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TRANSMISSION OF BURST RESPONSES THROUGH SLICES OF RAT CEREBRAL CORTEX

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SUMMARY

1. Slices of rat's forebrain, 400 μm thick, have been cut and maintained in a bath perfused with warm oxygenated Krebs solution. Records were made with extracellular micropipettes of the neural responses to local stimulation of the cortex itself or the underlying white matter.

2. Single stimuli at either of these sites could produce an all-or-nothing burst response among nearby neurones. This response usually lasted 0·2–0·5 s during which repetitively discharging cortical units could be recorded at all cortical depths.

3. This burst response was transmitted from the stimulated point across the cortex in all directions with a velocity of roughly 0·1 m s^{-1}.

4. Complete recovery of excitability among neurones generating the burst response took about 10 s.

5. Removal of Ca^{2+} from the perfusate prevented transmission of this response, as did a high concentration of Mg^{2+} or 160 mg/100 ml of ethanol.

6. Propagation of the burst response was not dependent upon the integrity of the underlying white matter; it required only that any 20% of the cortical thickness was intact and undamaged.

7. In coronal sections of brain the response could be transmitted from one hemisphere to the other provided that the corpus callosum was intact.

INTRODUCTION

Slices of mammalian brain were first maintained *in vitro* by Hillman & McIlwain (1961) who showed that the isolated neurones possessed a reasonable resting membrane potential. However, these slices were cut parallel to the pial surface, which must have severed some part of almost every neurone, and did not provide any convenient afferent pathway through which the isolated neurones might be excited. Seven years later, Richards & Sercombe (1968) published a description of slices from guinea-pig olfactory cortex that were maintained *in vitro*. They were able to record the responses of neurones in the prepiriform area to electrical excitation of the lateral olfactory tract, and since 1968 a great deal of use has been made of this preparation. Another source of brain tissue providing slices that contain a convenient afferent pathway is the hippocampus (Richards, 1975). The main advantage of both these

preparations is that they provide facilities for the study of monosynaptic neural excitation by synchronous volleys in known brain pathways.

Since then, slices which include areas of the cerebral cortex have primarily been used for investigations of pharmacological and intracellular events (see for example: Dingledine, Dodd & Kelly, 1980; Connors, Gutnick & Prince, 1982; Thomson, 1986; McCormick & Prince, 1986; Bindman, Meyer & Prince, 1988). There has however been sparse systematic investigation of the properties of the preparation as a network of neurones. Many workers have observed that a single stimulus can cause neurones in isolated slices of cerebral cortex to fire repetitively. Vogt & Gorman (1982) reported that a single stimulus given to the fibres of the corpus callosum could produce '... an all-or-nothing burst of action potentials' from the isolated cortical neurones. However, none of the many reports of repetitive responses to single stimuli (Prince, 1968; Prince & Wong, 1981; Thomson, 1986) includes a statement that the burst response is transmitted transcortically. Only Bagust, Herron & Kerkut (1984), who worked with slices of hamster brain, showed that stimulation of the corpus callosum caused activity to spread throughout the cortex, over both hemispheres.

The results of our experiments with slices of rat's brain described below, confirm and extend the conclusions of Bagust *et al.* (1984). They show that a single local stimulus to the cortex itself or to the underlying white matter can cause an all-or-nothing burst response in nearby cortical neurones, and that this response is transmitted from the stimulated point, through intracortical synapses, at approximately 0·1 m s^{-1} in all directions.

METHODS

Rats of either sex were killed and decapitated, and the scalp incised to reveal the skull. The head was then submerged in ice-cold water for 3 or 4 min to cool the brain. The bone was then scored and reflected to expose the brain. The dura mater was removed, the whole brain excised and a section of forebrain, approximately 5 mm thick, was isolated. Coronal or parasagittal slices, 400 μm thick, were then cut in ice-cold Krebs solution, using a Vibroslice (Campden Instruments Ltd). Figures 13–26 and 49 of the stereotaxic maps of Paxinos & Watson (1982) indicate the structures contained in the coronal slices. Slices were kept on a nylon tea-strainer in a storage vessel, submerged in Krebs bicarbonate buffer, continuously gassed with 5% CO_2 in O_2 at room temperature, until needed for use. The composition of the Krebs solution was (mM): NaCl, 124·0; KCl, 5·0; $NaHCO_3$, 26·0; NaH_2PO_4, 1·25; $MgSO_4.7H_2O$, 2·0; D-glucose, 10·0; $CaCl_2$, 2·0. Individual slices were then transferred to a recording chamber and superfused at 34 °C using a Gilson Minipuls pump, at a flow rate of 1 ml min^{-1}. Both interface and submerged slice techniques were used: slices were maintained at the interface of the medium and a moist O_2–CO_2 atmosphere in experiments during which electrode positions were altered frequently; submerged slice techniques were used when the composition of the superfusing medium was to be changed.

Stimulating electrodes consisted of twisted pairs of 300 μm stainless-steel wires insulated with Teflon (Clark Electromedical, SST 300) and stimuli were square-wave pulses of 0·1 ms duration, generated by a Devices DS2 stimulus isolation unit, triggered by a Grass SS4 stimulator. Recording electrodes were 2·0 mm o.d. capillaries (Clark Electromedical), pulled to an inside tip diameter of 1–3 μm, and filled with Krebs solution. Grass P511 AC-coupled amplifiers with a rise time constant of 0·1 ms and a fall time constant of 600 ms were used to amplify the signal by 1000, which was then displayed on a Nicolet digital oscilloscope and recorded on videotape using a pulse code modulation device (Sony PCM 701). These methods have been demonstrated to the Physiological Society (Boakes, Burns & Webb, 1987).

RESULTS

The shape of the burst response

A single stimulus given to either the cerebral cortex itself, to the underlying white matter, or to the internal capsule produces a response that can be recorded from a

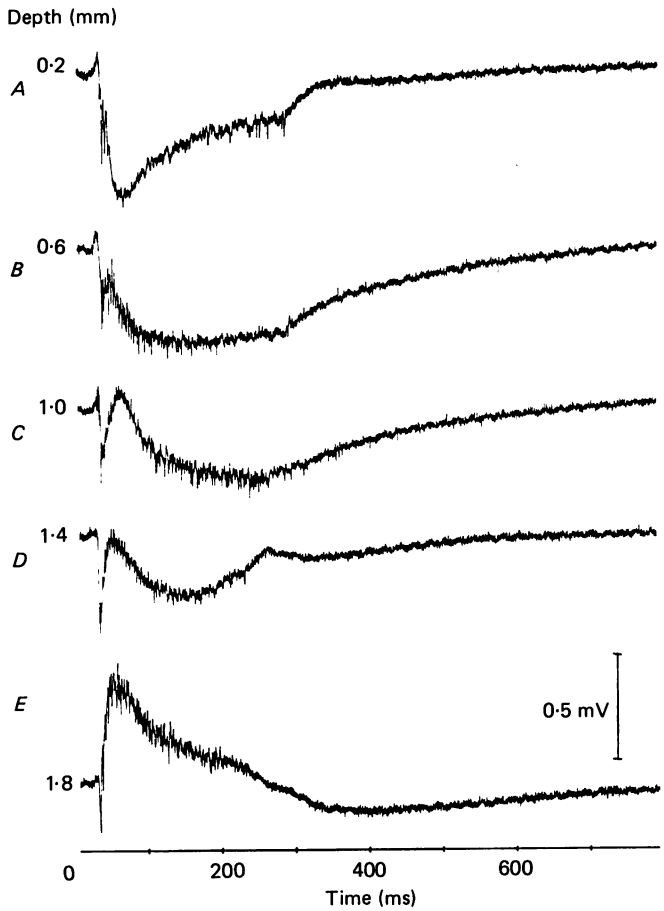

Fig. 1. The shapes of burst response recorded at different depths below the pial surface of the cerebral cortex. Stimuli were given to the white matter immediately below the cortex; the recording microelectrode was about 1·5 mm away. Stimulation frequency = 1 in 10 s; strength = 0·1 ms, 1·5 × threshold. In these and all similar records, negativity of the electrode's tip is indicated by a downward deflection; the abscissa shows time after stimulation.

microelectrode in the cortex. In the upper or pial half of the cortex, this response consists of the sudden development of local negativity, which usually persists for 0·2–0·5 s and often ceases as suddenly as it begins, so that the whole waveform appears to be nearly rectangular (Fig. 1*A*). The recorded shape of the burst response depends upon the distance below the pial surface from which a record is made.

Between 0·2 and 0·8 mm, relatively simple waveforms similar to that shown in Fig. 1*A* are recorded; between 0·8 and 1·4 mm cortical depth the simple 'rectangular' wave that can be recorded more superficially is preceded by a much shorter duration evoked potential (Fig. 1*C* and *D*) which is maximal at a depth of about 1·4 mm.

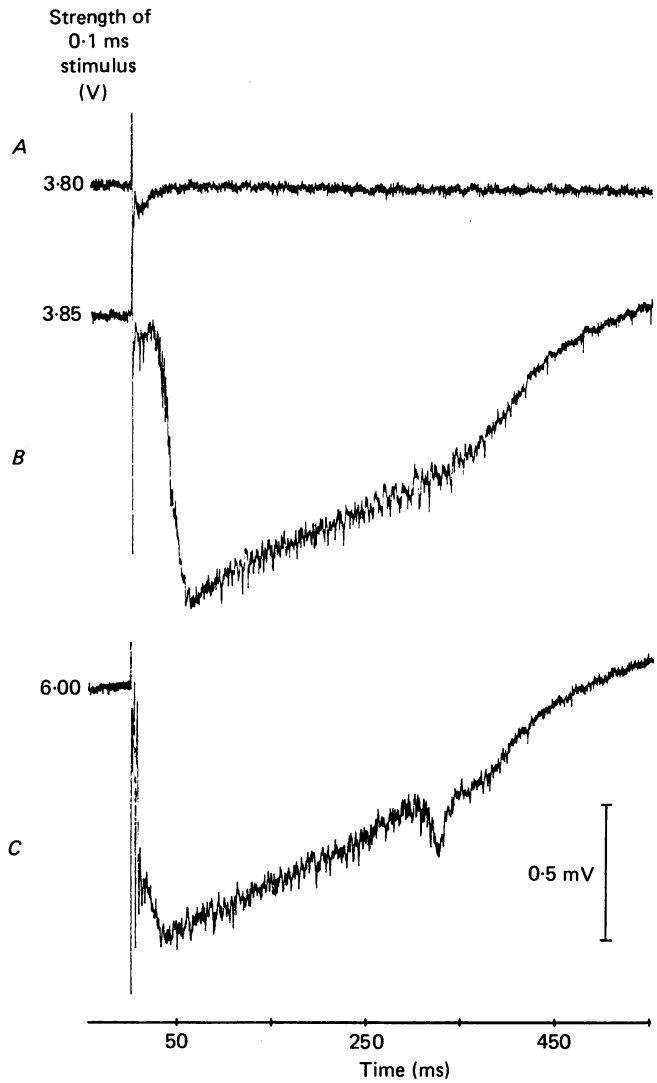

Fig. 2. The all-or-nothing relationship between the burst response and the strength of stimulus used to excite it. Both stimulating and recording electrodes were about 0·8 mm below the pial surface of the cerebral cortex, and some 0·5 mm apart. Stimulation frequency = 1 in 10 s; duration of stimulus = 0·1 ms.

Provided that the electrode's tip lies close enough to the body of a neurone, a repetitive discharge can be seen to accompany the gross response. The discharge of several individual neurones can just be seen in some of the records in Fig. 1. We have recorded a repetitive discharge from single neurones, at all depths below the pial

surface from 0·3 to 1·7 mm, which presumably implies that some of the neurones in each of the layers II–VI are involved.

All-or-nothing nature of the burst response

Provided that more than about 0·25 mm, measured along the cortex, separates the stimulated and recording points, the burst response bears an all-or-nothing relationship to the strength of stimulus. This is illustrated in Fig. 2, which shows a very small local response to stimulation of the underlying white matter with 3·80 V for 0·1 ms (Fig. 2*A*); when the stimulus strength was raised to 3·85 V a burst response was produced lasting about 0·4 s (Fig. 2*B*), which was the same in magnitude for all strengths of stimulus tested up to 6·00 V (Fig. 2*C*).

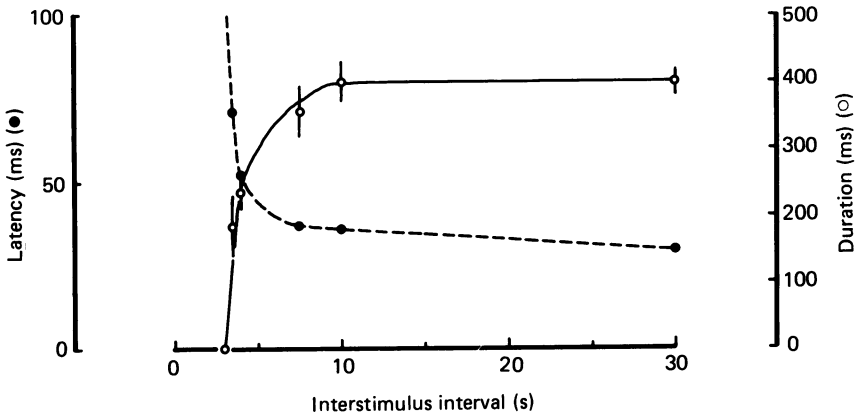

Fig. 3. The effects of various frequencies of stimulation upon the latency and duration of the burst response. Stimulation of the subcortical white matter with stimuli of 0·1 ms, 1·2 × threshold. The separation of stimulating and recording electrodes was approximately 2 mm. When the stimulation rate exceeded 1 in 3 s, the preparation failed to respond to every stimulus.

The burst response is easily fatigued

The burst response is easily fatigued and becomes shorter in duration and longer in latency if the stimuli used to initiate it are repeated at 3 s intervals. Responses cannot be made to follow every stimulus if the frequency of stimulation exceeds 0·3 Hz. On the other hand, stimulation which is not repeated more often than once every 10 s (0·1 Hz) produces responses of maximal duration and stable latency that can be elicited indefinitely (Fig. 3).

Transcortical transmission of the burst response

The greater the distance between the stimulated point and the recording microelectrode, the longer is the latency of the response. Figure 4 is constructed from the results of an experiment in which burst responses were produced by 1·1 × threshold stimuli of 0·1 ms given to fibres of the underlying white matter. In the first series of tests the stimulus was at position 0. When the recording electrode was

placed half-way between the surface and the bottom of the cortex, radially 'above' the stimulating electrodes, it was said to lie at position 0. The open circles of Fig. 4 show that as the recording electrode was moved to new positions, at the same 'cortical depth', latency increased rectilinearly with increase of distance from the stimulated point. In a second series of tests on the same slice, the stimulating

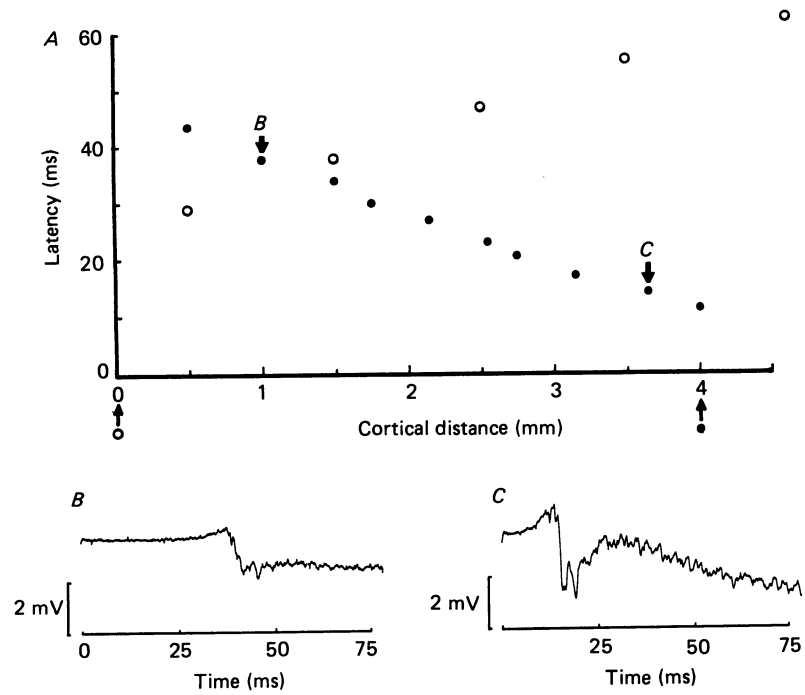

Fig. 4. The burst response is conducted at a constant velocity in both directions along a slice of cortex. Stimulation of the subcortical white matter once every 10 s; stimulus strength = 0·02 ms, 1·33 × threshold. *A*, the first series of tests was made with the stimulating electrode at position 0 (↑). The latencies of burst responses recorded at various cortical distances are shown as open circles. A second series of tests was made after shifting the stimulating electrode to a position 4·0 mm away (↑). The latencies of burst responses produced by stimulation at this point are shown as filled circles. *B* and *C* show sample records of responses to stimulation at position 4·0 mm. The abscissa measures time after stimulation. The recording electrode was always inserted at about $0{\cdot}4 \times T$ below the pial surface, where T = cortical thickness.

electrode was moved to a new point in the white matter, 4·0 mm distant from that originally stimulated. The filled circles show that stimuli at this second site produced burst responses which spread in the opposite direction, across the cortex at approximately the same velocity as before. We have performed many tests of this sort and the transcortical conduction velocity always lay between 0·03 and 0·125 m s^{-1} at 34·0 °C. Experiments like that illustrated in Fig. 4 showed that a just-suprathreshold stimulus of either white matter or cerebral cortex produced excitation of a patch of cortex about 0·25 mm long, from the edges of which a burst response spreads at a constant velocity of about 0·1 m s^{-1} in both directions to invade the

whole of the available cortex. This statement was true of brain slices that were cut in either coronal or in sagittal planes.

Synaptic transmission of burst response

The extremely slow velocity of transmission, without attenuation, in any plane in which the slice of cortex is cut, makes it seem unlikely that this response could be

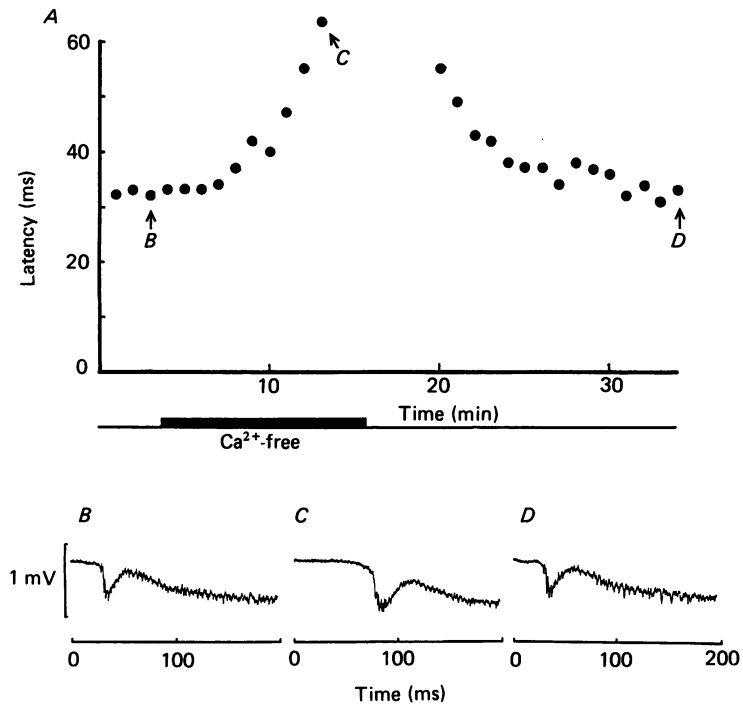

Fig. 5. The effects upon the burst response of temporarily exposing the brain slice to Ca^{2+}-free Krebs solution. Stimulation of the subcortical white matter once every 10 s; stimuli = 0·1 ms, 1·5 × threshold. Stimulating and recording electrodes were about 1·5 mm apart. Each point represents each sixth measurement. Sample records taken at points *B*, *C* and *D* indicated on the graph are shown below, with abscissa measuring time after stimulation.

transmitted only by nerve fibres. It would appear to be transmitted synaptically between neighbouring cortical neurones, an hypothesis which is supported by the sensitivity of the response to the concentration of Ca^{2+} in the bathing fluid. Figure 5 shows the effects of temporarily changing the perfusate from Krebs solution containing 2·0 mM-Ca^{2+}, to Krebs solution without any Ca^{2+}. It will be seen that the reduction in concentration of Ca^{2+} produced a progressive increase in latency, with ultimate complete failure of transmission. These effects were reversible. Almost identical records could be produced by either a temporary increase of $[Mg^{2+}]$ in the superfusion medium from 2 to 6 mM, or by the temporary addition of 35 mM-ethanol.

Where are the synapses necessary for transmission of the burst response?

If the synapses that are essential to transmission of the burst response lie in only one particular cortical layer, we should then expect that a surgical incision through that layer would stop transmission of the response across the cortex. With such thoughts in mind we have performed experiments of the type illustrated by Fig. 6.

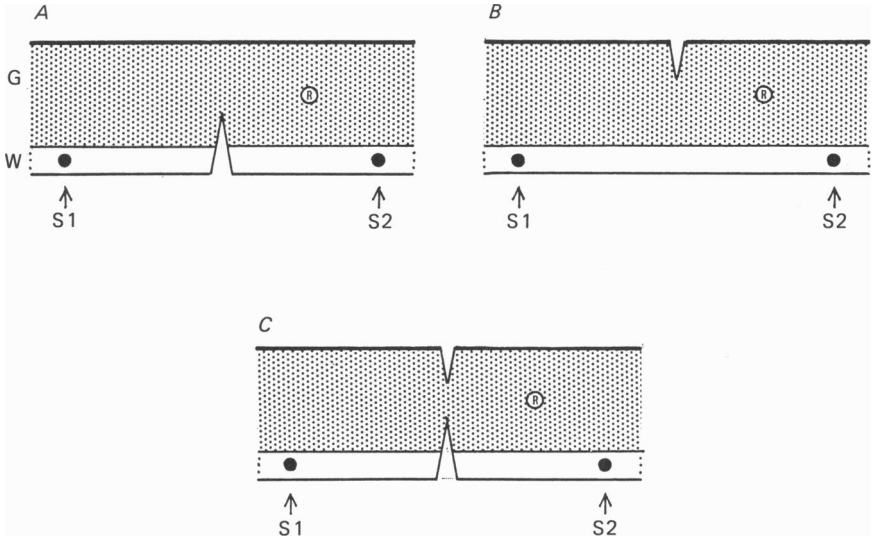

Fig. 6. Experiments to determine the cortical depth at which synapses, essential to transmission of the burst response, lie. G = grey matter; W = underlying white matter; R = recording site; S1 and S2 = sites of stimulation. For procedure, see text.

The white matter beneath the cortex was stimulated with just-suprathreshold stimuli once every 10 s at S1 and the resulting burst responses recorded at R, some 5 mm away. A radial cut of measured length was then made through the white matter and deeper part of the grey matter (Fig. 6*A*); stimulation and recording were continued for at least 10 min before the cut was extended further through the cortex. We found that each cut produced an immediate increase in latency of the burst response, which lasted a minute or two and then returned to the original value that was obtained before cutting. In fact, severence of the lower cortical layers and underlying white matter made no lasting difference to the transmission velocity of the burst response until nearly 80% of the cortical thickness had been incised. As we approached 80%, transmission velocity slowed, and cuts of approximately 80% caused transmission through the remaining, intact, superficial 20% of cortex to fail. Transfer of stimulation of S2 produced a burst response.

The same technique was used to find out how deep a radial cut had to extend from the cortical surface before transmission past the cut was stopped (Fig. 6*B*). Again, it appeared that transmission was possible provided that the superficial cut did not extend through more than some 80% of the cortical depth. By making experiments with superficial and deep cuts at the same cortical point (Fig. 6*C*), we found that the

burst response would transmit past the damaged area provided that some 20% of tissue remained undisturbed at any cortical depth.

It was therefore concluded that the synaptic connections essential to the spread of the burst response are to be found at all cortical depths.

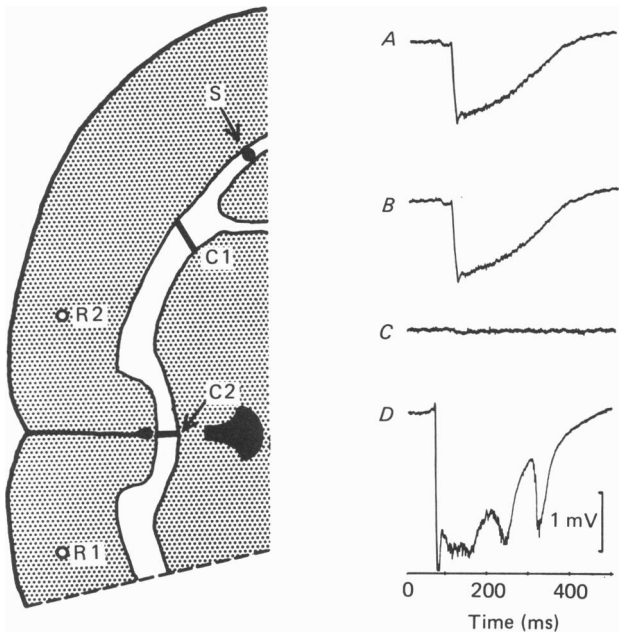

Fig. 7. Effects upon excitation of the burst response, of various cuts in the subcortical white matter. Stimulation at S once every 10 s, 0·1 ms, 1·2 × threshold. *A*, a burst response recorded at R1 when the slice was intact. *B*, a burst response recorded at R1 after cutting the white matter at C1. *C*, the burst response is abolished by a cut through the corpus callosum at C2. *D*, neither of the cuts, C1 or C2, has abolished the burst responses recordable in the ipsilateral cortex at R2.

Excitation of burst response by fibres of corpus callosum

Coronal brain slices made at about the mid-point of the corpus callosum provide bilateral sections in which some of the most medial callosal fibres travel uninjured between the hemispheres. In such slices it is possible to show that excitation of the white matter on one side of the brain can excite a 'typical' burst response in the contralateral hemisphere (Fig. 7). However, it does not seem that this excitation travels the whole of the distance between the stimulated and recording points via fibres of the corpus callosum. Figure 7*A* and *B* shows that cutting the white matter underlying the cortex, between the stimulated point and the mid-line, did not prevent the spread of excitation to the opposite hemisphere, unless the corpus callosum itself was severed (Fig. 7*C*). In the case of Fig. 7*B*, the stimulation of the white matter must have caused excitation to travel up into the overlying cortex (Fig. 7*D*); here, it was adequate to initiate a spreading burst response, which when it had travelled across the cortex towards the mid-line reached the cells of origin of intact

transcallosal fibres. Excitation of these neurones was then sufficient to transmit a burst response to the contralateral hemisphere.

DISCUSSION

We are not the first people to study the behaviour of neocortical neurones in slices of mammalian brain maintained *in vitro*. While there have been several reports that a single stimulus could produce a repetitive response from isolated cortical neurones (Prince, 1968; Prince & Wong, 1981; Bagust, Herron & Kerkut, 1984; Thomson, 1986), there has been neither a detailed description of this burst response nor an investigation of its mode of transmission. Shaw & Teyler (1982) examined neocortex in 400–700 μm slices of rat brain maintained *in vitro* but found no spreading cortical responses to local stimulation. Vogt & Gorman (1982) described the responses of cortical neurones to stimulation of the corpus callosum in slices cut from the brains of rats. They reported that a single stimulus could evoke 'an all-or-none burst of action potentials' from the neurones of layers V and VI. Bagust, Herron & Kerkut (1984) investigated slices of hamster brain 700–800 μm thick. They showed that activity spread over the cortex of both hemispheres following a single stimulus to the corpus callosum. A typical response consisted of a 'short-latency component' which was followed by a repetitive neuronal discharge lasting 1–2 s. They believed that the prolonged part of the response was maintained by synaptic conduction since it was reversibly abolished when they replaced Ca^{2+} with Mn^{2+} in the perfusion fluid. There seems little doubt from their description and from the figures they presented that this part of their response was similar to our own burst response.

The relatively slow velocity of the cortical burst response – around 0·1 m s^{-1} – would imply the average involvement of two synapses per 100 μm of cortex. This calculation assumes a delay of 0·5 ms per synapse. It is therefore clear that transmission along the cortex in a slice of rat's brain must involve a very large number of cortical synapses for every millimetre of tissue traversed. Consequently, the slice of rat's brain provides a system which should be very sensitive to drugs affecting cortical function. That this is so is indicated by the fact that the addition of 160 mg of ethanol per 100 ml of Krebs solution was sufficient to first slow and then stop the spread of the burst response. This concentration of alcohol is only twice the legal limit for driving motorists.

A similar, slow transmission velocity has been reported for epileptiform discharges in CA1 pyramidal neurones of the rat's hippocampal slice (Haas & Jefferys, 1984), and in CA2 and CA3 neurones in the guinea-pig's hippocampus (Knowles, Traub & Stowbridge, 1987). However, the similarity between cortical bursts and epileptiform discharges extends no further. Haas and Jefferys' epileptiform bursts were caused by low $[Ca^{2+}]$, and lasted for longer than 1 s. The cortical burst response that we have described was abolished by low $[Ca^{2+}]$ and lasted 0·2–0·5 s. Moreover, Haas and Jefferys found that lowering the $[K^+]$ from 6·0 to 3·0 mM abolished the bursts. Because of this observation we have dropped the $[K^+]$ from our usual 5·0 mM to 3·0 mM, but without any effect upon the neocortical burst response. Bagust *et al.* (1984) describing burst responses in hamster's cortex, used a perfusion fluid containing 2·5 mM $[K^+]$.

It is important to realize that the burst response to a single electrical stimulus that we have described is not epileptiform in nature. There was no evidence that it involved the periodic synchronous discharge of many neighbouring neurones. In their description of the properties of slices of hamster's brain, Bagust *et al.* (1984) make a clear distinction between 'seizure activity' sometimes seen in slices thicker than 800 μm and the response to single electrical stimuli of 400 μm thick slices of brain. The burst responses to single stimuli that they recorded were approximately 1 mV in amplitude; our own experience suggests that the amplitude of burst responses rarely exceeds 2 mV. The hippocampal bursts described by Haas & Jefferys (1984) were usually in the range of 5–10 mV, as were the drug-induced epileptiform discharges recorded by Knowles *et al.* (1987).

The properties of the cortical burst response recorded *in vitro* are similar to those of the surface-positive burst response of the cat's neurologically isolated cerebral cortex (Burns, 1951). Both responses appear to spread in all directions from a stimulated point to invade the whole of the available cerebral cortex; the magnitude of both bears an all-or-nothing relationship to the strength of the stimulus used to excite them. They spread across the cortex with velocities which are of the same order of magnitude, and both involve the repeated asynchronous firing of cortical neurones for about half a second. Spikes could be recorded during the burst response of cat's isolated cortex '... at almost any position in the cortex between 0·2 and 1·3 mm depth' (Burns, Grafstein & Olszewski, 1957). We have found the same to be true of rat's cortex maintained *in vitro*.

The evidence we have presented above, concerning the site of synapses responsible for transmission of the burst response in the rat's isolated cortical slice *in vitro*, appears to differ from the results obtained from the cat's isolated cortex *in vivo*. Superficial cuts through the cat's cortex, involving up to 50% of the cortical depth, were described as allowing the burst response to pass underneath; but cuts made from beneath the slab of isolated cortex to within 0·5–0·75 mm of the surface, obstructed the spread of the burst response (Burns & Grafstein, 1952). This does not agree with our present finding, that provided any 20% of the cortical thickness remains undisturbed, the burst response can be transmitted. It is, however, so much easier to make measured incisions in slices of cortex maintained *in vitro*, that we feel that the difference in results is probably due to unavoidable experimental error in the cat experiments.

It is remarkable that so similar a response can be recorded from the cortices of two different species. There is also some reason to believe that spontaneous bursts of activity recorded from human isolated cortex closely resemble the evoked activity found in the cat and the rat (Echlin, Arnett & Zoll, 1952; Henry & Scoville, 1952). These similarities suggest that such a response is a reflection of a fundamental architectural characteristic of the mammalian cerebral cortex.

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