

ELECTRICAL POTENTIALS OF THE PAROTID
SALIVARY GLAND RECORDED FROM THE
BODY SURFACE IN CATS

M. A. Sobakin* and V. P. Makhnev

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Correlation was found between the dynamics of the electrical potential on the surface of the parotid salivary gland and on the corresponding body surface in cats during stimulation of the secretory nerves and other procedures. The absence of a potential gradient when the electrode was on the body surface confirms the macrostructure of the global field of the salivary gland. The experimental results indicate the glandular origin of electrical responses on the body surface.

KEY WORDS: electrical potential of the salivary gland; field macrostructure; glandular origin.

The electronegativity of the outer surface of the submandibular and electropositivity of the sublingual salivary glands relative to the chyle in response to stimulation of the secretory nerves are linked with the global effect of electrical responses of the acinar cells to various degrees of hyperpolarization of their basal and apical membranes [3, 9, 10]. The macroscopic nature of the global effect points to the suitability of a distant investigation of electrical potentials of the salivary glands and, in particular, their recording from the body surface.

It was shown previously that on the surface of the human body in the region of the parotid salivary gland changes in electrical potential correlate with the dynamics of secretion of saliva by the gland [5], and that the distribution of electrical potentials points to a dipole representation of the equivalent source [6].

To study the relationship between the dynamics of electrical potentials on the body surface and on the surface of the salivary gland, and to obtain detailed information on the formation of electrical responses, an experimental investigation was made of electrical activity of the parotid salivary gland in cats.

EXPERIMENTAL METHOD

In eight cats anesthetized with pentobarbital (0.04-0.05 g/kg) and in nine cats anesthetized with urethane and chloralose (0.5 and 0.05 g/kg, respectively) a secretory response was induced by electrical stimulation of the auriculotemporal and sympathetic nerves supplying the salivary gland [7, 8], and also by subcutaneous injection of 0.5-0.7 mg/kg pilocarpine. A parasympathetic block was induced by subcutaneous injection of 3 mg atropine. The duct of the parotid gland was cannulated and the level of secretion recorded. Electrical potentials were derived from the body surface and also directly from the fascia covering the salivary gland. In both cases the reference electrode was placed in the same position 3-5 cm away from the gland. Nonpolarizing Zn-ZnSO₄ electrodes with a wick 3-4 mm in diameter were used to pick up the potentials, which were recorded within a frequency band from 0 to 0.1 Hz [5]. In five experiments the dynamics of the electrical resistance of the salivary gland was recorded simultaneously by the method described in [4]. Altogether about 500 responses to these procedures were recorded.

In response to stimulation of the secretory nerves the electrical potential of the parotid gland changed to negative (Fig. 1A, B) and did not differ significantly from the response recorded on the body surface (Table 1). In response to stimulation of the auriculotemporal nerve electronegativity developed faster than to stimulation of the sympathetic nerve ($P < 0.01$) and it was accompanied by a marked secretory response. The ab-

*Corresponding Member of the Academy of Medical Sciences of the USSR.

TABLE 1. Electrical Responses to Stimulation of Secretory Nerves ($M \pm m$)

Index	Recording	Stimulation of nerve	
		auriculo-temporal	sympathetic
No.; n	G B	4; 16 8; 64	5; 32 4; 25
A, mV	G B	$1,39 \pm 0,20$ $1,31 \pm 0,12$	$0,75 \pm 0,09$ $0,72 \pm 0,14$
t, mV/min	G B	$20,3 \pm 4,9$ $14,5 \pm 1,2$	$2,0 \pm 0,4$ $1,7 \pm 0,2$

Legend. No.) No. of experiments, n) number of responses, A) amplitude of response, t) rate of change of electrical potential, G and B) recording from surface of gland and body surface, respectively.

sence of any significant potential gradient as the recording electrode was moved away on the body surface is evidence of the macroscopic structure of the global electric field.

The independence of the electrical response of compression of the parotid duct and also of the subsequent rapid flow of saliva on releasing the duct (number of responses $n = 35$), just as in the case of the submandibular salivary gland [9], indicates that it is unrelated to the secretion of saliva. The phenomenon of increased secretion [2] with its initial changes on account of "sympathetic" activation of muscle cells likewise was not accompanied by a correlated change in electrical potential (Fig. 1C, D, $n = 40$), indicating no connection between the electrical and muscular responses of the salivary gland.

Stimulation of the auriculotemporal nerve 15-20 min after injection of atropine neither induced secretion nor caused any change in the electrical potential ($n = 12$), whereas the electrical resistance of the gland decreased, indicating the development of vasodilatation. In the same way, an increase in the pressure in the

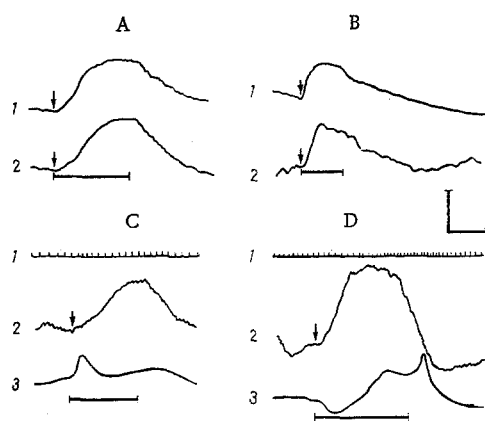


Fig. 1. Electrical potentials of parotid gland. A and B) Dynamics of electrical potential of parotid gland (1) and potential on body surface (2) during stimulation of sympathetic (A) and auriculotemporal (B) nerves. C and D) Examples of secretory (1,3) and electrical (2) responses to stimulation of sympathetic nerve against the background of secretion induced by pilocarpine or by stimulation of auriculotemporal nerve; 3) secretory response as a function of time T between drops of saliva; $f \sim 1/T$. Duration of stimulation shown by horizontal line below records. Time scale 1 min; calibration: 1 mV for A, B, and C, 0.3 mV for D.

ducts ($n = 14$) and retrograde injection of saliva into the ducts ($n = 17$), leading to vasodilatation of the gland [1] and accompanied in the present experiments by a reduction of electrical resistance, did not change the electrical potential when recorded by the two methods.

Considering previous data [8-10], these findings indicate the glandular origin of the electrical responses. Much greater hyperpolarization of the apical membranes than of the basal membranes must be assumed in the acinar cells of the parotid gland. Should the acini be oriented away from the hilus of the gland, summation of the set of dipole-acini into one equivalent source would take place, the distance from the gland to the surface of the body would be much less than the size of the source, and the potential gradient would be absent. Such a model, in agreement with the experimental data, explains the electrical response on the surface of the body and allows it to be used for investigations of glandular tissue.

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EFFECT OF ACETYLCHOLINE ON DISCHARGE FREQUENCY AND SHAPE OF ACTION POTENTIALS OF PACEMAKER CELLS

G. I. Bochkina, G. S. Sukhova,
and M. G. Udel'nov

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The action of exogenous acetylcholine on the isolated pacemaker of the frog heart was studied. Activity of single cells and the total activity of the preparation were recorded. Exogenous acetylcholine had not only an inhibitory, but also an accelerating effect on the rhythm of discharge of the pacemaker cells. As a rule an accelerating action was observed when relatively low concentrations of acetylcholine were used. An increase in the rate of rise of slow diastolic depolarization during the development of parasympathetic acceleration is evidence of the active mechanism of this process. The difference in the effects of acetylcholine was probably due to differences in the action of large and small concentrations of the drug on transmembrane ionic currents.

KEY WORDS: acetylcholine; pacemaker cells; acceleration of rhythm; inhibition of rhythm.

Investigations have shown that the parasympathetic innervation of the heart can cause opposite effects on the heart beat [4, 5]. It has recently been found that impulses acting through cholinergic mediator can either delay or accelerate the development of slow diastolic depolarization (SDD) and, at the same time, delay or accelerate the discharge frequency of pacemaker cells [1, 3]. Similar studies of the action of exogenous acetylcholine (ACh) on the development of SDD and on the rhythm of the pacemaker cells have not been under-

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