contains a cellular component. This possibility has been further examined in the present work which includes studies of the effects of drugs on the transport of glucose into the raffinose space.

Cerebral cortex slices were prepared from guinea-pigs and incubated for 30 min at 37° C in an oxygenated, Krebs-Ringer phosphate medium containing sodium pyruvate (4 mM) as nutrient. The temperature of the medium was then reduced to 1° C and glucose (10 mM, final concentration) or glucose and inulin (5 mg/ml) or glucose and raffinose (10 mM) was added to the medium and the incubation continued. When drugs were used they were present throughout the incubation procedure.

The volume of distribution of inulin (effective hydrodynamic radius 1·5 nm) was only 75–80% of that of raffinose (0·6 nm) at equilibrium and the difference between the volumes of distribution of the two solutes is likely to represent an intracellular compartment which is accessible to raffinose under these conditions. Phenobarbitone (2 mM) but not barbitone (2 mM) or thiopentone (0·8 mM) increased the uptake of glucose by this intracellular compartment of the slices. Diphenylhydantoin (0·5 mM) also increased the glucose uptake but ethosuximide (0·5 mM) and acetazolamide (0·2 mM) were without detectable effect.

These findings, considered with those of previous studies (Gilbert, Ortiz & Millichap, 1966; Gray & Gilbert, 1970) suggest that monosaccharide uptake by cerebral cortex slices involves movement of the monosaccharide into at least two intracellular compartments, one best studied at 37° C (A) and the other at 1° C (B). The anticonvulsants tested can influence the permeability of either compartment A or B or both. The permeability of compartment A to monosaccharides is sensitive to phenobarbitone, ethosuximide, dimethadione and acetazolamide at 37° C. The permeability of compartment B to monosaccharides is sensitive to phenobarbitone and diphenylhydantoin at 1° C. It is speculated, on the basis of this and other work (Tower, 1968) that compartment A contains neurones and compartment B contains glial cells.

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Some characteristics of cortical inhibition induced by local stimulation and by an acute seizure focus in the cat

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In the present study the inhibition of cortical neuronal firing produced by epicortical stimulation (Krnjević, Randić & Straughan, 1966) has been compared

with that produced in the same neurone by an adjacent penicillin seizure focus (Prince & Wilder, 1967), or with that produced by a seizure focus in the contralateral cortex (Crowell, 1970). The inhibitions have been characterized by their time course and by their response to several putative neurotransmitters and their antagonists, using conventional microiontophoretic techniques.

All experiments were performed in cats anaesthetized with halothane-nitrous oxide, paralysed with gallamine, and artificially respired. The seizure focus was created by microinjection of a solution of sodium benzylpenicillin (300 mg/ml in 0·9% saline) into the supra sylvian cortex. Neurones were examined when firing spontaneously and also when driven by iontophoretic applications of acetylcholine or excitant amino acids. The inhibitions were assessed both from continuous and superimposed film records, and also from computer-generated post-stimulus histograms.

Qualitatively the inhibitions were similar whether induced by epicortical stimulation or by an ipsilateral or contralateral seizure focus. The duration of inhibition varied between 75 and 600 ms and in any particular neurone did not appear to be related to the type of inhibitory stimulus. In stable neurones, reproducible inhibitions could be obtained for several hours and the duration of focally evoked inhibition was inversely and linearly related to the magnitude of the ejecting currents of L-glutamate, DL-homocysteate, or acetylcholine. Thus, the seizure focus evoked inhibition does not appear to be due to a type of depolarization block and, as it can be detected against a background of iontophoretically driven firing, it must be entirely post-synaptic.

The proposed GABA antagonist, bicuculline (Curtis & Felix, 1971) given intravenously (0·2–0·6 mg/kg) readily antagonized the inhibition provoked by epicortical stimulation but had no clear effect on the inhibition induced by a seizure focus. Consistent antagonism of the inhibition evoked by any of the stimuli was not achieved with iontophoretic applications of bicuculline or picrotoxin. None of the inhibitions appeared to involve a cholinergic mechanism since depressant effects of acetylcholine were not seen in these neurones and iontophoretic applications of atropine or (+)-tubocurarine had no effect on the duration or character of the inhibition.

Despite the overall similarities, the difference in the sensitivity of epicortical stimulation evoked inhibition and seizure evoked inhibition, in their response to systemically administered bicuculline, implies either subtle differences in the inhibitory pathways activated or is a reflection of the mechanism by which bicuculline reduces inhibition.

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