flow.[6, 7] However, we assume that this blood flow (in one internal carotid artery) does represent a sample of the cerebral blood flow in the dog.

This assumption is based on three considerations: (1) It is one of the three main arteries supplying blood to the dog's brain. (2) As can be seen in our experiments (figs. 1, 4 and 6; tables 1 and 2), blood flow changes in the internal carotid artery were followed in all experiments by changes in the cerebrospinal fluid pressure. These follow changes in cerebral blood volume and are considered as a reliable indicator of changes in cerebral blood flow.[8] This relationship between left internal carotid blood flow and cerebrospinal fluid pressure was predictable and reproducible for every measurement. (3) When CO_2 was administered in this series of experiments (fig. 6, table 2) and in other work,[9] the results were similar and in complete agreement with experiments made in man using CO_2 and the N_2O technique.[10, 11]

Thus, in our view, the internal carotid blood flow in the dog provides a reasonably reliable sample of cerebral blood flow. Estimations of its vascular resistance can be considered as representative of the cerebrovascular resistance. However, these estimations are in fact relative and not absolute values.

Halothane decreased internal carotid vascular resistance. During its administration the internal carotid blood flow was normal, while mean blood pressure was reduced (40.7 per

cent). At first, cerebrospinal fluid pressure increased sharply, after which it declined to new levels related to the internal carotid blood flow, mean blood pressure, superior vena cava pressure and cerebrospinal fluid volume. The reduction of the vascular resistance (measured in the internal carotid artery) was proportional to the depth of anesthesia with a maximum of 50 to 60 per cent, which is slightly higher than the reduction produced by 60 mm. of mercury in arterial P_{CO_2}. The decrease in mean blood

FIG. 4. Recovery from deep halothane anesthesia. S.V.C.P. = Superior vena cava pressure. C.S.F.P. = Cerebrospinal fluid pressure. A.B.P. = Arterial blood pressure. I.C.B.F. = Left internal carotid blood flow.
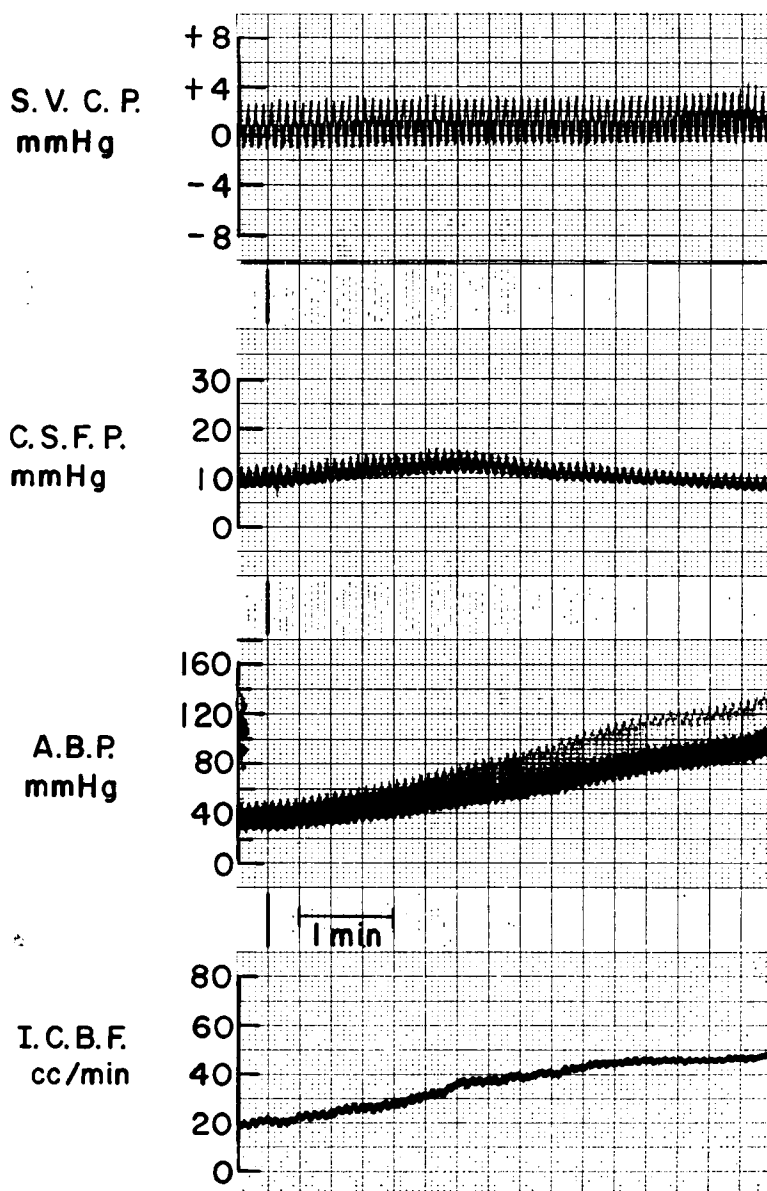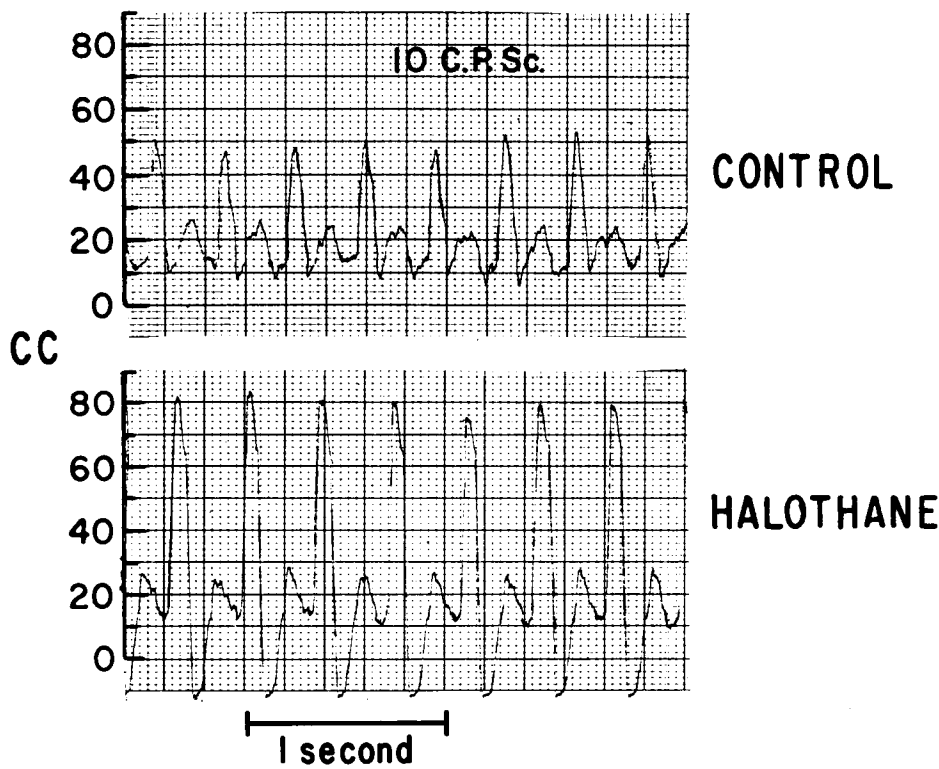
FIG. 5. The blood flow wave form modifications, produced by halothane anesthesia, in the internal carotid artery. Note the negative flow in diastole (dip) present during halothane anesthesia.

pressure was 40.7 per cent against 56.4 per cent in cerebrospinal resistance. It is difficult to differentiate between the halothane and the hypotensive effect on vascular resistance. A correlation with cardiac output is needed to clarify this point. This matter is discussed in more detail elsewhere.[7, 9] Nonetheless, an apparent change in cerebrovascular resistance follows halothane administration. Evidence for this was found in the increased internal carotid blood flow and cerebrospinal fluid pressure immediately after the beginning of halothane ad-

ministration with no substantial mean blood pressure depression (fig. 3). The same effect was observed during recovery (fig. 4).

The internal carotid blood flow was noted to be higher than the control values during light anesthesia and lower with deep anesthesia. No change in heart rate was observed, perhaps due to the administration of succinylcholine.[12] In man, Albert[13] found similar directional changes in cerebrovascular resistance and cerebral blood flow with halothane. Changes in cerebrospinal fluid pressure may be explained

TABLE 2. Results from One of the Four Experiments in Which Various Concentrations of CO_2 were Administered to Dogs

Arterial P_{CO_2} (mm./Hg)	M.B.P. (mm./Hg)	M.B.F. (ml./minute)	V.R. (units)	C.S.F.P. (mm./Hg)	V.P. (mm./Hg)
39.5	145	22	6.5	5	0
50.2	165	45	3.5	12	1
60.7	152	52 (+136%)	2.9 (−55%)	18	2

M.B.P. = Mean arterial blood pressure. M.B.F. = Mean internal carotid blood flow. V. R. = Internal carotid vascular resistance. C.S.F.P. = Cerebrospinal fluid pressure (cisterna magna). V.P. = Superior vena cava pressure.
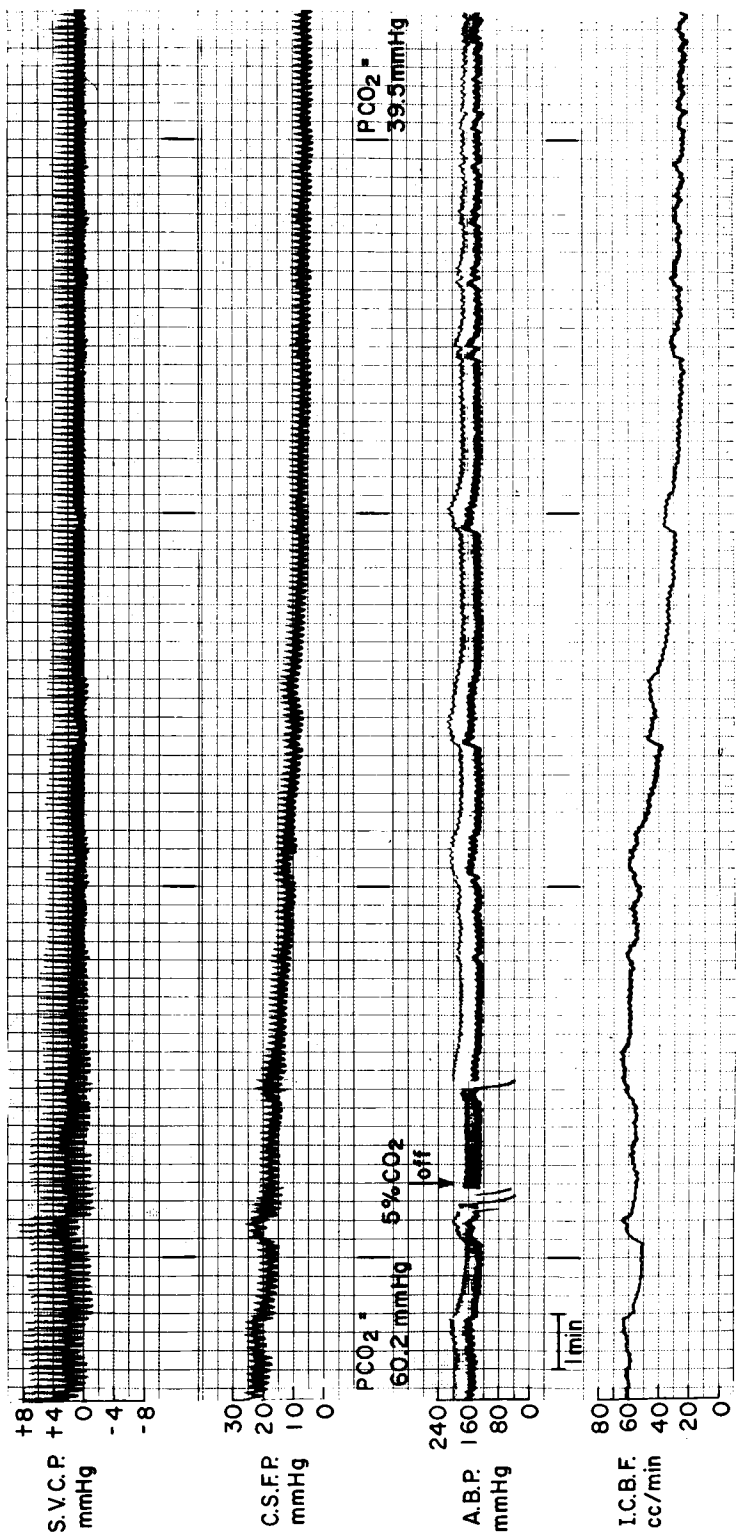
Fig. 6. Recovery after CO_2 administration. S.V.C.P. = Superior vena cava pressure. C.S.F.P. = Cerebrospinal fluid pressure (cisterna magna). A.B.P. = Arterial blood pressure. I.C.B.F. = Left internal carotid blood flow.

by the Monro-Kellie hypothesis.[14] An increase in one of the intracranial components (cerebrospinal fluid, brain tissue, brain water or cerebral blood volume) will be followed by a decrease in one or more of the other components. While this new equilibrium is taking place, there are changes in the intracranial pressure. The new pressure will be adjusted according to the final cerebral blood flow.[8] Thus, with halothane anesthesia, the cerebrospinal fluid pressure may be expected to oscillate through a wide range of values because of its rapid action on mean blood pressure, heart rate, cerebrovascular resistance and cerebral blood flow. The sustaining of a normal heart rate during halothane administration in man (atropine) may give similar results.

Summary and Conclusions

The effect of halothane anesthesia on internal carotid blood flow and intracranial pressure was studied in the dog. Internal carotid vascular resistance was calculated as the ratio between mean blood pressure and mean internal carotid blood flow per minute. Control of arterial P_{CO_2}, with a maximal decrease of 5 mm. of mercury for halothane determinations, was maintained. Administration of CO_2 was used as a reference point for the changes induced by halothane.

Halothane decreased vascular resistance, as measured in the internal carotid artery, a change related to the depth of anesthesia. The maximal decrease in resistance was −56.4 per cent with a reduction of 40.7 per cent in the mean blood pressure. This effect is roughly similar to that produced by 60 mm. of mercury in arterial P_{CO_2}. However, it was difficult to differentiate between the effects of hypotension or halothane itself on the internal carotid vascular resistance.

Intracranial pressure rose sharply and returned to new levels in accordance with the altered cerebrovascular hemodynamics and not with the changes in venous pressure alone. The intracranial pressure was high during light anesthesia and normal or low during deep anesthesia.

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