tained normal, — not asphyxial in respect either to the oxygen or carbon dioxide contents. When the stomach was distended with air, and the large intestine and lower ileum with bread mush, movements in these three parts of the alimentary canal were seen identical with those shown by the radiographs of Cannon. In the stomach a deep constriction developed at the pre-antral groove every 15 seconds and moved toward the pylorus where it disappeared as its successor was developing. In the colon there was active anti-peristalsis. In the ileum vigorous rhythmic segmentation was seen.

In other experiments I have found that animals under ordinary operative conditions develop, and remain in, a state of acapnia. This lowered carbon dioxide content of the blood and tissues, by inducing loss of tonus, is the cause of the failure of peristalsis after laparotomy. The essential point in the above described method is the prevention of acapnia.

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Studies on the effects of carbon mon-oxide poisoning.

By **A. I. RINGER.** (By invitation.)

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If an animal be allowed to breathe an atmosphere containing carbon mon-oxide, it will soon present a series of circulatory, respiratory, cerebral and metabolic disturbances, which, if carried too far, will result in death. These disturbances are believed to be brought about by the reduction of the oxygen-carrying capacity of the blood, due to the formation of the relatively stable carbon mon-oxide-hemoglobin, thus producing a state of progressive asphyxiation of the tissues. The severity of these disturbances depends entirely upon the degree of asphyxiation; and, with the exception of some individual peculiarities in a few of the twenty-one dogs that I have experimented upon under anæsthesia, all presented the same symptoms at the same stage of asphyxiation.

In nine experiments the following subjects were studied in their relationship to the degree of saturation of the hemoglobin with carbon mon-oxide: (1) the pulse, (2) the blood pressure,

(3) the number of respirations per minute, (4) the volume of air respired per minute; in twelve experiments: (1) the physical symptoms, (2) the point of onset of coma, (3) the point of death.

As soon as the animal begins to breathe the carbon mon-oxide there is an immediate acceleration of the pulse, which steadily gains in frequency until 45–50 per cent. of the hemoglobin is saturated with carbon mon-oxide. Then the pulse rate is at its maximum. After that it declines gradually until a point is reached between 71–74 per cent. of carbon mon-oxide saturation, when the pulse rate falls abruptly and the heart ceases to beat.

The blood pressure in about half the cases was found to go gradually downward in spite of the markedly increased rate in the heart beat. In the rest of the cases there was a preliminary rise in pressure of about 10–15 mm. of Hg, which reached its maximum when 45–50 per cent. was saturated with carbon mon-oxide. This was followed by a gradual decline, until when about 67 per cent. of the hemoglobin was saturated, there was an abrupt fall in blood pressure.

The number of respirations per minute, taking two characteristic examples, was found to increase from a normal of 19 and 22 to 32 and 45, respectively, when about 50 per cent. of the hemoglobin was saturated with carbon mon-oxide. In other cases there was no increase in the rate of respiration at all, but there was a marked increase in the depth of each inspiration. The volume of air respired per minute, however, increased in all cases as soon as the animal began to breathe the carbon mon-oxide. It presented a curve with an ascending and descending limb. The maximum ventilation of the lungs took place when about 45 per cent. of the hemoglobin was combined with carbon mon-oxide. After that it declined gradually. At about 65 per cent. saturation, however, breathing became irregular, sometimes of the Cheyne-Stokes variety. Respiration always stopped about one to three minutes before the heart ceased beating.

When about 40 per cent. of the dog's hemoglobin is deprived of its oxygen carrying capacity, the animal begins to get weak. It cannot stand on its legs. It is in a state of general indifference. It does not partake of any food or drink, though it may be hungry. It is seized with vomiting and occasionally has convulsive spells.

It responds to a call very slowly, and sometimes falls into a sleep, from which it is aroused with difficulty. Actual coma sets in at a point immediately following the decline in the pulse rate, or when about 50 per cent. of the hemoglobin is saturated with carbon mon-oxide. This has been found to be constant in all cases.

Death also takes place in all dogs at about the same point of saturation. Not a single one reached the 75 per cent. mark. All died between 71 and 74 per cent. of saturation of the hemoglobin with carbon mon-oxide. Neither size, weight nor strength seemed to influence the point of death.

From the foregoing it is seen that the symptoms of carbon mon-oxide poisoning may be conveniently divided into three stages: First or Compensatory Stage, which lasts up to 50 per cent. saturation; second or Stage of Depression which lasts up to about 70 per cent. saturation; third or Stage of Collapse.

SYMPTOMS OF THE FIRST OR COMPENSATORY STAGE.

1. Gradual increase in pulse rate.
2. High blood pressure.
3. Spasmodic attacks of vomiting.
4. Slight dyspnœa at first — more marked at the end.
5. Muscular weakness.
6. Drowsiness, indifference and deep sleep from which dog can be aroused.

SYMPTOMS OF THE SECOND OR STAGE OF DEPRESSION.

1. Ushered in by clonic convulsion and muscular rigidity which lasts for a few minutes.
2. Deep narcosis from which animal cannot be aroused.
3. Pulse declines gradually in rate, tension and volume.
4. Respiration either rapid and shallow, or slow and deep; it is more or less regular.
5. Absolute loss of sensation.

SYMPTOMS OF THE THIRD OR STAGE OF COLLAPSE.

1. Pulse slow, irregular and of low tension.
2. Respiration irregular.
3. Loss of tone of sphincters.
4. Conjunctival reflex lost.
5. Death due to respiratory paralysis.

Typical experiment of first series.— February 19, 1908. Dog's weight 9.7 Kg. Ether anæsthesia.

Time.	Respiration.	Pulse.	Blood pressure in mm. of Hg.	Volume of air (in c.c.) respired per minute.	Per cent. of Hb saturated with CO.
2.15 P. M.	22	140	90	1,750	
2.18 "	1 per cent. CO respired				
2.35 "	24	148	90	1,925	
2.50 "	36	164	92	2,000	20
3.05 "	40	180	96	2,125	
3.22 "	45	204	104	2,250	47
3.35 "	41	194	94	2,250	
3.45 "	40	166	88	2,100	54
3.50 "	36	120	72	1,925	
3.55 "	32	82	62	1,650	66
3.58 "	8 irregular	56	40	1,175	
4.05 "	10 "	68	—	750	73
4.08 "	Death				

Typical experiment of second series.— March 2, 1908. Dog's weight 6.6 Kg.

Dog placed in air-tight cage through which the gas mixture was driven by means of bellows, operated by a water pressure engine. The ventilation of the cage was 5,000 c.c. of air per minute.

Time.	Respiration.	Pulse.	General condition.
10.30 A. M.	16	104	Dog in good condition.
10.35 "	0.4 per cent. CO respired		
11.00 "	16	104	Dog comfortable and quite active.
11.45 "	16	116	" " " " "
12.00 "	18	124	" " " " "
12.30 P. M.	17	140	Lies quietly; slightly drowsy.
12.45 "	21	170	Drowsiness marked.
1.00 "	18	182	Slight convulsion and vomiting.
1.20 "	16	208	Responds to sensory stimulus after long latent period.
1.35 "	16	192	Convulsions of clonic type.
1.55 "	17	196	Slightly comatose; when called he only opens his eyes.
2.05 "	16	180	Coma quite deep.
2.30 "	—	166	All muscles and sphincters relaxed.
2.45 "	—	—	Taken out of cage. Blood sample shows 52 per cent. of the Hb saturated with CO. Dog placed in warm place near radiator.
3.45 "			Respiration slow and deep, 10–12 per minute.
4.00 "			Dog began to move about.
4.25 "			Able to stand up and walk but falls frequently, due to lack of coördination.
5.10 "			Walked about fairly well, but very slowly.
Next morning			Dog perfectly well.

Experiments have also shown that a dog with as much as 69 per cent. of hemoglobin tied up with carbon mon-oxide which corresponds to the end of the second stage, can be resuscitated if proper treatment be instituted promptly.

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Intestinal excretion during diarrhea.

By **GEORGE B. WALLACE** and **HUGO SALOMON.**

[*From the Laboratory of the Von Noorden Clinic, Vienna.*]

Analyses were made of the fæces of a number of patients with diarrheas of different origin. During one period of observation the patients were on the Schmidt-Strassburger diet, during a second period the diet consisted of 250 grm. sugar daily. In those cases where there was present an ulcerative process in the intestine — tuberculosis, carcinoma — the amount of nitrogen in the fæces was markedly increased — being from 1.7 to 4. grm. daily on the sugar diet. In cases of severe catarrhal inflammation it was not over 1.5 grm.; in light catarrh it was within normal limits. The fat and carbohydrate elimination showed no such striking differences although it was highest where an ulcerative condition was present. Of the inorganic constituents the alkali excretion was fairly parallel to that of nitrogen. The other inorganic constituents were increased by the ulcerative processes but in some instances were increased equally where ulcerations were absent.

The most striking result of the analyses is the high nitrogen excretion which occurs in ulcerative processes in the intestine.

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The vascularity of the kidney as influenced by sensory impulses.

By **R. BURTON-OPITZ** and **DANIEL R. LUCAS.**

[*From the Physiological Laboratory, Columbia University.*]

Quantitative determinations of the blood-flow through the left kidney were made with the aid of the stromuhr of Burton-Opitz. On stimulation of the central end of the sciatic nerve, a slight de-