CORTICAL REPRESENTATION OF COMPONENTS OF THE GASTRODUODENAL COMPLEX AND ROLE OF THE VAGUS NERVES IN REGULATION OF VISCERAL AFFERENTATION

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There are currently two principal experimental approaches to the study of visceral afferent systems: first, evoked activity is recorded in different parts of the CNS, during electrical stimulation of the central end of the divided nerve trunk (pelvic, splanchnic, etc.); second, spike activity is recorded in single nerve fibers or bundles of fibers, during stimulation of the test organ [3, 4, 5, 6, 7]. Without in any way belittling the advantages of such approaches, it must nevertheless be pointed out that the technique of direct electrical stimulation of part of a test organ followed by recording evoked potentials (EP) in structures of the CNS, which is used comparatively rarely, can contribute to a fuller elucidation of the central projections of these regions. The use of direct electrical stimulation of the sinus node zone has enabled a detailed picture to be produced of the representation of this functionally important region of the heart in the cerebral cortex [1]. Detailed corticography has not yet been used as a method of studying the representation of components of the gastroduodenal complex (GDC). The role of the vagus nerves in the regulation of visceral afferentation likewise has not been fully explained. At the same time, it is evident that the solution of these problems would not only widen our existing ideas on the physiology of visceral afferent systems, but would also deepen our understanding of the mechanisms of development of various pathological states of the visceral organs.

The aim of this investigation was to study the cortical representation of parts of the stomach (fundus, body, and pylorus) and also of the duodenal bulb when the vagus nerves are intact and after bilateral trunk vagotomy.

EXPERIMENTAL METHOD

Acute experiments were carried out on 18 adult cats weighing about 3 kg, under chloralose anesthesia (40-50 mg/kg) and muscle relaxation. The investigation was conducted on an empty stomach, not less than 12-14 h after the last meal. After laparotomy, bipolar stimulating electrodes (area of the silver contacts 4 mm^2, interelectrode distance 10 mm from the centers of the contacts), insulated from the surrounding tissues, were fixed with interrupted sutures to the visceral peritoneum in the region of the fundus, body, and pylorus of the stomach and also of the duodenal bulb. The operation wound was closed without drainage. The structures of GDC were stimulated with square pulses (duration 0.3 msec, amplitude 6-10 mA, frequency 0.1 Hz) from an ÉSU-2 electrostimulator. EP were recorded from the exposed cortex of both cerebral hemispheres by an active silver electrode (the reference electrode was fixed in the frontal bone). The EP were amplified and recorded on a "Neuroaverager" (OTE Biomedica, Italy) on coherent averaging mode with 10 presentations. Throughout the experiment the animals' temperature was kept constant and the brain surface was irrigated with warm (37°C) physiological saline. In half of the experiments both vagus nerves were divided simultaneously by means of special neurotomes, and they were blocked at the level of the thyroid cartilage by injection of a 2% solution of lidocaine through a catheter fixed to the nerve trunks.

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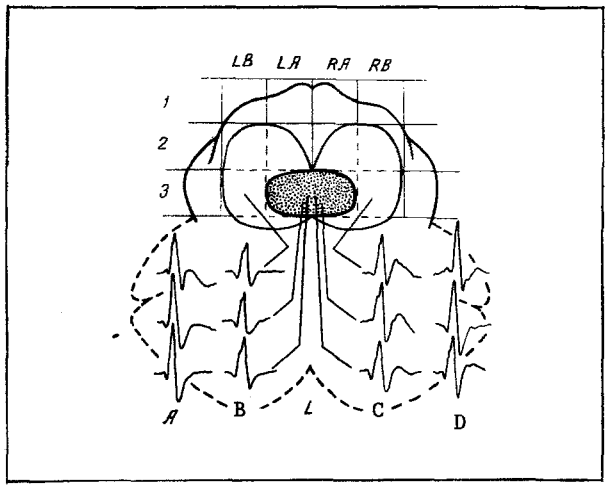

Fig. 1. EP recorded in FMA (location of FMA for each part of GDC indicated by arrows) of cortex of left (A, B) and right (C, D) cerebral hemispheres during electrical stimulation of duodenal bulb (top series of traces), body of stomach (middle series), and pylorus (bottom traces), with intact vagus nerves (B, C) and after bilateral trunk vagotomy (A, D). Calibration: 30 μV, 20 msec.

TABLE 1. Amplitude of EP Recorded during Experiments

Part of GDC stimulated \ Amplitude of phases of EP, in μV	Left hemisphere		Right hemisphere	
	initially	after vagotomy	initially	after vagotomy
Body of stomach				
Phases 1 and 2	70,7±8,6	72,1±6,15	78,7±1,99	89,8±3,9*
Phases 3 and 4	74,1±11,6	96,9±15,02	83,7±6,5	121±15,9*
Pylorus				
Phases 1 and 2	81,7±4,9	94,1±7,3	71,3±3,9	88,3±2,14*
Phases 3 and 4	101,4±8,4	131,9±10,9*	74,3±3,6	120,7±6,2*
Duodenal bulb				
Phases 1 and 2	89,8±5,4	111,6±2,03*	81,3±4,21	93,3±3,45*
Phases 3 and 4	110,4±9,03	134,6±6,3*	77,4±4,16	128,7±12,9*

Legend. Asterisk indicates significant increase in amplitudes of phases of EP after vagotomy compared with initial values.

EXPERIMENTAL RESULTS

Electrical stimulation of the body and pylorus of the stomach and the duodenal bulb led to the formation of distinct EPs in the cortex of both right and left hemispheres. Stimulation of the fundus of the stomach at the same intensity did not cause the appearance of any visible potentials. The EP were recorded after a latent period of 13.3 ± 0.13 msec and had the appearance of multiphased fluctuations of potential with initial positivity (Fig. 1). The focus of maximal activity (FMA) of EP from all three components of GDC was located caudally to the cruciform fissure, corresponding to areas LA-3 and RA-3 according to the detailed corticography data. FMA for the body and pylorus of the stomach was bounded by the cortex of these same zones of the left and right hemispheres, whereas FMA for the duodenal bulb was wider and extended symmetrically to zones La-2-3-4, LB-2-3, and RA-2-3-4, and RB-2-3, while preserving the shape and amplitude of the phases of EP and the short latent period within these limits. With increasing distance from FMA the EP acquired an early negative component and the amplitude of the phases of potentials fell.

After identification of the position of FMA for all the structures stimulated, one-stage bilateral vagotomy or blocking conduction along the vagus nerve fibers by means of lidocaine solution was carried out. Pharmacological vagotomy had the aim of eliminating any possible effect of nerve trauma on the results. In all cases vagotomy led to changes in amplitude of the phases of EP without any change in the latent period and shape of the potentials. The combined amplitude of the initial positive-negative

deviation of potential (phases 1 and 2) and of the secondary positive-negative wave (phases 3 and 4) was analyzed. Statistical analysis of these results revealed the patterns reflected in Table 1.

The results demonstrate that with the vagus nerves intact, the amplitude of the initial phases of EP during stimulation of these parts of GDC did not differ significantly in the cortex of the two hemispheres. The combined amplitude of phases 3 and 4 of EP in the left hemisphere was significantly greater during stimulation of the pyloric part of the stomach (101.1 ± 8.4 μV and 74.3 ± 3.6 μV, respectively, $p < 0.05$) and of the duodenum (110.4 ± 9.03 μV and 77.4 ± 4.16 μV, $p < 0.001$). A tendency was also noted for greater dominance of the left hemisphere for these parts of GDC and of the right hemisphere for the body of the stomach.

Bilateral vagotomy, regardless of how it was done, not only did not disturb EP formation in the cerebral cortex but, on the contrary, it led to an increase in amplitude of all phases of EP. In the right hemisphere the combined amplitude of phases 1 and 2 of EP was increased statistically significantly in all cases, whereas the combined amplitude of phases 3 and 4 was increased during stimulation of the pylorus and the duodenal bulb. In the left hemisphere all phases of EP were significantly increased in amplitude of phases 3 and 4 to stimulation of the pylorus also was significant. Comparison of the combined amplitude phases 1 and 2 of EP recorded in the left and right hemispheres after vagotomy revealed significant dominance the left hemisphere for stimulation of the duodenal bulb (111.6 ± 2.03 μV and 93.3 ± 3.45 μV, $p < 0.01$) and of the right for stimulation of the body of the stomach (89.8 ± 3.9 μV and 72.1 ± 6.15 μV, $p < 0.01$). These differences are illustrated by the series of traces in Fig. 1.

The experiments thus showed that the structures of GDC studied have bilateral representation in the cerebral cortex in zones LA-3 and RA-3 — caudally to the cruciform fissure; the regions of projection of the duodenum, moreover, are much wider than those of the body of the stomach and pylorus. All the tests were conducted under standard conditions (before a meal), for there is evidence of the inhibitory effect of a functional load on spontaneous spike activity in the afferent fibers of the pyloroantral part of the stomach [4]. The absence of EP to stimulation of the fundus of the stomach may be connected with the long "hungry" period or with the character of innervation and of the function of this part of the stomach. The clarification of these issues was not among the aims of this investigation. In our view the most important fact is that bilateral trunk vagotomy (or pharmacological "vagotomy") causes facilitation of visceral afferentation, manifested by an increase in amplitude of the phases of EP. These results do not contradict the widely known data on the role of the vagus nerves in visceral afferentation [2], but they demonstrate their new functional role as regulators of the afferent "output" from the internal organs to the CNS. It was shown previously that bilateral vagotomy likewise does not inhibit (it may even facilitate to some extent) cardiac afferentation [1]. This, in turn, suggests an essential role for the vagus nerves in the regulation of visceral afferentation as a whole. After vagotomy, afferent volleys from the stimulated parts of GDC reach the cerebral cortex via the spinal afferent systems. The increase in amplitude of the phases of potentials in these cases is evidently a reflection not so much of the unimportant role of the vagus nerves in the transmission of impulsation ascending to the CNS, but rather of the elevation of thresholds of excitability of the stimulated structures, when they have lost the efferent depressive influences of the vagus nerves, realized at the level of the intramural nervous system of the organs.

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