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L-Glutamate excitations on cortical neurones: influence of endogenous neuronal activity and halothane

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L-Glutamate applied microiontophoretically onto single cortical neurones invariably results in an increase in firing rate. This is usually attributed entirely to a postsynaptic effect, the possibility of a presynaptic contribution to the response usually being overlooked. These experiments were designed to explore this possibility more fully.

In the present study L-glutamate was applied microiontophoretically (Clarke, Forrester & Straughan, 1974) onto quiescent neurones in the cerebral cortex of urethane anaesthetized rats (1.8 g/kg). The electro-corticogram (E.Co.G.) of a urethane anaesthetized rat appears as intermittent surface positive bursts with high frequency activity on the plateaux separated by quiescent periods (Bindman, Lippold & Redfearn, 1964). On all of the 100+ neurones tested the neuronal firing induced by L-glutamate occurred initially only during the positive plateaux in the E.Co.G. (recorded at the cortical surface directly above the microelectrode) and as each response to L-glutamate developed the number of action potentials within each positive plateau increased. This firing pattern is similar to that shown by spontaneously active cells deep in the cortex. This suggests therefore that these L-glutamate induced excitations depend upon the presynaptic drive (depicted by the number of E.Co.G. positive plateaux) being facilitated by the postsynaptic depolarizing action of L-glutamate.

All depressants tested (including barbiturates, halothane and benzodiazepines), when superimposed upon this existing level of anaesthesia decrease the frequency of occurrence of these positive plateaux (Forrester & Gartside, 1975) and consequently they decrease the spontaneous activity of cortical neurones. On the 8 occasions when the animals were forced to breathe 1.5% halothane for between 5 and 10 min L-glutamate responses such as those described were consistently reduced as the frequency of occurrence of the positive plateaux fell, although the number of action potentials per E.Co.G. plateau at a given stage in the L-glutamate response remained unaffected. Blood pressure also remained unaffected.

When larger expelling currents of L-glutamate were applied (particularly when the depth of anaesthesia was increased and the periods between the positive plateaux in the E.Co.G. extended) then action potentials (with relatively constant interspike intervals) were generated between the positive plateaux. This firing pattern is typical of that induced by direct depolarizing currents injected into single cells (Calvin & Schwindt, 1972). There is a period of inhibition immediately following the plateaux during which there are no action potentials generated. Halothane does not inhibit the appearance of action potentials occurring between the plateaux, and often potentiates the overall response possibly because the number of inhibitory periods is reduced as the plateau frequency falls. Potentiation of glutamate-induced responses by halothane and other anaesthetics has been reported before (Catchglove, Krnjević and Maretić, 1972).

Hence it is concluded that endogenous excitation can contribute to the increase in firing rate evoked by L-glutamate and that by decreasing the endogenous excitatory drive the response to L-glutamate is altered in a manner determined by the power of the exogenous and initial endogenous input to the neurone.

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