PHARMACOLOGICAL ANALYSIS OF SLOW POTENTIALS RECORDED IN FROG OLFACTOR Y BULB DURING NATURAL STIMULATION

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Pharmacological agents (strychnine, picrotoxin, pentobarbital, chloralose, GABA, penicillin, morphine) were used to investigate the nature of the slow potential recorded in the frog olfactory bulb in response to natural stimulation. Three possible hypotheses were tested: 1) The slow potential is neuroglial in nature; 2) it is the analog of the dorsal-root potential of the spinal cord and reflects depolarization of primary afferents arising in the terminals of the olfactory nerve and responsible for presynaptic inhibition in the frog olfactory bulb; 3) the slow potential reflects postsynaptic processes. The results showed great similarity between changes in the slow and dorsal-root potentials of the spinal cord in response to the action of pharmacological agents. However, the slow potential is evidently a complex response and incorporates at least one other component — depolarization of the dendrites of unknown nature.

INTRODUCTION

As Ottoson [12, 13] first showed, the slow potential (SP) in the frog and rabbit olfactory bulb is characterized by a steep rising phase, in which fast potentials (FPs) are recorded, and a slow decline. Its total duration is several seconds. In the rostral part of the olfactory bulb the SP is negative, but in the layer of glomeruli its sign is reversed (Fig. 1). It is claimed that the SP is generated in the glomeruli [12, 13, 19], but whether pre- or postsynaptic structures are responsible for its generation is not yet clear. According to Ottoson, for example, the SP is generated by structures responsible for the P1 component of the orthodromic potential, i.e., mainly presynaptic structures, and it plays an important role in the activation of secondary neurons and in FP generation. However, Ueki and Fukuda [17] showed that nictethamide can selectively block SPs; under these circumstances the FPs are increased. These workers concluded that no relationship of cause and effect exists between the SPs and FPs and that the SP has no significant effect on the olfactory afferent activity of the olfactory bulb. The genesis of the SP and its functional role thus remain uncertain. It was therefore decided to carry out a pharmacological analysis of the SP. Pentobarbital, chloralose, strychnine, picrotoxin, GABA, penicillin, and morphine were chosen as the pharmacological agents for the following reasons. On the whole, the results pertaining to the SP parameters, which agreed with Ottoson's findings, showed that the SP corresponds in its shape and temporal course to the inhibition which affected the orthodromic potential (OP) after natural stimulation. The present writers showed previously that presynaptic inhibition arises in the olfactory bulb [1, 2]; it was therefore suggested that this process of inhibition may also be presynaptic and that the SP reflects predominantly afferent depolarization of primary afferents (DPA), and this lies at the basis of the inhibition of OP after natural stimulation. Most of the drugs specified have been used previously to study presynaptic inhibition and the dorsal-root potential (DRP) in the spinal cord of cats and frogs [8, 10, 15]. The second hypothesis was that the SP reflects certain postsynaptic processes. This hypothesis was tested with the aid of the same pharmacological agents, for which purpose parallel recordings also were made of the OPs and FPs, a previous analysis [3] of which revealed their concrete postsynaptic nature. Finally, the third initial hypothesis was that the SP is neuroglial in nature, in which case it must be modified by GABA, strychnine,
EXPERIMENTAL METHOD

Experiments were carried out on frogs (Rana temporaria) immobilized by intraperitoneal injection of 0.3% diplacin. The olfactory nerve (orthodromic stimulation) was stimulated electrically and the olfactory epithelium by a jet of air (natural stimulation). Activity was recorded with Ag—AgCl agar-agar electrodes. A piece of filter paper soaked in a solution of the pharmacological agent was applied as near as possible to the recording electrode. Fuller details of the method were given earlier [3].

RESULTS

Effect of Pentobarbital and Chloralose. Pentobarbital (0.8%) was applied in seven experiments and chloralose (1-1.5%) in nine. Both substances greatly increased the duration of SP in all the experiments: pentobarbital by 52% and chloralose by 80% on the average. The amplitude of SP in the first case was increased on the average by 29%, whereas in the second case it was reduced in five experiments (on the average by 18%), increased in three (by 16%), and remained unchanged in one. Parallel with the increase in duration of SP, the duration of the recovery cycle of the P1 and P2 components of OP increased in all experiments with pentobarbital and in six of the nine experiments with chloralose (Fig. 2). The increase in amplitude of SP in four experiments with pentobarbital corresponded to deepening of inhibition of the recovery cycles of P1 and P2, but in the other three experiments this was not observed. So far as the effect of these drugs on the amplitudes of P1, P2, and FP is concerned, both substances were found to depress P2 and FP: pentobarbital on the average by 26 and 17%, respectively, chloralose by 36 and 35%, respectively. The duration of the changes usually exceeded 30-40 min. Pentobarbital also reduced (on the average by 13%) the amplitude of P1 in five experiments and left it unchanged in two. Chloralose increased the amplitude of P1 (approximately by 35%) in seven experiments and left it unchanged in two.