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A DEPOLARIZING INHIBITORY POTENTIAL IN NEURONES OF THE OLFACTORY CORTEX *IN VITRO*

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SUMMARY

1. Stable intracellular recordings were obtained from neurones in slices of the guinea-pig olfactory cortex maintained *in vitro*.

2. Single stimuli applied to the lateral olfactory tract (l.o.t.) produced an excitatory post-synaptic potential (e.p.s.p.) usually generating a single spike.

3. The e.p.s.p. was followed by a long (200–500 msec) after-depolarization (l.a.d.) of peak amplitude 5–16 mV. This was accompanied by a very large conductance increase and was associated with an inhibition of the intracellularly recorded e.p.s.p. and of spike generation.

4. The l.a.d. was more susceptible than the e.p.s.p. to depression by (i) repetitive l.o.t. stimulation and (ii) raising external [Mg^{2+}]. The l.a.d. could be generated without a preceding spike.

5. At an average resting membrane potential of -74 mV the average reversal potential for the l.a.d. ($E_{l.a.d.}$) was -63 mV. $E_{l.a.d.}$ became more positive on reducing $[Cl^-]_{out}$ or on using KCl-filled electrodes.

6. It is concluded that the l.a.d. represents a Cl^--mediated inhibitory post-synaptic potential, generated through deep-lying recurrent inhibitory loops.

INTRODUCTION

In the preceding paper (Scholfield, 1978*a*) some basic properties of superficial neurones in slices of guinea-pig olfactory cortex maintained *in vitro* were described. These neurones receive a direct excitatory synaptic input from fibres in the lateral olfactory tract (l.o.t.). There have been several descriptions of extracellular (field) potentials in this preparation evoked by l.o.t. stimulation (Yamamoto & McIlwain, 1966; Richards & Sercombe, 1968; Harvey, Scholfield & Brown, 1974; Halliwell, 1976). The present paper reports the effects of l.o.t. stimulation as revealed with intracellular electrodes. Essentially two synaptically evoked responses were detected: an initial excitatory post-synaptic potential (e.p.s.p.) and a prolonged after-depolarization (l.a.d.) distinguishable from the spike after-depolarization referred to in the preceding paper (Scholfield, 1978*a*). Attention is concentrated primarily on the

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l.a.d. and evidence is presented to suggest that it represents an inhibitory post-synaptic potential. A preliminary communication of these results has been presented (Scholfield, 1976).

METHODS

Synaptic potentials were recorded from forty-four neurones in the guinea-pig olfactory cortex *in vitro* with intracellular electrodes as previously described (Scholfield, 1978*a*). Extracellular synaptic potentials were recorded from the pial surface using glass micropipettes filled with 0·15 M-NaCl, with resistance about 5 MΩ. These were placed within 1 mm of the intracellular recording electrode. The excitatory afferent input to the cell was stimulated using a pair of platinum electrodes (0·5 mm in diameter, 2 mm apart and insulated to the flattened tips) placed across the l.o.t. 1 mm from the severed end at the junction of the olfactory bulb. Stimuli were delivered to the l.o.t. at less than 0·1 Hz from a Devices 2533 output unit with supramaximal pulses of 5 V and 0·2–0·5 msec duration.

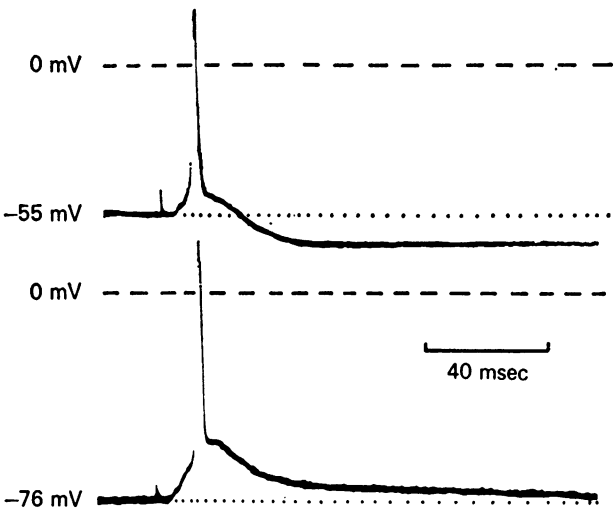

Fig. 1. An intracellular recording from a synaptically activated neurone in the olfactory cortex. The upper trace is a recording on stimulating the l.o.t. immediately after impalement when the membrane potential was − 55 mV. After 5 min, this increased to a stable value of − 76 mV when the l.o.t. was again stimulated. The dashed line represents a zero recorded potential with the electrode outside the cell and the dotted line is the recording base line.

RESULTS

As noted previously (Scholfield, 1978*a*) the resting potential (E_m) of the superficial neurones was usually quite low (< 50 mV) immediately after impalement, but thereafter increased gradually to a stable value of around − 75 mV.

Stimulation of the l.o.t. during the initial period of low membrane potential produced, sequentially, (i) a transient (20–30 msec) depolarization usually with a superimposed single spike and (ii) a prolonged (200–500 msec) hyperpolarization (Fig. 1*A*). Subsequent stimulation during the stable high membrane potential state produced a larger initial depolarization followed by a prolonged after-*de*polarization.

The initial depolarization, commencing after a latency of around 5 msec, has the characteristic of an excitatory post-synaptic potential (e.p.s.p.). The after-hyper-

polarization in Fig. 1*A* and the after-depolarization in Fig. 1*B* have rather similar time courses and are assumed to represent the same phenomenon. It is hereafter referred to as the *late after-depolarization* (l.a.d.). This l.a.d. has quite different properties to the shorter depolarization following the spike (Scholfield, 1978*a*).

Properties of the l.a.d.

In Fig. 1 the absolute values of the membrane potentials attained during the after-hyperpolarization and after-depolarization were very close (−67 and −69 mV respectively). If the only difference between these two situations was the different initial resting potential, this suggests that (*a*) membrane conductance is very high during the l.a.d. and (*b*) the l.a.d. reverses in direction at around −68 mV. These

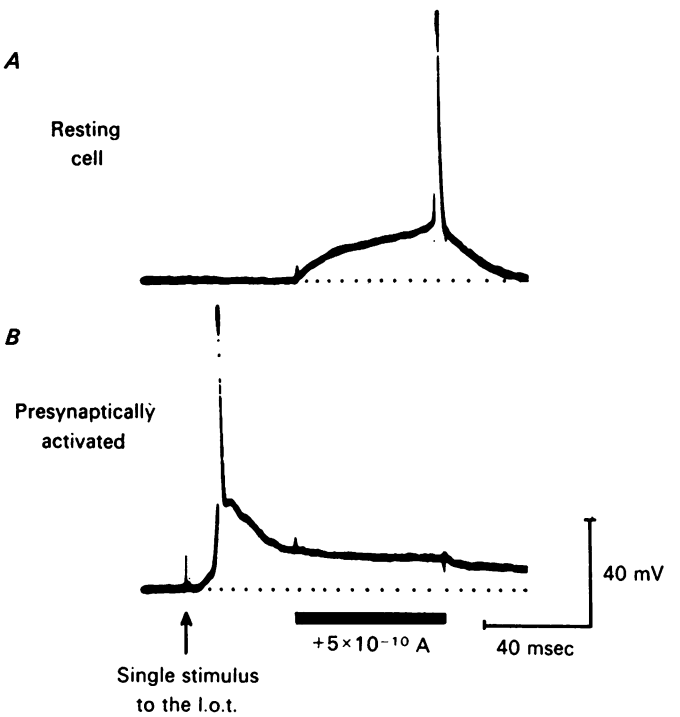

Fig. 2. An intracellular recording from a synaptically activated neurone in the olfactory cortex. *A*, the result of the application of a depolarizing pulse of 0·5 nA passed through the recording electrode in the resting neurone during the period marked by the bar. This produced an action potential towards the end of the pulse and represents an input resistance of 50 Mohm. *B*, the affect of synaptic activation when an e.p.s.p., a spike and a l.a.d. were generated. During the l.a.d. 0·5 nA of depolarizing current was again passed through the electrode (marked by the bar and the current artifacts on the intracellular recording). Scales: 40 mV and 40 msec. The membrane potential was −76 mV.

properties were confirmed in further experiments on the l.a.d. alone, generated at high initial resting potentials.

Thus, Fig. 2 shows the effect of applying a depolarizing current pulse of 0·5 nA and 50 msec duration during a l.a.d. which followed an e.p.s.p. generated at −76 mV. In the upper record (*a*) the pulse was passed before l.o.t. stimulation, and produced a

depolarizing potential of 27 mV amplitude, just sufficient to generate a spike. Application of the same pulse during the l.a.d. (in the lower trace) produced a negligible voltage excursion and failed to generate a spike. Two points clearly emerge. *First*, the input resistance of the cell has fallen dramatically during the l.a.d., from around 50 MΩ to less than 5 MΩ. *Secondly*, in the face of this high membrane conductance, the membrane potential at the peak of the l.a.d. must be very close to the null potential for the ionic mechanism generating the l.a.d. (or equilibrium potential, $E_{l.a.d.}$) about -60 mV in this instance.

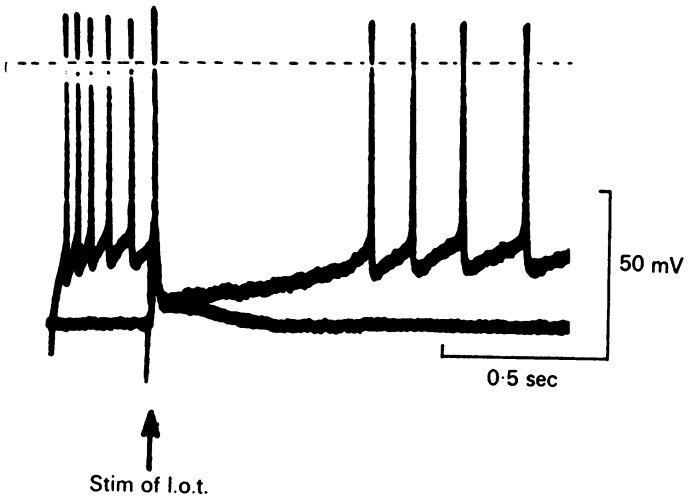

Fig. 3. The l.o.t. was stimulated in the resting cell (lower trace). An e.p.s.p. followed by a l.a.d. were generated. The cell was depolarized 27 mV by passing 0·3 nA through the recording electrode and balancing out the electrode resistance. During the train of resulting spikes, the l.o.t. was again stimulated which produced a barely discernible e.p.s.p., followed by a prolonged hyperpolarization. Scales: 50 mV, 0·5 sec. Dashed line is zero recorded potential.

Fig. 3 shows the converse experiment of depolarizing the neurone with a long positive current pulse and then stimulating the l.o.t. during the depolarization. The l.a.d. is reversed in direction to a hyperpolarization such that (as in Fig. 1) the peaks of depolarization and hyperpolarization are approximately coincident at -71 mV.

Equilibrium potentials for the l.a.d. From a number of experiments of the type shown in Figs. 2 and 3, $E_{l.a.d.}$ was determined as $-62{\cdot}9 \pm 0{\cdot}7$ mV (mean ± S.E. of mean; twenty-nine cells). The average resting potential in the same neurones was $74{\cdot}3 \pm 0{\cdot}5$ mV.

Equilibrium potential for the e.p.s.p. The equilibrium potential for the initial e.p.s.p. ($E_{e.p.s.p.}$) could not be measured directly or by extrapolation since the current–voltage curve is highly non-linear in the depolarizing direction (Scholfield, 1978*a*). Estimates of $E_{e.p.s.p.}$ by passing current into the soma may in any case be invalid because the e.p.s.p. is generated in the superficial dendrites some distance from the soma (Biedenbach & Stevens, 1969*a*; Haberly & Shepherd, 1973; Halliwell, 1976). However, it would appear from Figs. 1–3 that $E_{e.p.s.p.}$ lies well in the depolarizing direction from $E_{l.a.d.}$

The normal amplitude of the e.p.s.p. after a single l.o.t. volley was 15–25 mV at the normal resting membrane potential of −75 mV. However, much larger e.p.s.p.s could be obtained under certain circumstances, for example, with the second of two paired shocks 0·2–0·5 sec apart (see later) or in the presence of picrotoxin or bicuculline (10^{-5} M). Under the latter circumstances, the e.p.s.p. attained a value of up to 70 mV (i.e. an E_m of about −5 mV). Since the membrane resistance during this e.p.s.p. is low, then this suggests that $E_{e.p.s.p.}$ is slightly more positive than −5 mV. It may also be noted that *duration* of the e.p.s.p. recorded after a single l.o.t. volley is abbreviated by the subsequent l.a.d. Thus, e.p.s.p.s of several hundred msec duration were recorded in cells where the l.a.d. was suppressed with picrotoxin or bicuculline and in one cell where no l.a.d. could be evoked.

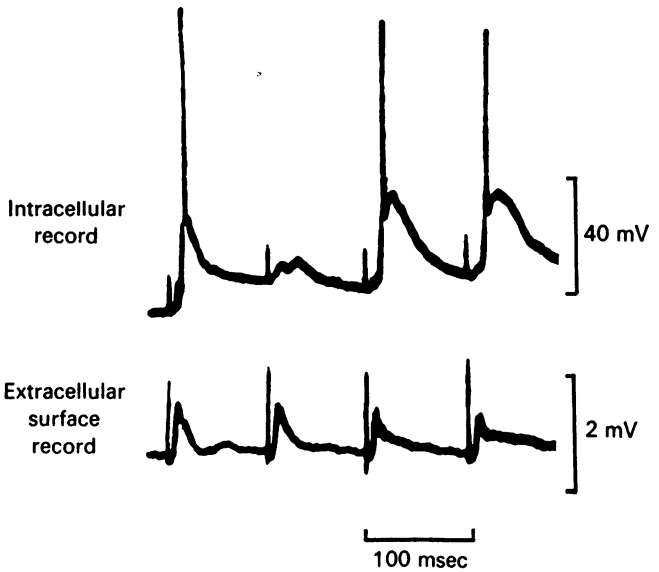

Fig. 4. Simultaneous intracellular (upper trace) and extracellular recordings (lower trace) during a train of 4 stimuli delivered to the l.o.t. at 10 Hz (marked by the stimulus artifacts). Scales: 40 mV for intracellular recording; 2·0 mV for extracellular recording; sweep speed, 100 msec.

Inhibition by the l.a.d. The l.a.d. inhibited spike generation both by direct depolarizing current and also by l.o.t. stimulation. Profound effects of the l.a.d. on direct spike generation are apparent in Figs. 2 and 3. Fig. 4 shows the effect of the l.a.d. on orthodromic transmission. In this test the l.o.t. was stimulated at such a frequency (10 Hz) that the second e.p.s.p. occurred during the l.a.d. following the first impulse. The intracellularly recorded e.p.s.p. (upper trace) was then severely reduced in amplitude and failed to reach the threshold for spike generation. Subsequent e.p.s.p.s recovered. The lower trace in Fig. 4 shows an extracellular record of the evoked potentials recorded from the surface of the slice in the manner described previously (Harvey *et al.* 1974). In contrast to the intracellular record, the extracellularly recorded e.p.s.p. is well-maintained during repetitive stimulation. The most likely reason for this is that the extracellular potential reflects the amplitude of the e.p.s.p.

at its point of generation, in the superficial dendrites. On the other hand, the large conductance increase associated with the l.a.d. suggests that the latter is generated at the soma, and so shunts the electrotonic component of the e.p.s.p. recorded in the soma but not the e.p.s.p. generated in the dendrites. Thus, the depression of the intracellular e.p.s.p. in Fig. 4 may be attributed to the somatic shunt rather than to depression of the e.p.s.p. itself.

Ionic mechanism of the l.a.d. Two lines of evidence suggest a primary role for Cl^- ions in generating the l.a.d. First, the l.a.d. was increased in duration and amplitude by reducing external [Cl^-] (Fig. 5). Secondly, the l.a.d. was more pronounced in cells impaled with Cl^--filled electrodes (Fig. 6), in spite of a lower resting membrane potential (which would otherwise reduce the l.a.d., cf. Fig. 1).

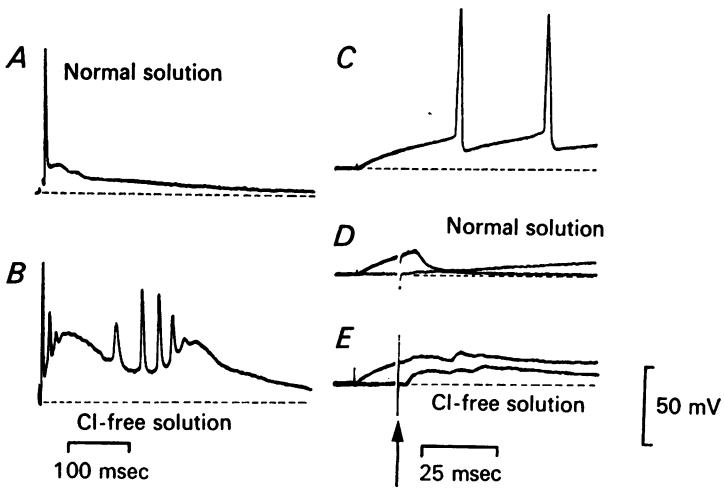

Fig. 5. The effect of solution containing low [Cl] on the response to l.o.t. (*A* and *B*) and local stimulation (*C–E*). *A*, the slice was bathed in normal solution (128 mM-Cl) and the l.o.t. stimulated. *B*, the slice was bathed in a solution containing 23 mM-Cl ('Cl-free solution') for 15 min and again a single supramaximal stimulus delivered to the l.o.t. *C*, the slice was in 128 mM-Cl and 0·2 nA of depolarizing current was passed through the recording electrode, hence initiating two action potentials. *D*, the cell was then synaptically stimulated through a focal tungsten micro-electrode at the arrow under trace *E* (↑), and this produced a small depolarization. +0·2 nA was passed through the electrode and the cell again stimulated synaptically through the focal stimulating electrode. This now resulted in a hyperpolarizing potential superimposed on the depolarizing potential. Thus the reversal potential for this synaptic event was 2 mV less than resting E_m. *E*, the slice was bathed in 23 mM-Cl and the stimulation procedure in *D* repeated. Here, the reversal potential for the synaptic event was about 20 mV less than E_m. The size of the synaptic response was erratic with successive stimuli in contrast to the constant response in normal solution. The stimulus voltage was sub-maximal but variation in this did not produce a synaptic event with a reversal potential any less than 20 mV depolarizing of E_m. The dashed lines represent the extrapolation of the resting E_m. The voltage calibration is 50 mV and the time calibrations are 100 msec (*A* + *B*) and 40 msec (*C* − *E*).

The latter may be interpreted by supposing that leakage of Cl^- from the electrode increases internal [Cl^-] sufficiently to make the Cl-equilibrium potential (E_{Cl}) less negative. The assumptions here are that (*i*) the l.a.d. reversal potential = E_{Cl} and (*ii*) acetate ions do not participate in the conductance increase underlying the l.a.d.

This latter point is appropriate for spinal motoneurone i.p.s.p.s (Eccles, 1957; Araki, Ito & Oscarsson, 1961; Eccles, Eccles & Ito, 1964; Matsuura & Endo, 1971) but appears not to be true for cortical neurone i.p.s.p.s (Kelly, Krnjević, Morris & Yim, 1969). Some additional observations support the view that acetate ions are relatively 'inert' in pyriform neurones. (i) A depolarizing l.a.d. was also recorded using citrate-filled electrodes: citrate and acetate differ with respect to their ability to participate in cortical e.p.s.p.s (Kelly *et al.* 1969). (ii) There was no time-dependent shift in $E_{\text{l.a.d.}}$ with acetate-filled electrodes. (iii) $E_{\text{l.a.d.}}$ was not altered by prolonged negative current (1 nA for 7 min) through the micro-electrode.

K^+ ion movements seem unlikely to fulfil a substantial primary role in generating the l.a.d. because reducing $[K^+]_{out}$ from 6 to 0·5 mM increased l.a.d. by some 5 mV. Since the resting membrane potential increased by 15 mV (see Scholfield, 1978*a*), the increase in l.a.d. might result if changes in E_{Cl} lagged behind the change in resting potential, i.e. the membrane were relatively impermeant to Cl^- ions at rest (Scholfield, 1978*a*).

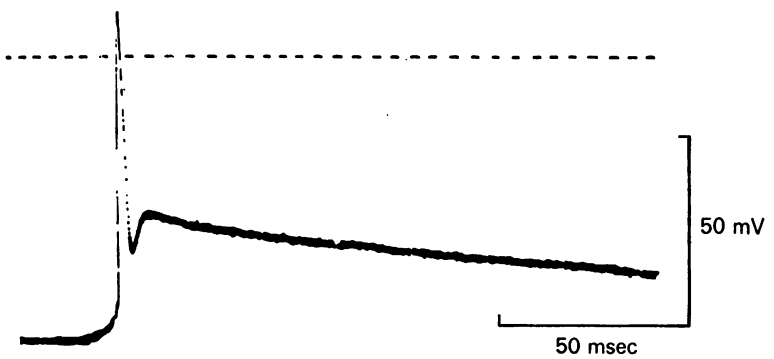

Fig. 6. A recording from a neurone with an electrode filled with 4 M-KCl 14 min after impalement. The cell was synaptically activated by stimulating the l.o.t. once. Throughout this period, E_m remained a fairly constant − 60 mV. The l.a.d. on initial impalement was hyperpolarizing. Scales: 50 mV and 50 msec.

Stimulus requirements for the l.a.d.

In the experiments so far described, the l.a.d. followed a spike generated from the preceding e.p.s.p. However, the e.p.s.p. and/or spike generation was not an absolute requirement for generating a l.a.d. The following experiments illustrate this point.

(i) No l.a.d. followed a spike generated directly by the injection of current into a neurone of the olfactory cortex (see Fig. 5 of Scholfield, 1978*a*). (ii) In a few cells, local stimulation with a tungsten micro-electrode (Scholfield, 1978*a*) produced a l.a.d. where the initial e.p.s.p. was very small and an antidromic spike was absent. Under these circumstances, the l.a.d. could be readily detected by depolarizing the cell with positive current (Fig. 5*D*). $E_{\text{l.a.d.}}$ of the locally generated response was nearer to the membrane potential than for the l.a.d. generated by stimulating the l.o.t. (cf. Fig. 5*A* and 5*D*).

Conversely, the amount of orthodromic activity was reduced by reducing the stimulus voltage or increasing Mg or decreasing Ca concentration, a small e.p.s.p. *without* a subsequent l.a.d. was generated. The l.a.d. was also reduced during repeti-

tive stimulation (as noted above, Fig. 4), or after the second of two paired pulses delivered at 0·2–0·5 sec intervals (Fig. 7). Under these conditions, the second e.p.s.p. was enhanced and prolonged, but the membrane conductance increase characteristic of the l.a.d. was less.

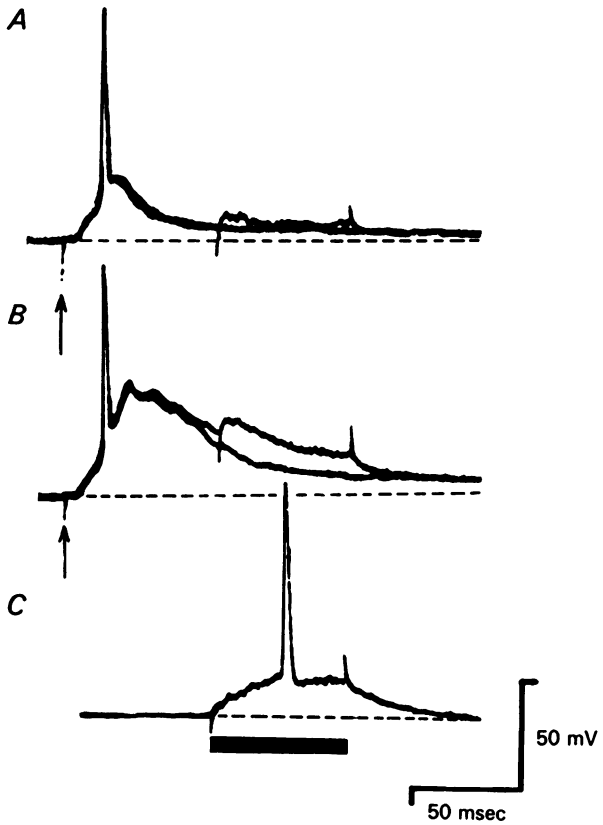

Fig. 7. Synaptic potentials produced by delivering paired stimuli 380 msec apart. In *C*, +0·5 nA of current was injected into the cell (during the filled bar) which gave rise to a depolarizing potential and a single action potential. The cell was synaptically activated (↑) in *A* without any current injection (continuous trace). 380 msec later the l.o.t. was again stimulated (*B*). Then a similar pair of stimuli were delivered to the l.o.t. and +0·5 nA injected into the cell during the falling phase of the l.a.d. The transient at the beginning and end of the current pulses are current switching artifacts. Voltage calibration: 50 mV. Time calibration: 50 msec.

Action of temperature

A temperature of 25 °C was selected for this study because it permits the use of thicker slices (in which deep-lying neural elements remain viable) over prolonged incubation periods without deterioration in their synaptic activity or ionic batteries, presumably because oxygen requirements are less critical (see Harvey *et al.* 1974). When the temperature of the bathing solution was set at values 17–38 °C for up to 30 min periods, $E_{\text{l.a.d.}}$ did not alter (Fig. 8*A*). As might be expected, the time course

of both the e.p.s.p. and the l.a.d. decreased with increasing temperature (Fig. 8*B*). The excitability (threshold voltage for the action potential) did not change (Scholfield, 1978*a*).

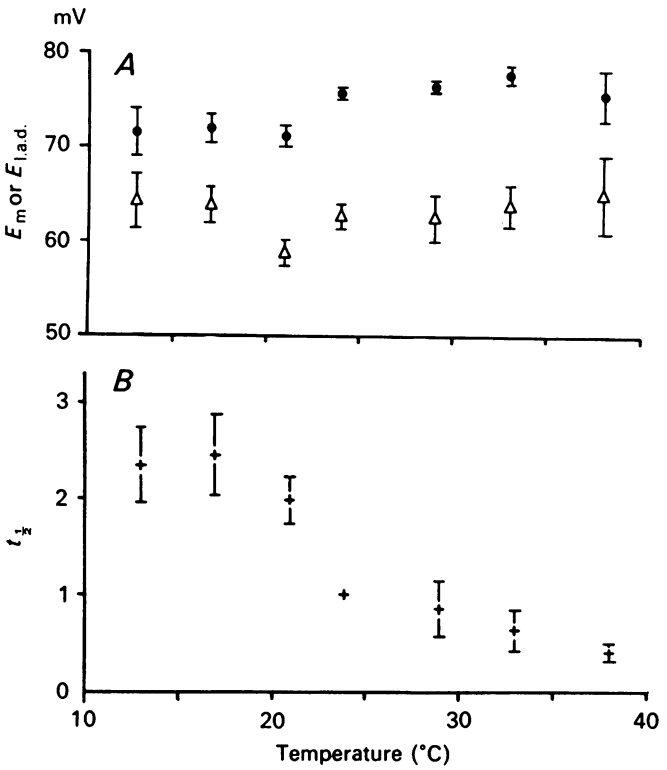

Fig. 8. Effect of temperature of the l.a.d. *A*, (i) resting E_m (filled circles). (ii) $E_{l.a.d.}$ for the l.a.d. (open triangles). Ordinate: membrane potential, mV. *B*, time course of the l.a.d. Ordinate: half-time, $t_{\frac{1}{2}}$, for the decline in the voltage amplitude of the l.a.d. after the stimulus artefact, normalized to that at 24 °C (= 1). Abscissae: temperature of the bathing solution, °C. Points are means of measurements from three to ten cells and bars are the S.E. of means.

DISCUSSION

The l.a.d. succeeding the e.p.s.p. appears to represent an i.p.s.p. for the following reasons: (*a*) It is not a direct consequence of spike generation, since no l.a.d. follows a spike activated directly by injecting depolarizing current. This distinguishes the l.a.d. from spike after-potentials. (*b*) The l.a.d. can be distinguished from the proceeding e.p.s.p. by its differential sensitivity to repetitive stimulation and Mg^{2+} ions and by its more negative reversal potential. The latter distinction is made clearer when the cell is depolarized; the e.p.s.p. and l.a.d. are then in the opposite directions. (*c*) The l.a.d. is associated with a large conductance increase and a strong inhibition of both direct and synaptically evoked spike generation.

Like the conventional i.p.s.p., the l.a.d. appears to be generated (at least in substantial part) by an increased Cl^- conductance. However, it is clearly unconventional in the direction of the potential change. The principal reason for this is undoubtedly the high resting potential recorded in these cells (−74 mV on average).

The reversal potential for the l.a.d. (−63 mV), though rather more positive than usually obtained for an i.p.s.p., accords quite well with that reported for i.p.s.p.s in olfactory cortex *in vivo* (Biedenbach & Stevens, 1969*b*). Nor is a reversal potential positive to the membrane potential *ipso facto* incompatible with a Cl^--mediated event, as indicated by several studies on peripheral neurones (Nishi, Minota & Karczmar, 1974; Obata, 1974; Adams & Brown, 1975). As pointed out by Ginsborg (1967), effective reduction of these e.p.s.p.s may be produced by an i.p.s.p. which, of itself, is depolarizing, if (i) it is accompanied by a large enough conductance increase, and (ii) if $E_{\text{i.p.s.p.}}$ is sufficiently negative to $E_{\text{e.p.s.p.}}$: these conditions are well satisfied in the situation described in this paper.

The amount of depolarization associated with the i.p.s.p. is itself rather uncertain because of the residue of the e.p.s.p. Thus under conditions when the e.p.s.p. is very small (by focal stimulation), the i.p.s.p. remains depolarizing but is clearly smaller in amplitude than during orthodromic stimulation (Fig. 5).

It is suggested that the l.a.d. arises through activation of deep-lying inhibitory interneurones by exciting superficial neurones. Interpolation of an additional synapse is compatible with the strong frequency dependence of the l.a.d. and with its sensitivity to Mg^{2+}. Failure to evoke a l.a.d. after stimulating a single superficial neurone with injected current, or with very low strength l.o.t. stimulation (subthreshold for spike generation) suggests that the interneurones require a simultaneous excitatory input from several superficial neurones. (This may also explain the difficulty experienced in obtaining a 'pure' l.a.d. with focal antidromic stimulation: the output from the pyramidal cells is too diffused to permit a sufficient number of axons to be stimulated.) Appropriately innervated polymorphic interneurones giving off highly branched processes over a 0·2 mm radius have been detected histologically (Stevens, 1969; Halliwell, 1976). In accordance with the scheme of Biedenbach & Stevens (1969*b*), the inhibitory input from these inter-neurones most likely terminates either on the superficial cell soma or on the dendrites at a point much nearer to the soma than the primary excitatory input. This is suggested by (*a*) the very large conductance change associated with the l.a.d. and (*b*) the absence of a clear representation of the l.a.d. in surface recordings. Biedenbach & Stevens (1969*b*) described a surface-positive component of the evoked potential which correlated in time course with the i.p.s.p., but in their experiment the i.p.s.p. was *hyperpolarizing*. Although the surface-positive component of the evoked potential can be readily detected in isolated guinea-pig cortex (Harvey *et al.* 1974), its time-course is much briefer than the l.a.d. Recently Pickles & Simmonds (1978) have described a small long duration surface *negative* wave in the isolated rat cortex which appears a more appropriate representation of the l.a.d.

The possibility exists that the depolarizing i.p.s.p. may occur naturally in other brain areas. If so, they might be more difficult to detect than conventional hyperpolarizing i.p.s.p. or might be confused with the tail of the e.p.s.p. unless revealed by concurrent conductance/reversal potential measurements.

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REFERENCES

ADAMS, P. R. & BROWN, D. A. (1975). Actions of γ-aminobutyric acid on sympathetic ganglion cells. *J. Physiol.* **250**, 85–120.

ARAKI, T., ITO, M. & OSCARSSON, O. (1961). Anion permeability of the synaptic and non-synaptic motoneurone membrane. *J. Physiol.* **159**, 410–435.

BIEDENBACH, M. A. & STEVENS, C. F. (1969*a*). Electrical activity in cat olfactory cortex produced by synchonous orthodromic volleys. *J. Neurophysiol.* **32**, 193–201.

BIEDENBACH, M. A. & STEVENS, C. F. (1969*b*). Synaptic organisation of cat olfactory cortex as revealed by intracellular recording. *J. Neurophysiol.* **32**, 204–214.

ECCLES, J. C. (1957). *The Physiology of Nerve Cells*, p. 115. London: Oxford University Press.

ECCLES, J. C., ECCLES, R. M. & ITO, M. (1964). Effects produced on inhibitory post-synaptic potentials by the coupled injections of cations and anions into motoneurones. *Proc. R. Soc.* B. **160**, 197–210.

GINSBORG, B. L. (1967). Junctional transmission. *Pharmac. Rev.* **19**, 289–316.

HABERLY, L. B. & SHEPHERD, G. M. (1973). Current-density analysis of summed evoked potentials in opossum prepyriform cortex. *J. Neurophysiol.* **36**, 789–802.

HALLIWELL, J. V. (1976). Synaptic interaction underlying piriform evoked responses studied in vitro. Supplementum 1. *Expl Brain Res.* 223–228.

HARVEY, J. A., SCHOLFIELD, C. N. & BROWN, D. A. (1974). Evoked surface-positive potentials in isolated mammalian olfactory cortex. *Brain Res.* **76**, 235–245.

KELLY, J. S., KRNJEVIC, K., MORRIS, M. E. & YIM, G. K. W. (1969). Anion permeability of cortical neurones. *Expl Brain Res.* **7**, 11–31.

MATSUURA, S. & ENDO, K. (1971). Anion permeability of the inhibitory subsynaptic membrane of the spinal motoneurone of the toad. *Jap. J. Physiol.* **21**, 265–276.

NISHI, S., MINOTA, S. & KARCZMAR, A. G. (1974). Primary afferent neurones: The ionic mechanism of GABA-mediated depolarization. *Neuropharmacology* **13**, 215–219.

OBATA, K. (1974). Transmitter sensitivities of some nerves and muscle cells in culture. *Brain Res.* **73**, 71–88.

PICKLES, H. G. & SIMMONDS, M. A. (1978). Field potentials, inhibition and the effect of pentobarbitone in the rat olfactory cortex slice. *J. Physiol.* **275**, 135–148.

RICHARDS, C. D. & SERCOMBE, R. (1968). Electrical activity observed in guinea-pig olfactory cortex *in vitro*. *J. Physiol.* **197**, 667–683.

SCHOLFIELD, C. N. (1976). A depolarising inhibitory post-synaptic potential in mammalian brain slices. *J. Physiol.* **263**, 120–121*P*.

SCHOLFIELD, C. N. (1978*a*). Electrical properties of neurones in the olfactory cortex slice *in vitro*. *J. Physiol.* **275**, 535–546.

SCHOLFIELD, C. N. (1978*b*). A barbiturate induced intensification of the inhibitory potential in slices of guinea-pig olfactory cortex. *J. Physiol.* **275**, 559–566.

STEVENS, C. F. (1969). Structure of cat frontal olfactory cortex. *J. Neurophysiol.* **32**, 184–192.

YAMAMOTO, C. & MCILWAIN, H. (1966). Electrical activities in thin sections from the mammalian brain maintained in chemically-defined media *in vitro*. *J. Neurochem.* **13**, 1333–1343.