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EFFECT OF ARTERIAL BLOOD LOSS ON MYOELECTRICAL ACTIVITY OF THE PYLORIC SPHINCTER AND DUODENUM

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Blood loss is a stress factor leading to the formation of gastric and duodenal ulcers [3, 9, 13-15]. Arterial blood loss leads to considerable release of catecholamines [8, 9, 15], which mainly inhibit the motor function of the gastrointestinal tract [1, 2, 5, 7, 12] and participate in ulcer formation [4, 6, 13]. Meanwhile the influence of this stress factor on the motor function of the pyloroduodenal zone (that most prone to ulcer formation) has not been finally elucidated. The aim of this investigation was to study changes in electrical activity of the smooth muscles of the pyloric sphincter and duodenum under the influence of arterial blood loss.

EXPERIMENTAL METHOD

Chronic experiments were carried out on six male rabbits weighing 2.6-3.2 kg. Two weeks before the experiment, silver loop electrodes were implanted into the smooth muscles beneath the serous membrane of the pyloric sphincter and duodenum, by the method described previously [10, 11]. Electrical activity of the smooth muscles of the pyloroduodenal zone was recorded on an encephalograph with recording speed of 7.5 mm/sec and with time constant of 0.3 sec. The rabbits received an ordinary diet (vegetables, oats, hay) and were used in the experiments without any preliminary restrictions of food taking. The right common carotid artery was exteriorized in the neck 1 week before the experiments, into a skin bridge 2-3 cm long. Blood loss was produced by puncturing this vessel in animals immobilized in the supine position by the method in [3]. The blood loss amounted to about 5, 10, and 25% of the total blood volume, and its duration was 2 min. Electrical potentials of the smooth muscles of the sphincter and duodenum were recorded for 1 h before, during, and for 1 h after blood loss. The frequency of bursts of action potentials of the smooth muscles of the pyloric sphincter and duodenum was analyzed before and after blood loss, and the pulse rate (as a parameter of activation of the adrenergic system) was analyzed on the basis of the electrocardiogram. The statistical significance of differences was determined by Student's test, with a 95% level of significance.

EXPERIMENTAL RESULTS

Arterial blood loss (5, 10, and 25% of the total blood volume) gave rise to a biphasic change in electrical activity of the smooth muscles of the pyloric sphincter and duodenum. Phase I of the action of arterial blood loss was manifested by inhibition of activity of the pyloroduodenal zone, phase II by its gradual recovery, depending on the volume of blood lost.

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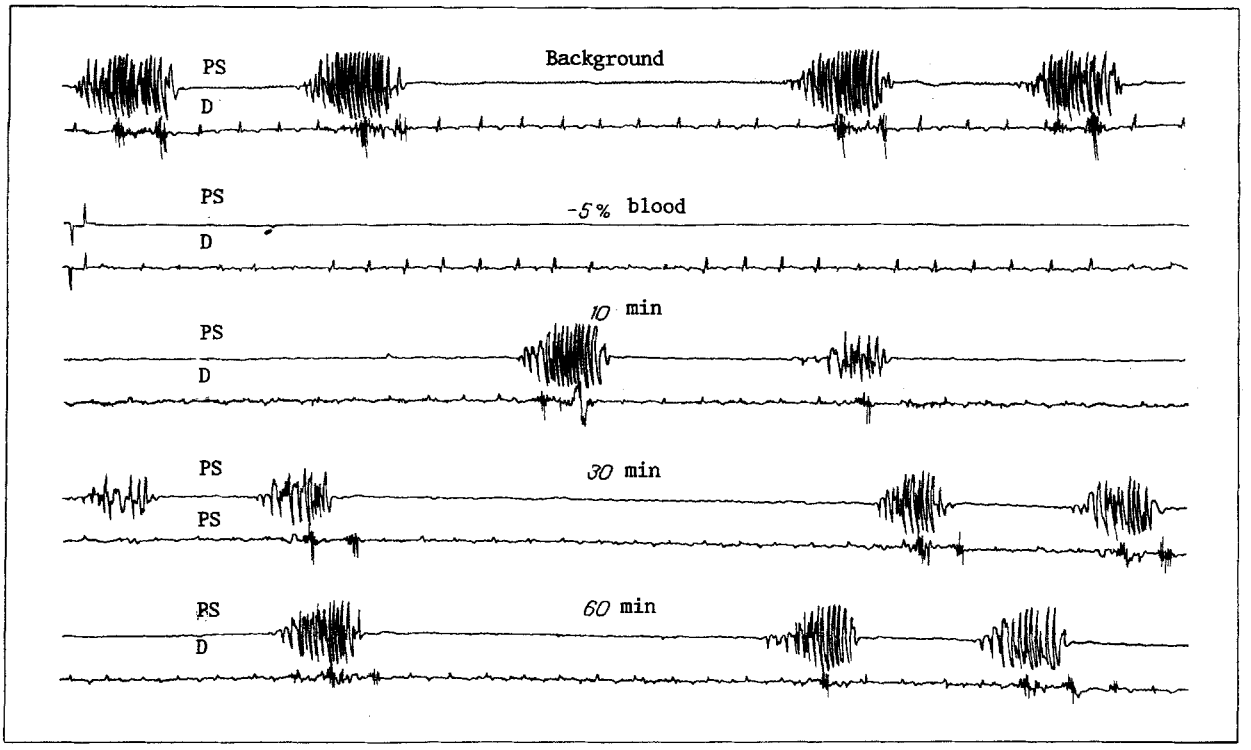

Fig. 1. Myoelectrical activity of pyloric sphincter and duodenum before, during, and 10, 30, and 60 min after 5% arterial blood loss. PS) Pyloric sphincter, D) duodenum. Time marker 1 sec, calibration 500 V (for all three figures).

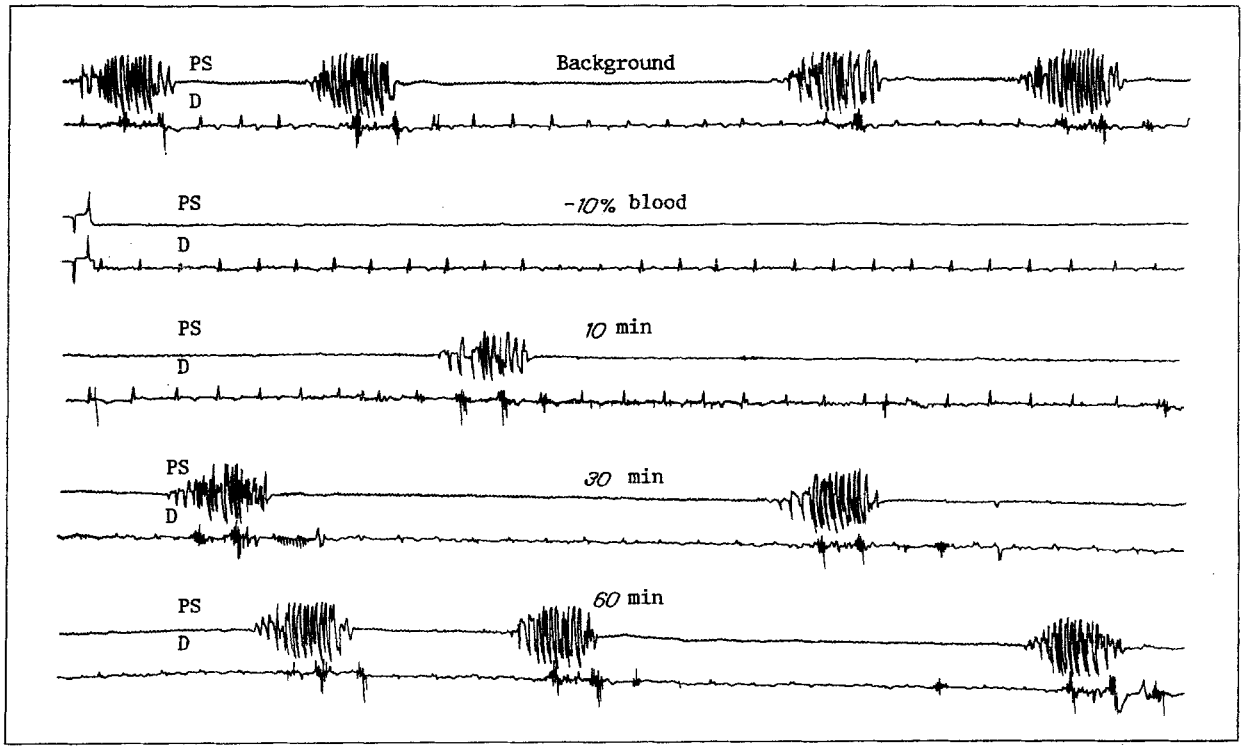

Fig. 2. Myoelectrical activity of pyloric sphincter and duodenum before, during, and 10, 30, and 60 min after 10% blood loss.

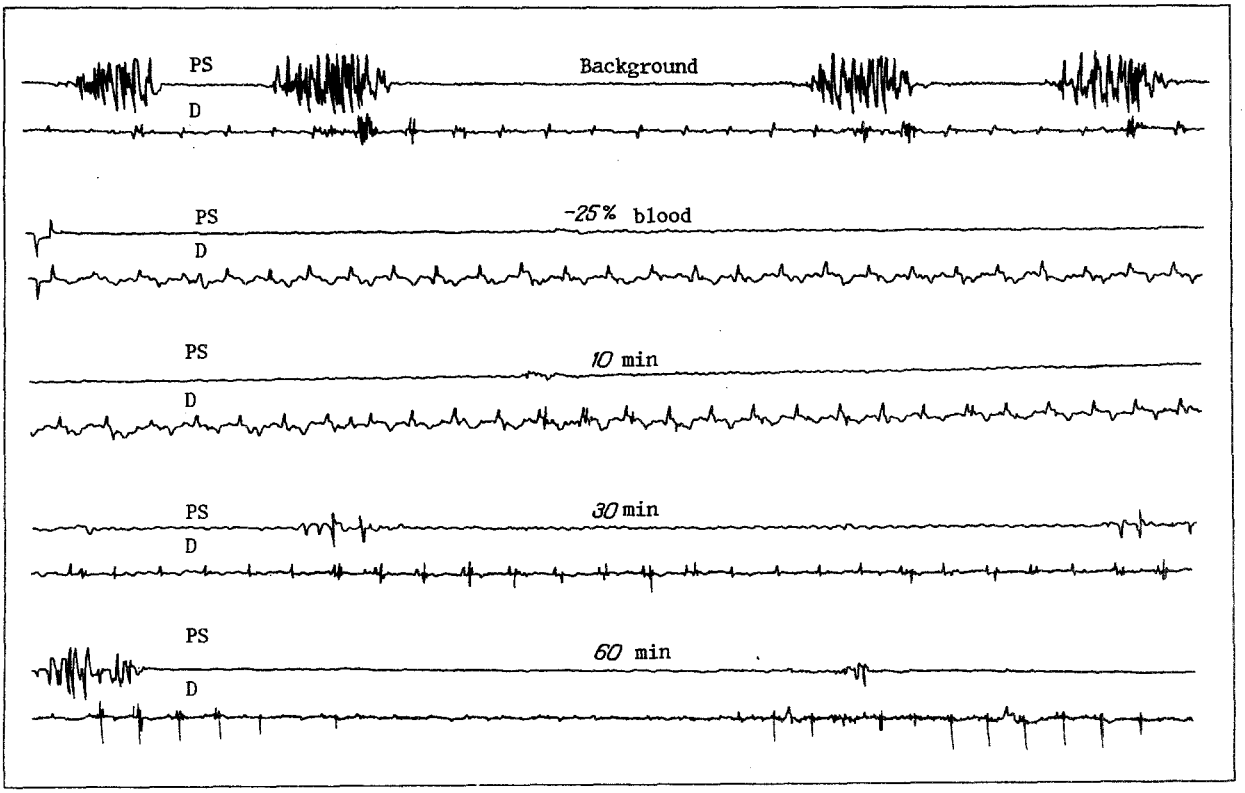

Fig. 3. Myoelectrical activity of pyloric sphincter and duodenum before, during, and 10, 30, and 60 min after 25% blood loss.

In the control experiments immobilization for 2 min without blood loss led to inhibition of activity of the sphincter and duodenum; this activity, moreover, recovered completely during the first 5 min after bleeding. The pulse rate rose during immobilization on average by 12% and recovered after 10-20 min. Changes in activity of the sphincter and duodenum were observed, depending on the size of the blood loss, 10, 30, and 60 min after bleeding.

During the 5% blood loss bursts of action potentials were completely abolished from the trace of electrical activity both of the pylorus and of the duodenum (Fig. 1). At the 10th minute after the end of blood loss about 50% of bursts of action potentials from the sphincter and duodenum was restored. Restoration of activity of the sphincter was virtually complete at the 30th minute after blood loss, when activity of the duodenum was not fully restored. Activity of the smooth muscles of the pyloroduodenal zone 1 h after blood loss did not differ significantly from the control (Table 1). The heart rate after 5% blood loss was increased on average by 19% and was restored after 10-20 min.

Consequently, a 5% arterial blood loss caused inhibition of activity of the smooth muscles of the pyloroduodenal zone; recovery of this activity, moreover, took place 30-60 min after blood loss.

During a 10% arterial blood loss complete disappearance of bursts of action potentials of the smooth muscles of the sphincter and duodenum was observed (Fig. 2). Activity of the sphincter was restored only one-third as strongly 10 min after the end of blood loss as activity of the duodenum. At the 30th minute after this blood loss the degree of recovery of activity of the duodenum was greater than that of the sphincter. Complete recovery of duodenal activity, accompanied by weakened activity of the sphincter, was observed 1 h after blood loss (Table 1). The heart rate during 10% blood loss rose on average by 22% and recovered after 20-40 min.

Consequently, a 10% arterial blood loss inhibited muscular activity in the pyloroduodenal zone, and recovery of activity of the duodenum after blood loss was more marked than recovery of activity of the pyloric sphincter.

During 25% arterial blood loss complete inhibition of activity of muscles of the pyloroduodenal zone was observed (Fig. 3). Activity of the sphincter was restored 3.6 times less strongly 10 min after the end of blood loss than activity of the duodenum. At the 30th minute after 25% blood loss activity of the sphincter had recovered by less than 50%, whereas activity of the duodenum was more than 1.5 times higher than initially. Predominance of duodenal activity over activity of the sphincter also

TABLE 1. Frequency of Bursts of Action Potentials of Smooth Muscles of Pyloric Sphincter and Duodenum before and after Blood Loss ($M \pm m$)

Magnitude of blood loss (%)	Number of experiments	Location of electrodes	Burst of freq. of action potentials (min^{-1})			
			before blood loss (control)	after blood loss		
				at 10th minute	at 30th minute	at 60th minute
5	9	Pyloric sphincter	2,7±0,4 (100)	1,3±0,3* (48)	2,6±0,5 (96)	2,2±0,7 (81)
		Duodenum	6,6±0,8 (100)	3,3±0,4* (50)	4,7±0,6* (71)	5,3±0,8 (80)
10	6	Pyloric sphincter	2,7±0,5 (100)	0,7±0,2* (26)	1,2±0,3* (44)	2,1±0,5* (78)
		Duodenum	6,5±0,7 (100)	5,1±0,6* (78)	4,5±0,5* (69)	6,6±0,7 (101)
25	7	Pyloric sphincter	2,6±0,5 (100)	0,5±0,2* (19)	1,1±0,3* (42)	1,0±0,4* (40)
		Duodenum	6,4±0,6 (100)	4,5±0,6* (70)	9,8±1,4* (153)	10,2±1,5* (159)

Legend. Figures in parentheses are percentages. *$p < 0.05$ compared with control.

persisted 1 h after 25% blood loss (Table 1). The heart rate during 25% blood loss rose on average by 39% and did not recover in the course of 60 min.

Consequently, 25% arterial blood loss inhibited muscular activity in the pyloroduodenal zone; duodenal activity, moreover, recovered by a greater degree than activity of the pyloric sphincter.

Thus the stressor action of arterial blood loss inhibits smooth muscle activity in the pyloroduodenal zone, and judging by the tachycardia, this is connected with activation of the adrenergic system. Recovery of myogenic activity takes place more rapidly in the duodenum than in the sphincter, especially after a large blood loss. The results are evidence that blood loss can lead not only to inhibition of activity of the muscles of the sphincter and duodenum, but also to duodenogastric dyskinesia, in the form of predominance of duodenal activity over activity of the pyloric sphincter.

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