RESISTANCE OF FROG OLFACTORY BULB SLOW POTENTIAL AND AFFERENT INPUT INHIBITION TO HYPOXIA AND SYNAPTIC TRANSMISSION BLOCKADE BY MANGANESE, COBALT, AND MAGNESIUM IONS

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The effect of hypoxia and application of manganese, cobalt, and magnesium ions on electrical responses of the frog olfactory bulb to adequate stimulation and to direct electrical stimulation of the olfactory nerve were studied. The slow potential evoked by adequate stimulation and the associated inhibition of the afferent input of the olfactory bulb were found to be much more resistant to inhibition of synaptic transmission by all methods used than the postsynaptic components of the orthodromic response and associated postsynaptic inhibition. A slow potential was recorded even when synaptic transmission in the olfactory bulb was completely blocked by magnesium ions. It is concluded that the slow potential of the olfactory bulb and inhibition of its afferent input are nonsynaptic in nature. It is postulated that the slow potential reflects mainly depolarization of glial cells in the glomerular layer of the bulb evoked by accumulation of potassium ions. The possible mechanisms of inhibition of the afferent input are discussed.

INTRODUCTION

It can now be taken as established that an important role in information processing in the olfactory bulb (OB) is played by inhibition of its afferent input. The mechanisms of this inhibition are located in the region of the glomeruli, where olfactory impulses relay from axons of receptor cells to second-order neurons. This inhibition is expressed as depression of all postsynaptic potentials in OB evoked by a single orthodromic volley in the course of many seconds after directing a jet of odoriferous or ordinary room air on to the olfactory epithelium of land vertebrates [2, 5], and also after the action of certain chemical stimuli and of a jet of water on the fish olfactory epithelium [8]. A matter of great importance in the study of the mechanisms and role of this inhibition of the OB input is that, simultaneously with it, a long negative potential (what Ottoson [20] calls the slow potential) is recorded in the region of the glomeruli, and as has been shown [4], it correlates well in its time course with suppression of the postsynaptic components of the orthodromic potential (OP). It has been suggested that depression of the afferent input of OP is due to presynaptic inhibition [3] and that the slow potential (SP) reflects mainly depolarization of primary olfactory afferents - axons of olfactory receptors - and that it is the analog of the dorsal root potential of the spinal cord [4, 7]. However, the concrete mechanism of this presynaptic inhibition and the nature of the SP still remain unexplained, for no axo-axonal synapses have been

Fig. 1. Effect of hypoxia on electrical responses and inhibition in OB.

a) Time course of changes in amplitude of SP (1) and of first (2) and second (3) components and of positive wave (4) of OP during hypoxia. Abscissa, time from moment of stopping blood flow, h; ordinate, amplitude of potential, mV. b, c) Depression of second component of OP after single electrical (b) and adequate (c) stimulation in control (1) and 2 h 30 min after beginning of hypoxia (2). Abscissa, interval between conditioning and testing stimuli, sec; ordinate, amplitude of response to testing stimulation, % of control. d) Lengthening of SP during hypoxia. Short vertical lines indicate time when SP falls to half its amplitude. Time from beginning of hypoxia, in min, shown on left. e) Another example of depression of first (circles) and second (triangles) components of OP after adequate stimulation. Control) empty circles and triangles. Control and experimental values located in region surrounded by thin lines. Remainder of legend as in Fig. 1c.

found on fibers of the olfactory nerve in the course of electron-microscopic study of the glomerular layer of OB [10, 14, 21]. Meanwhile, elucidation of the nature of the SP and the mechanisms of inhibition of the afferent input of OB is also of great importance to an understanding of the principles of operation and the functional role of the glomerular formations of the brain as such [23].

In the investigation described below the effect of both nonspecific inhibition of synaptic transmission in OB during hypoxia, and of more selective blocking of the mechanism of transmitter release in the synapses as a result of application of Mn++, Co++, and Mg++, on electrical responses of OB evoked by adequate stimulation and by single direct electrical stimulation of the olfactory nerve was studied [13, 18, 19, 24]. In addition, changes in inhibitory processes in OB due to the two types of activation of its input were studied. Evidence is given in support of the nonsynaptic nature of the SP and associated inhibition of the afferent input of OB.

METHOD

Frogs (Rana temporaria) were immobilized by intramuscular injection of 2% diplacin solution (0.05-0.1 ml). Craniootomy was performed and the surface of OB and the nerve exposed. The olfactory nerve was stimulated through a bipolar nichrome wire electrode with pulses of current (20-45 V, 0.1 msec). The adequate stimulus was blowing a short jet of room air, by means of a syringe so that the volume and velocity of the jet could be controlled, on to the olfactory epithelium, exposed by removal of the roof of the nasal cavity. The volume of air was 3-7 ml. Evoked potentials were derived from the surface of OB by means of nonpolarizing Ag-AgCl electrodes. The potentials were amplified by a dc amplifier (band 0-1000 Hz) and photographed on motion picture film. Conditions of hypoxia were created by application of a ligature to the vessels leaving the heart, and arresting the blood flow in the intact animal or after decapitation. To obtain more selective inhibition of synaptic transmission, solutions

\*1,3-Di(β-platynecinmethoxy)benzene hydrochloride.