slight. The serotonin effect on the splanchnic circulation was also slight and often statistically not significant. Its infusion elicited a moderate reduction of hepatic blood flow and portal venous pressure and a slight rise of splanchnic resistances. Differences of material, dose and technic probably account for differences with the data obtained by Gibertini(5). There was no evidence that serotonin could represent, as suggested by Magdaleine *et al.*(6), a vasoconstrictor agent, able to cause or to maintain in man a portal hypertensive state.

Summary. The effect on splanchnic circulation of a serotonin constant infusion was investigated on 8 normal subjects and 8 cirrhotic patients with portal hypertension. The rate of serotonin infusion was of 30 γ/kg/min.

Mean arterial pressure, cardiac output and portal venous pressure were not significantly affected.

The estimated hepatic blood flow was reduced by 9.2% ($P<0.05$) and the splanchnic resistances were raised by 13.0% ($P<0.05$).

There was no evidence that a raised serotonin blood level in man could cause or maintain a portal hypertensive state.

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Arterial Blood Acid-Base Balance in Unrestrained Waking Cats.* (28032)

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Available data on the blood acid-base balance of the intact cat(1,2) refer either to venous blood or to mixed venous and arterial blood, in both cases on animals depressed by anesthesia. Because of the susceptibility of the cerebral circulation and of neural responses to changes in carbon dioxide tension, reliable reference values are needed for standardizing acid-base balance in neurophysiological experiments. Such values are reported here for animals free from experimental restraints and with an intact nervous system.

Healthy cats weighing 2.5 to 3.5 kg, observed for 2 weeks before operation, were anesthetized with pentobarbital, 35 mg/kg body weight, injected intraperitoneally. After ligation of the left common carotid artery a 17-gauge polyethylene catheter was introduced below the ligature and advanced into the aorta. The peripheral end of the catheter, mounted on an 18-gauge hypodermic needle capped with rubber, was anchored to the parietal bone. The needle hub projected through the skin, leaving the rubber cap easily accessible for sampling. Blood samples of one ml were withdrawn at intervals up to 15 days after the operation. pH was measured at 37°C. Carbon dioxide content was determined micromanometrically and pCO_2 calculated from the Henderson-Hasselbalch equation ($pK = 6.1$). In 3 animals samples of 2 ml were drawn, permitting determination of serum sodium and potassium with a Patwin flame photometer, chlorides with a Cotlove chloridimeter, packed red cell fraction by microhematocrit and hemoglobin oxygen saturation with a Beckman DU spectrophotometer and Nahas cuvette. Two ml of heparinized saline (50 mg per 1000 ml) were flushed through the catheter after each

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TABLE I.

Cat No.	P.O.D.	CO_2, mM/l	pH	Pco_2, mm Hg	HCO_3, mM/l
1	2	16.69	7.45	23.9	16.0
2	4	14.66	7.37	25.0	13.9
3	1	15.83	7.38	26.5	15.8
4	1	16.30	7.28	33.6	15.3
	4	16.78	7.38	27.7	16.0
	6	16.96	7.39	27.7	16.1
5	1	16.96	7.35	30.0	16.1
	2	17.66	7.35	31.2	16.7
	4	17.23	7.34	31.3	16.3
	6	18.17	7.44	26.3	17.4
6	1	18.86	7.33	34.6	17.8
7	1	24.0	7.42	24.0	14.9
	4	24.2	7.34	24.2	12.8
	5	22.0	7.39	22.0	12.9
8	1	17.33	7.50	22.3	16.7
	4	14.72	7.32	28.1	13.9
	5	16.24	7.42	24.5	15.5
	6	16.14	7.40	25.3	15.4
	7	17.95	7.43	26.6	17.2
9	4	18.48	7.38	32.2	18.5
	5	14.28	7.40	23.5	14.3
10	1	20.46	7.35	36.2	19.4
	4	15.52	7.43	23.0	14.8
11	1	19.37	7.33	36.1	18.3
	4	15.34	7.43	22.6	14.7
12	1	15.74	7.32	29.6	14.8
	5	19.34	7.42	29.0	18.5
	7	18.77	7.38	30.8	17.8
	8	17.58	7.36	30.1	16.7
	13	19.06	7.38	31.6	18.1
	15	16.21	7.34	29.0	15.3
Mean		17.70	7.38	28.0	16.06
S.D.		2.41	.06	4.1	1.62

sample was drawn. Clots formed in spite of this and the catheters became plugged in most animals within a week. The animals looked healthy and behaved normally throughout the observations. At autopsy the viscera were free from disease.

Results of the analyses are shown in Tables I and II. Some of the samples on the first post-operative day probably reflected residual respiratory depression from the anesthetic. When first-day samples were excluded, the remaining 22 samples yielded somewhat lower average values. (Table I, bottom row.) Ngai (3) found an average pCO_2 of 27 $\pm$ 3.5 mm Hg in the decerebrate cat, in good agreement with our findings. It may, therefore, be concluded that with respect to carbon dioxide, respiratory regulation in the intact resting animal is similar to that of the midcollicular decerebrate preparation. However, Ngai assumed the relatively low pCO_2 was compensatory to a metabolic acidosis induced by anesthesia. Our results indicate that a relatively low arterial pCO_2 is a normal feature in laboratory cats(4), probably reflecting a smaller buffering capacity of the blood in this species. The Van Slyke buffer value $\Delta BHCO_3$/unit ΔpH for cat blood is 19.8(5) compared to a buffer value of 30.8 for man(6). Serum electrolytes analyses (Table II) were somewhat lower than reported by Yannet(2). The excess of measured cations over measured anions in Table II averages 25.5 meq/l. Since this omits calcium and magnesium, ion, the total of undetermined protein anion exceeds 25.5 meq/l, and is probably appreciably greater in the cat than in man. Previous data on cat hematocrit do not seem to have been published. The present determinations suggest that a low hematocrit may not be unusual in the cat.

Differences between the acid-base balance of the cat and of man should be taken into account when this animal is subjected to artificial ventilation, particularly during experiments on the central nervous system. The known sensitivity of the cerebral circulation and of the electroencephalogram to carbon dioxide suggest that the volume of ventilation may influence neuronal activity *in vivo* quite critically(7). Ideally, the acid-base balance of the arterial blood should be standardized in all neurophysiological studies on paralyzed animals.

Summary. Thirty-one blood samples from 12 animals were obtained through a catheter implanted in the left common carotid artery. Analyses yielded the following mean values: pH 7.39 $\pm$ 0.4; (HCO_3^-) 15.8 $\pm$ 1.8 meq/l; and pCO_2 27.2 $\pm$ 3.4 mm Hg. These figures reveal a low blood bicarbonate and carbon

TABLE II.

Cat No.	P.O.D.	Het	HbO_2, %	K	Na	Cl	HCO_3
				meq/l			
10	4	37.5	96.2	3.5	146	109	14.8
11	1	28	90	4.0	144.5	110	18.3
	4	23	86.1	4.0	141.3	107.3	14.7
12	1	33.1	94	4.0	148.3	111.1	14.8
	5		89	5.5	150	107.5	18.5
	7	25.4	82	4.2	154.2	108.5	17.8
	13	25.9	94	7.6	148.1	113.2	18.1
Mean		28.8	90.1	4.7	147.5	109.5	16.7

dioxide content relative to man. They should be regarded as the standard values to be maintained in cats undergoing artificial ventilation in neurophysiological experiments.

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Plasma Potassium Changes During Rebreathing in the Dog.* (28033)

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A gradual rise in the plasma potassium concentration during breathing of high CO_2 mixtures in the dog has been reported by several investigators(1,2,3). On the other hand, when cats are made to breathe 35% carbon dioxide there is an immediate but transient rise in potassium concentration in the plasma followed by a fall to normal or near normal levels until the animal is allowed to breathe air again, at which time a second elevation takes place(4). This second elevation on reversing the severe hypercarbic acidosis is also regularly observed in the dog(2). Serial sampling of arterial blood at one minute intervals during the first 10 minutes of 30% CO_2 breathing in the dog has revealed no evidence of a transient elevation at this time. However, a transient elevation of plasma potassium concentration has been reported in dogs rebreathing from a bag and therefore, subjected to a gradual elevation of CO_2 over a period of 90 minutes(5). The purpose of this paper was to reexamine this phenomenon.

Methods. Experiments were performed on 10 mongrel dogs anesthetized with intravenous thiopental sodium. After the initial induction, additional anesthetic was administered as required to maintain surgical anesthesia. Arterial blood samples were drawn from a catheter passed through the femoral artery into the abdominal aorta. Rebreathing was carried out by connecting the dog to a 35 liter bag by a cuffed endotracheal tube. Blood pH was determined with a glass electrode at 38° and correction was made, using the factor –.014 pH/°C, when the body temperature of the dog deviated from this value by as much as 0.5°C. Plasma potassium concentration was determined on an internal standard flame photometer. CO_2 and O_2 concentrations in the rebreathing bag were estimated with the Scholander rapid gas analyzer. Oxygen concentration in the bag was maintained well above 20 volumes per cent at all times. Arterial blood samples were drawn before, at regular intervals during the rebreathing, and at 5 minutes following the return to room air breathing. Electrocardiograms were recorded before, during, and following the experimental rebreathing period.

Results. The results are presented in Fig. 1. No transient rise in potassium concentration was evident in these 10 experiments. A gradual rise in plasma potassium concentration took place during the first 30 minutes of the rebreathing period. This increase was statistically significant at this time (P–.01). Between 30 and 60 minutes potassium concentration continued to rise with little or no change between 60 and 90 minutes.

CO_2 tension increased during the 90 minutes and blood pH fell correspondingly. On returning the animals to room air breathing, blood pH rose rapidly to almost normal val-

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