during inflammation evidently takes place with the involvement of a complex system of factors and cell-tissue reactions.

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GASTRIC SECRETION AND EXCRETION UNDER MINERALOCORTICOID DEFICIENCY

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Besides its basic digestive function the stomach also participates in the regulation of the acid–base balance and water metabolism of the body.

The object of this investigation was to study gastric secretion in dogs with mineralocorticoid deficiency resulting from bilateral adrenalectomy and receiving replacement injections of hydrocortisone (1.2-1.7 mg/kg body weight), maintaining the blood 11-hydroxycorticosteroid concentration at the control level. As a result of complete removal of the adrenals a deficiency of gluco- and mineralocorticoids only develops, for compensatory shifts of sympathetic nervous activity and of gonadal function are observed [6-9].

EXPERIMENTAL METHOD

Experiments were carried out on 14 mongrel male dogs with Pavlov gastric pouches. Gastric secretion was stimulated by subcutaneous injection of histamine and by feeding with meat (samples of juice were collected after 15 min and hourly, respectively). The volume of gastric juice (expressed per kg body weight), its acidity, and its content of ammonia [2] and also of the exogenous dye neutral red [1] were determined. After a control series of experiments on eight dogs, one-stage bilateral adrenalectomy was performed. The subsequent experiments began on the 8th-9th days and ended on the 16th-17th days after the operation. Experiments were carried out at these same times on six dogs undergoing mock operations.

EXPERIMENTAL RESULTS

The results showed that all changes in gastric secretion were due to a primary mineralocorticoid deficiency, for the corresponding parameters in dogs undergoing the mock operation did not differ from the controls (Tables 1 and 2).

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TABLE 1. Time Course of Secretion of Gastric Juice (in ml/kg) in Dogs with Mineralocorticoid Deficiency (M ± m)

Experimental conditions	Stimulator of secretion	Number of experiments	Sample No. 1	2	3	4	5
Adrenalectomy:							
intact dog	Histamine	32	0,51±0,02	0,54±0,05	0,39±0,04	0,24±0,03	0,11±0,04
	Meat	36	0,87±0,10	0,38±0,04	0,31±0,03	0,30±0,03	0,27±0,03
mineralocorticoid deficiency	Histamine	22	0,67±0,05*	0,41±0,05	0,21±0,04*	0,07±0,02*	
	Meat	20	0,56±0,09*	0,29±0,04	0,24±0,02	0,15±0,02*	
Mock adrenalectomy:							
intact dog	Histamine	8	0,62±0,04	0,54±0,03	0,22±0,02	0,06±0,006	
	Meat	16	0,32±0,05	0,29±0,03	0,19±0,03	0,14±0,01	0,09±0,01
dogs undergoing mock operation	Histamine	10	0,60±0,03	0,51±0,03	0,25±0,03	0,06±0,01	
	Meat	20	0,31±0,04	0,26±0,01	0,19±0,02	0,14±0,01	0,07±0,01

*Here and in Table 2, $P < 0.05$ compared with initial value.

TABLE 2. Time Course of Excretion of Neutral Red (M ± m)

Experimental conditions	Stimulator of secretion	Excretion of neutral red, nanomoles/kg, sample No. 2	3	4	5
Adrenalectomy:					
intact dog	Histamine	51,90±4,84	19,72±1,73	5,88±0,69	2,07±0,17
	Meat	14,53±1,38	12,45±1,04	6,92±0,69	3,11±0,27
mineralocorticoid deficiency	Histamine	40,13±3,11*	14,87±1,38		
	Meat	23,52±2,07*	8,30±0,69*	2,07±1,73*	
Mock adrenalectomy:					
intact dog	Histamine	45,67±4,39	18,40±3,04		
	Meat	32,83±8,06	11,14±2,35	6,78±1,24	5,12±0,79
dogs undergoing mock operation	Histamine	41,52±3,49	22,38±3,04		
	Meat	23,18±3,04	13,21±1,83	8,89±1,14	2,76±1,38

Mineralocorticoid deficiency had an inhibitory action on secretion of gastric juice: The volume of juice over the whole period of the experiment was reduced from 2.51 ± 0.51 ml/kg (before adrenalectomy) in the case of stimulation by feeding with meat ($P < 0.05$) and from 1.76 ± 0.18 to 1.34 ± 0.10 ml/kg ($P < 0.05$) in the case of stimulation by histamine. However, whereas in response to eating meat by dogs with mineralocorticoid deficiency secretion of juice was retarded: a long latent period of 17.3 ± 2.3 min (11.3 ± 0.2 min before adrenalectomy, $P < 0.05$), a small volume of the hourly portions of juice, coupled with a decrease in the total duration of secretion (Table 1), histamine secretion followed a more rapid course. This was shown by changes in the time course of juice secretion and a shortening of the total period of secretion (Table 1). The latent period of secretion of juice in response to histamine in dogs with mineralocorticoid deficiency was unchanged at 7.2 ± 0.8 min (7.5 ± 0.4 min before adrenalectomy).

The rate of secretion of active H^+ ions also was reduced in mineralocorticoid deficiency: The rate of secretion in response to meat feeding before adrenalectomy was 0.71 ± 0.08 mmole H^+/h and after the operation 0.29 ± 0.03 mmole H^+/h ($P < 0.05$). During stimulation of secretion by histamine the corresponding figures were 2.18 ± 0.21 and 0.67 ± 0.05 mmole H^+/h ($P < 0.05$). The role of mineralocorticoid deficiency in the decrease in volume of gastric secretion and in acid secretion is evidently linked with its influence on water and mineral metabolism in general, and with the development of such consequent reactions as progressive loss of sodium and water, a decrease in the circulating blood volume, and a fall in blood pressure [3]. Under these circumstances redistribution of extra- and intracellular water takes place, in favor of an increase in the quantity of intracellular water. Despite the fact that both groups of hormones (gluco- and mineralocorticoids) duplicate each other's action to some extent, injection of hydrocortisone nevertheless cannot restore the normal content of tissue water when disturbed after adrenalectomy [10]. Mineralocorticoids can perhaps regulate transport processes in the gastric mucosa directly through the induction of synthesis of enzymes and of transport proteins, as has been demonstrated for other organs [5].

Changes in hemodynamic conditions and perhaps, in urease activity in mineralocorticoid deficiency also are reflected in ammonia excretion. After meat feeding ammonia excretion during the period of the experiment fell to 7.27 ± 0.75 μmoles/kg (12.24 ± 1.13 μmoles/kg before adrenalectomy, $P < 0.05$). This fall by almost 40% below the background level was as-

sociated with a reduction in juice secretion, which fell in the same proportion, for the ammonia concentration in the blood and gastric juice was unchanged. The rate of ammonia production in response to injection of histamine was unchanged: 5.79 ± 0.59 μmoles/kg before adrenalectomy and 7.02 ± 0.68 μ moles/kg during mineralocorticoid deficiency. The amount of ammonia excreted after injection of histamine probably is independent of the state of the renin-aldosterone system and characterized the actual state of excitation in the stomach [4].

The level of excretion of exogenous dyes with the gastric juice depends on two principal factors: binding and transport of the dye in the stomach wall and the volume of blood flowing to the stomach. Neutral red (2 mg/kg) was injected intravenously at the end of collection of the first sample of juice, i.e., when secretion was maximal. The rate of excretion of neutral red in mineralocorticoid deficiency during the period of the experiments was found to be reduced when secretion was stimulated by both methods. Whereas before adrenalectomy the total quantity of neutral red excreted during the period of the experiment in response to histamine secretion was 82.0 ± 7.3 nmoles/kg, it fell during mineralocorticoid deficiency to 56.0 ± 4.84 nmoles/kg ($P < 0.05$); in the case of meat feeding the corresponding figures were 42.21 ± 3.8 and 33.90 ± 3.11 nmoles/kg ($P < 0.05$). The duration of excretion was reduced and the latent period before appearance of the dye was increased: When secretion was stimulated by histamine the latent period before adrenalectomy was 1.2 ± 0.5 min and in mineralocorticoid deficiency 4.5 ± 0.4 min ($P < 0.05$); when secretion was stimulated by meat the corresponding figures were 8.7 ± 0.7 and 12.0 ± 1.2 min ($P < 0.05$). Consequently, the character of the change in neutral red excretion was closely similar to the character of the change in juice secretion, and it evidently reflects the changed hemodynamic conditions in the stomach. However, changes in the time course of neutral red excretion in the course of the experiment (Table 2) show that mineralocorticoid deficiency also is reflected in the level of metabolism in the stomach wall.

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