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FREQUENCY-RELATED INHIBITORY MECHANISMS CONTROLLING RHYTHMICAL ACTIVITY IN THE SEPTAL AREA

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SUMMARY

1. The response patterns of identified neurones in the medial and lateral septal regions to varying rates of repetitive stimulation of the fimbria were investigated in rats anaesthetized with urethane.

2. Neurones in the lateral septum which characteristically respond to single pulse stimulation of the fimbria with an activation–inhibition sequence, exhibited a reduction or complete elimination of the inhibitory component both during and following tetanic volleys delivered at 7–12 Hz. Stimulation at lower frequencies did not alter the response.

3. Concurrently with this effect on the inhibitory component of the response exhibited by lateral septal cells, repetitive volleys eliminate the small amplitude burst discharges which are correlated with the onset of the inhibitory period and are considered to indicate the firing of inhibitory interneurones.

4. Tetanic stimulation of the fimbria at rates which eliminate this interneuronal response in the lateral septum, produce an irregular pattern of firing in medial septal neurones which previously exhibited a synchronized bursting discharge to single pulses.

5. Ipsilateral section of the fimbrial input to the septum resulted in the elimination of the burst discharge pattern exhibited by medial septal neurones.

6. The results suggest that a frequency gating mechanism in the lateral septum, activation of which is dependent upon the level of hippocampal output, is responsible for controlling the firing pattern of medial septal neurones.

INTRODUCTION

Sensory or mesencephalic reticular stimulation is known to evoke characteristic burst responses in neurones of the medial and diagonal

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band regions of the septum (*Ms*) which are synchronous with hippocampal slow wave ('theta') activity (Macadar, Roig, Monti & Budelli, 1970; Petsche, Gogolak & Van Zwieten, 1965; Petsche, Stumpf & Gogolak, 1962). Stimulation of the reticular formation and lateral hypothalamus at intensities greater than that necessary to evoke maximal 'theta' activity results in low voltage, high frequency hippocampal activity (Anchel & Lindsley, 1972; Gogolak, Petsche, Sterč & Stumpf, 1967; Gogolak, Stumpf, Petsche & Sterč, 1968; Stumpf, 1965). Concurrent with this pattern of hippocampal desynchronization, medial septal neurones, which were previously firing in burst discharges, begin to exhibit an irregular response pattern (Gogolak *et al.* 1967; Macadar *et al.* 1970). The results of these studies have suggested that, because of the apparent correlation between the pattern of neuronal discharge in the septum and the hippocampal activity, the role of the septal area may involve the transformation of reticular and sensory input into either discontinuous rhythmic bursts or a continuous irregular pattern of discharge which in turn determines the type of activity present in the hippocampus. The mechanisms by which the septal area produces these different response patterns remain unclear.

In a recent study McLennan & Miller (1974*a*) postulated, on the basis of electrophysiological data, a recurrent collateral system within the lateral septal region which may be responsible for the production of the burst discharge of neurones in the medial septum. Neurones in the lateral septal (*Ls*) region exhibited an activation–inhibition sequence following single pulse stimulation of the fimbria, the inhibitory component being mediated by interneurones. Since stimulation of the medial septal region only evokes an antidromic activation of lateral septal neurones it would appear that these latter cells relay efferent hippocampal influences through the recurrent collateral system and on to target cells in the medial septal region. In view of these data it might be expected that the pattern of firing of medial septal neurones is dependent upon the level of hippocampal efferent activity mediated via the fimbria and the effect which this activity exerts on interneurones in the recurrent collateral system of the lateral septum.

The present investigation was therefore undertaken to study the response patterns of medial and lateral septal neurones to varying levels of hippocampal output by repetitive stimulation of the fimbrial pathway, and to determine whether elimination of this hippocampal efferent system would alter the firing patterns of septal neurones.

METHODS

Experiments were performed on nineteen male Wistar rats acutely prepared under light urethane anaesthesia (1·5 g/kg I.P.) and maintained at a body temperature of 36–38° C. Animals were positioned in a stereotaxic frame and a skull flap removed, extending from lambda to 2·0 mm anterior to bregma and laterally 4·0 mm on either side of the mid line. Following ligation of the sagittal sinus at each end and removal of the dura mater, the underlying cortex and corpus callosum were aspirated bilaterally to expose the dorsal hippocampi and the septal area. The exposed areas were covered with warm mineral oil or saline throughout the experiment.

Concentric bipolar electrodes with a tip separation of 0·3–0·5 mm and a DC resistance in saline of 30–50 kΩ were used for stimulation. The electrodes were positioned visually, with the aid of a dissecting microscope, into the ipsilateral fimbria. Stimuli consisted of square wave pulses of 0·1 msec duration and 0·1–0·4 mA intensity delivered at rates of 1–20 Hz. The duration of the tetanic train was varied between 1 and 15 sec. In nine preparations stimulating electrodes were stereotaxically positioned in the region of the medial hypothalamus in order to evoke the burst discharge pattern of medial septal neurones which could also be elicited following sensory stimulation.

Extracellular activity was recorded using glass micro-electrodes filled with 3M-NaCl, having tip diameters of 1–2 μm and a DC resistance in saline of 1–3 MΩ. The recording electrodes were stereotaxically placed in the medial and lateral regions of the septum at various anterior–posterior and lateral co-ordinates using the juncture of mid line and anterior extremity of the hippocampus as the zero reference.

The signals recorded from the electrode were amplified and displayed on an oscilloscope for photography in the conventional manner. Further analysis was made of the neuronal data using a PDP/8 computer to produce post-stimulus histograms on line. The sites of stimulating and recording electrode placements were confirmed histologically (see details McLennan & Miller, 1974*a*).

RESULTS

Effects of single volley stimulation on lateral and medial septal neurones

A total of seventy-six neurones were examined in this study, forty-one from the lateral septum and thirty-five from the medial septum. Neurones in each of these regions were identified by their characteristic response patterns to single stimuli delivered to the fimbria. A detailed description of these responses has been presented elsewhere (McLennan & Miller, 1974*a*); however a brief review of the findings will be presented here.

Single stimuli delivered to the fimbria evoked single action potentials in lateral septal neurones with latencies of 4–7 msec or 12–18 msec, or both, depending upon the anterior–posterior position of the fimbrial stimulating electrode. The excitation (Fig. 1*G*) in all spontaneously firing lateral septal neurones was followed by a period of inhibition, the duration of which ranged from 50 to 700 msec (Fig. 1*A* and *H*). Small amplitude 'ripples' or spikes occurring in bursts of high frequency were frequently recorded either at the same site as the larger amplitude spikes exhibiting the activation–inhibition sequence or 50–100 μm distant along the recording track (Figs. 3*C*; 4*A*, *B*). The latencies of onset of these high frequency bursts were 0·5–2·0 msec longer than the activation of the large amplitude spikes (Fig. 3*C*) and in all cases coincided with the start of the inhibitory period. These bursts were taken to represent the firing of the inhibitory interneurones of a recurrent collateral system which requires the prior activation of the large amplitude lateral septal relay

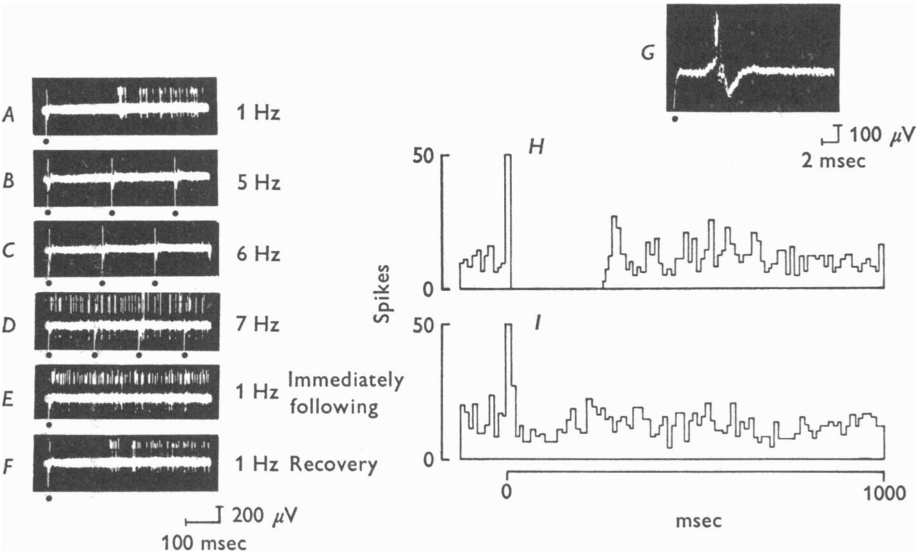

Fig. 1. The response of a lateral septal neurone to different rates of fimbrial stimulation before, during and immediately following tetanic volleys. *A*, the characteristic activation–inhibition sequence evoked by single pulse stimulation (the activation phase is shown on a faster time scale in *G*); *B*–*D*, the effects of stimulation at 5, 6 and 7 Hz respectively. Note the elimination of the inhibitory component during 7 Hz stimulation. The response to single pulse stimulation immediately following a tetanic train of 10 Hz is shown in *E*, and *F*, illustrates recovery to the pre-tetanic response sequence. Each trace represents five superimposed sweeps with the initial deflexion (marked by a black dot) indicating the stimulus artifact and the spike activation. Post-stimulus histograms showing the response to single pulse stimulation before (*H*) and immediately following tetanic volleys of 10 Hz (*I*). Each histogram shows the summed responses to fifty stimuli. In this and subsequent Figures the shock artifact is indicated by the first large deflexion of the histograms, and this bin (10 msec width) also contains the spike activation.

neurones (cf. DeFrance, Kitai & Shimono, 1973; McLennan & Miller, 1974*a*).

Neurones in the medial septal region were identified on the basis of their bursting pattern of firing which occurred either spontaneously or following sensory or hypothalamic stimulation. Single pulse stimulation of the fimbria elicited a short period of inhibition (30–100 msec) in medial septal neurones which exhibited a bursting discharge and a synchronization of this pattern such that each burst became temporally locked to the stimulus (Figs. 5*A*, 6*A*).

Effects of repetitive volleys on lateral septal neurones

The effects of repetitive volleys delivered to the fimbria were examined on lateral septal neurones identified by their responses to single stimuli. Stimulation at 2–6 Hz applied for periods of 1–15 sec produced no change in the spike activation which was seen to follow each stimulus of the train. Spontaneous firing of the neurones however was completely inhibited during the repetitive volley (Fig. 1*B*, *C*), even when the interval between each pulse was greater than the period of inhibition evoked by single stimulation. This effect appeared 2–4 sec after the beginning of the repetitive train and persisted throughout its duration. Following the tetanic volleys single stimuli evoked the same activation–inhibition response sequence as that obtained before repetitive stimulation.

When the frequency of stimulation was increased to 7–12 Hz (optimal 10 Hz) either a decrease in the duration of inhibition or a complete elimination of the inhibitory component was observed (Fig. 1*D*). This effect occurred 3–4 sec after the onset of the repetitive train and persisted for 2–20 sec following its cessation (Fig. 1*E*, *I*) before recovery to the pre-tetanic level (Fig. 1*F*).

Fig. 2 illustrates graphically the effects of different frequencies of fimbrial stimulation on seven lateral septal neurones. The duration of inhibition produced by a single pulse is expressed as 100 % and is compared to the duration of inhibition elicited by single pulses in the first 5 sec after tetanic volleys. There is a consistent decrease in the period of inhibition at rates above 6 Hz indicating this to be a critical frequency for elimination of the inhibitory component. In a few other neurones in which higher frequencies of stimulation were delivered (to 15 Hz), the degree of inhibition was similar to that elicited following 10 Hz stimulation.

Effects of repetitive volleys on inhibitory interneurones

The inhibitory component of the activation–inhibition sequence following single stimuli delivered to the fimbria, was previously considered to be mediated by the excitation of inhibitory interneurones (McLennan

& Miller, 1974*a*). Since repetitive stimulation above 6 Hz influenced this inhibitory component it appeared likely that such stimulation altered the activity of these presumed inhibitory cells. At sixteen sites in the lateral septal region both large and small amplitude spikes were recorded simultaneously and it was possible to analyse the effect of repetitive volleys on both responses. Fig. 3*A* and *B* illustrate the post-stimulus histograms of the firing of a lateral septal neurone to single pulse fimbrial stimulation before and immediately after a period of stimulation at 10 Hz, together with sample records showing the large amplitude spike activation (4 msec) and small amplitude 'ripples' denoting an inhibitory interneurone at various times during the experimental procedure (Fig. 3*C*). The interneuronal activity was not eliminated until 2–3 sec after the start of the tetanic train, and did not reappear until 2–5 sec after the end of the train. Concurrent with the elimination of the interneuronal activity there was a marked decrease in the period of inhibition of the large amplitude spike (Fig. 3*B*). Recovery of the inhibition to pre-tetanic levels occurred simultaneously with the recovery of the interneuronal activity.

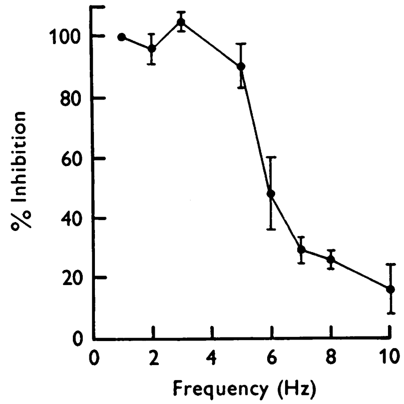

Fig. 2. The effect of tetanic stimulation on the duration of inhibition evoked in lateral septal neurones by single fimbrial stimuli. The points indicate the mean durations of inhibition measured during the 5 sec period after the end of a tetanic train delivered at the indicated frequencies, expressed as a percentage of the values obtained before the tetanus. The vertical bars are S.D. of the observation: results from seven separate neurones have been pooled.

The responses of inhibitory interneurones of the lateral septum to various frequencies of fimbrial stimulation are shown in Fig. 4*A* and *B* where the responses of cells uncomplicated by the larger amplitude spikes of the relay neurones are illustrated. There is relatively little change in the evoked activity with repetitive volleys up to 6–7 Hz. At higher frequencies (7–12 Hz) the interneuronal response is eliminated, an effect which persists for a period of 2–5 sec after the stimulus train before complete recovery to the pre-tetanic response is obtained.

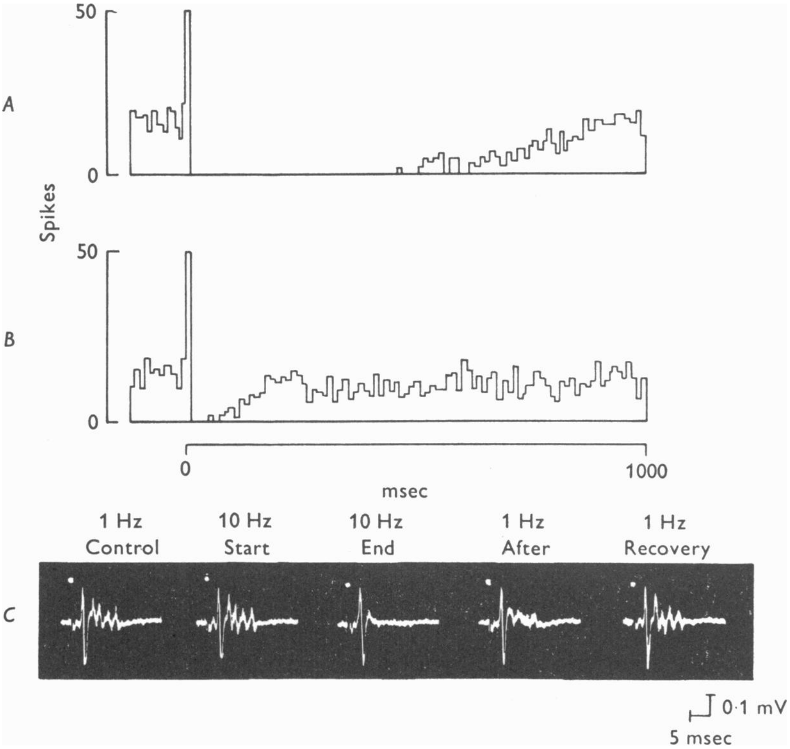

Fig. 3. Post-stimulus time histograms showing two different responses recorded from the same lateral septal neurone following single pulse stimulation of the fimbria (*A*) before and (*B*) in the first 5 sec after a 10 Hz train. Note the decreased duration of inhibition in the latter case. *C*, the large amplitude spike and small amplitude 'ripples' recorded simultaneously in the lateral septum and the changes which occur in these responses during and immediately following 10 Hz stimulation. Each trace represents one sweep; the initial deflexion (marked by a dot) indicates the shock artifact.

Response of medial septal neurones to repetitive volleys

The response pattern of a bursting medial septal neurone to single stimulation of the fimbria before and after repetitive volleys is illustrated in Fig. 5. In all cases examined, the short period of inhibition (30–100 msec) which occurs immediately after the stimulus, is followed by a synchronization of the bursting pattern (Fig. 5*A*). Repetitive volleys of 2–7 Hz did not alter this pattern either during the 10 sec tetanic stimulation or when single volleys were delivered in the period following the repetitive

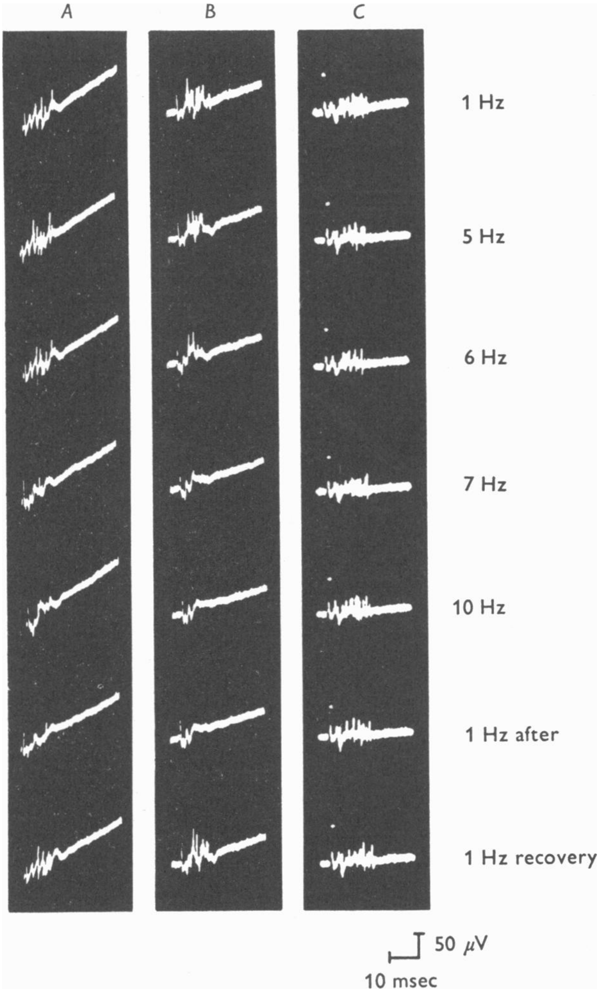

Fig. 4. The responses of inhibitory interneurones to different frequencies of fimbrial stimulation. Rates up to 6 Hz produce relatively little change in comparison to the response to single pulse stimulation (1 Hz). Above 6 Hz the small amplitude burst discharge of lateral septal cells (*A*, *B*) is eliminated, an effect which persists for up to 20 sec after the end of the tetanic stimuli before recovery is complete; however cells of the medial septum (*C*) are unaffected. The stimulus artifacts in (*C*) are marked by dots.

train. However when the frequency of the tetanus was increased above 7 Hz the bursting discharge was eliminated and replaced by an irregular pattern of firing both during and following the train. This effect occurred 2–5 sec after the onset of the train, and persisted for 10–15 sec following its termination. Single volleys delivered during this latter period had no effect other than to produce the initial short inhibition (Fig. 5*B*), with recovery to the first response occurring subsequently (Fig. 5*C*).

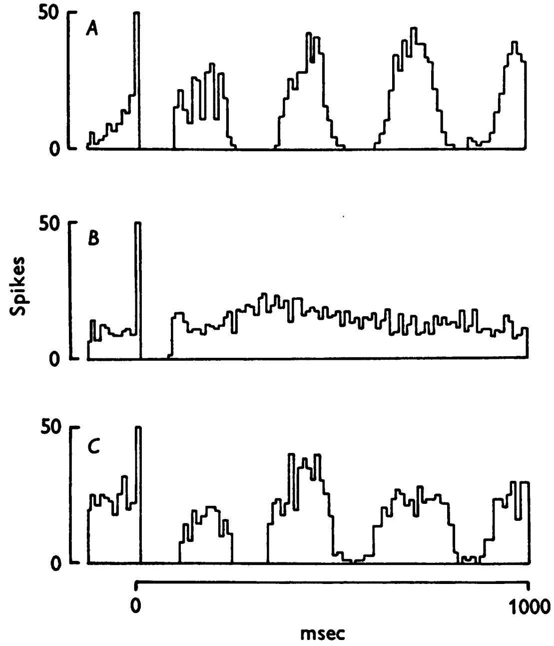

Fig. 5. Post-stimulus histograms showing the responses recorded from the same medial septal neurone to single pulse fimbrial stimulation (*A*) before and (*B*) immediately after tetanic volleys at 10 Hz. Note the elimination of the burst discharge pattern and its consequent recovery 10 sec after the end of the tetanic train (*C*).

A burst of high frequency low amplitude spikes was frequently recorded in the medial septum during the period immediately after stimulation of the fimbria. These responses, which coincide with the beginning of the initial inhibitory period exhibited by medial septal cells, were previously considered to indicate the activation of interneurones which mediate this inhibitory effect (McLennan & Miller, 1974*a*). Tetanic stimulation at rates which blocked the bursting pattern of medial septal cells did not alter the firing of these interneurones (Fig. 4*C*).

Fourteen medial septal neurones did not exhibit the spontaneous bursting pattern of firing, nor could it be evoked by hypothalamic or sensory stimulation (paw pinch). The response of these neurones to single volleys in the fimbria was a period of inhibition lasting 70–400 msec. Repetitive volleys below 7 Hz produced relatively little change in this inhibitory response, whereas volleys at 7–10 Hz resulted in a decrease in the duration of inhibition although never to < 60 msec. The time course of this effect was similar to that observed for lateral septal neurones, i.e. it began 2–3 sec after the start of the train and persisted 3–10 sec after its end.

Effects of section of the fimbria on septal activity

It has been shown that repetitive stimulation of the fimbria at 7–12 Hz resulted in a decrease of the inhibition of lateral septal neurones and elimination of the bursting discharge pattern of medial septal neurones. It would appear therefore that the level of neuronal activity mediated by the fimbria is of critical importance in determining the pattern of

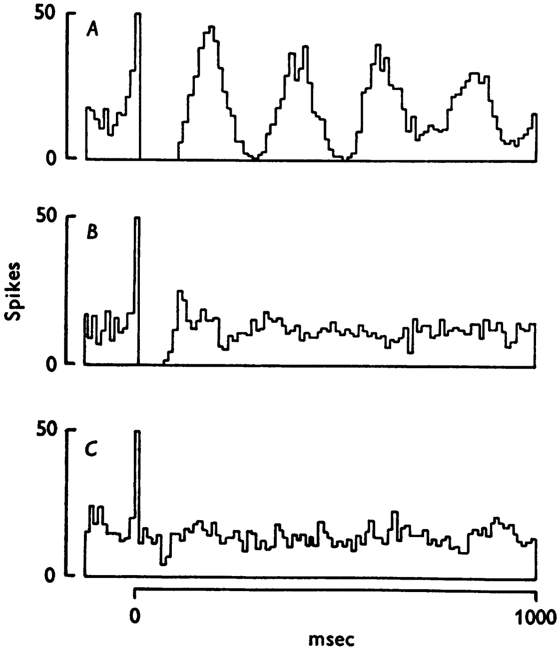

Fig. 6. *A* and *B*, histograms illustrating the characteristic response of a medial septal neurone to single stimuli before and after a tetanic train of 10 Hz delivered to the fimbria. *C*, the effect of single stimuli following section of the fimbrial pathway. Note the elimination of the initial inhibitory period and of the synchronized bursting pattern.

discharge of septal neurones. This possibility was tested by section of the fimbrial input to the septum in eleven experiments, and the effects of this procedure were observed on the discharge of medial septal neurones. Fig. 6 (*A* and *B*) illustrates the typical response of a medial septal neurone to single stimuli before and after a tetanic train of 10 Hz delivered to the fimbria. Ipsilateral section of the fimbria resulted in the complete elimination of any inhibitory component and synchronized bursting pattern elicited by stimulation on the hippocampal side of the lesion (Fig. 6*C*).

In eleven neurones examined following sections of the fimbria, two continued to show bursts spontaneously or as a result of hypothalamic or sensory stimulation, or both. The other nine neurones exhibited an irregular pattern of discharge and were unresponsive to either hypothalamic or sensory inputs. Furthermore, stimulation of the fimbria on the hippocampal side of the section was ineffective in altering either lateral or medial septal neuronal activity, whereas stimulation on the septal side of the section elicited the characteristic activation–inhibition or inhibition sequences respectively, and the effects of increasing frequencies of stimulation were unaltered from those described above.

DISCUSSION

In a previous study it was suggested that a recurrent collateral inhibitory system located in the lateral septal region was responsible for the development of the rhythmic discharge exhibited by medial septal neurones (McLennan & Miller, 1974*a*). Activity arising in the hippocampus and mediated by the fimbrial pathway was shown to activate lateral septal relay cells, the output of which excites both medial septal neurones and recurrent inhibitory interneurones within the lateral septum. Since the output of the lateral septal cells is regulated by a powerful recurrent inhibitory process, they exert a phasic effect on medial septal target neurones producing the characteristic bursting pattern of discharge. The results of the present study provide further evidence to support the notion that this recurrent collateral system is important in regulating the discharge pattern of medial septal cells, and further that the activation or inactivation of the interneurones of this system is dependent upon the frequency of output mediated by the hippocampal fimbrial pathway.

Lateral septal relay neurones, which characteristically respond to single pulse stimulation of the fimbria with an activation–inhibition sequence, exhibited a reduction or complete elimination of the inhibitory component during and following tetanic stimulation delivered at 7–12 Hz. Since the inhibitory component was considered to be mediated by interneurones interposed in the recurrent pathway of the lateral septum, it appeared likely that these cells might respond in a differential manner to varying frequencies of stimulation.

The present results demonstrate that concurrent with the reduction of the inhibitory component of the response shown by lateral septal cells after tetanic stimulation above 6 Hz, there is a reduction and eventual elimination of the discharge of the inhibitory interneurones. By contrast the response sequences of both the relay cells and inhibitory interneurones to lower frequencies of stimulation (2–6 Hz) were identical to those evoked by single pulse stimuli. The time course of these alterations in lateral septal activity was consistent for all neurones influenced by tetanic stimulation. During the initial 4 sec of this stimulation, at rates above 6 Hz, the inhibitory interneurones are still firing thereby inhibiting the discharge of lateral relay cells and keeping their output relatively low. However, with longer periods of tetanic stimulation (4–15 sec) there is a reduction in the inhibitory interneuronal firing and consequently the output from the lateral relay cells increases. This effect persists for periods up to 20 sec following termination of the tetanic stimulation before recovery to the pre-stimulus response sequence.

Similar frequency-related mechanisms have been shown to play a significant role in regulating the output from hippocampal pyramidal neurones. Andersen & Lømo (1966, 1970) demonstrated that single pulse stimulation of the perforant pathway activates a relatively small number of pyramidal neurones but when the frequency of stimulation was increased to 10–12 Hz the number of cells recruited was greatly increased. They suggested that because synapses of the inhibitory interneurones were in close proximity to the soma, pyramidal cells would be under the influence of these neurones and thereby exhibit only a low level of activity. However, with tetanic stimulation the synaptic efficiency of the more distally located excitatory synapses increases sufficiently to produce a depolarization of the neurone which will counteract the recurrent inhibitory process. In the present system however this does not appear to be the case since the inhibitory interneurones are unable to follow frequencies of stimulation above certain levels. Furthermore, relatively few lateral septal neurones exhibited an increased discharge rate to fimbrial stimulation either during or following the tetanic stimuli, an effect which would be expected if there was a recruitment of the excitatory inputs impinging on these cells.

The significance of this frequency gating mechanism in the lateral septal region is made apparent by the effects which different rates of fimbrial stimulation exert on the discharge pattern of medial septal neurones. Tetanic stimulation of the fimbria at rates sufficient to eliminate the inhibitory interneuronal response in the lateral septum, produces an irregular pattern of firing in those medial septal neurones which previously had exhibited a bursting discharge; while, on the other hand, stimulation at rates below 7 Hz appears to maintain and, in many cases, to accentuate the burst–inhibition sequence. Since the frequency of stimulation required to block the bursting discharge response in medial septal neurones is the same as that which eliminates the inhibitory interneuronal activity of the lateral septum, and the time course of the alteration in this firing pattern and of cells of the lateral septum is the same, it is proposed that the recurrent collateral system in the lateral septum is responsible for controlling the discharge pattern of medial septal target neurones. It is noteworthy also that the same frequency of stimulation is identical to that of the upper limit of theta activity described as occurring in unanaesthetized rats (Macadar *et al.* 1970), and under urethane anaesthesia there is evidence that the mean frequency will be even lower (Stumpf, Petsche & Gogolak, 1962). Thus, as long as the lateral interneurones are capable of transmitting impulses, the output from the lateral relay neurones will be phasic and can elicit a rhythmic bursting discharge of the medial cells. However blockade of transmission in the recurrent collateral system will eliminate the usual inhibitory influence of the interneurones, and an irregular output of the medial septal cells will ensue.

Since stimulation of the fimbria has been shown to produce an inhibitory effect on medial septal neurones which is mediated by interneurones synapsing directly on to these cells (McLennan & Miller, 1974*a*), it might be postulated that this mechanism alone could account for the production of the bursting discharge pattern of these cells. Several pieces of evidence indicate that this is not the case. In the present study repetitive stimulation above 6 Hz did not influence either the initial period of inhibition shown by the medial cells (Figs. 5, 6*A*, *B*) or the discharge of the interneurones which mediate this response (Fig. 4*C*), even though the burst pattern of firing was eliminated. This would suggest that the firing characteristics of the inhibitory interneurones in the lateral and medial septum are different and that only the former are responsive to varying frequencies of fimbrial stimulation. Furthermore, the electrophoretic application of bicuculline, which selectively antagonized the GABA-induced and fimbrial evoked inhibition of medial septal neurones, did not influence the bursting pattern in a majority of these cells if they were lying at a distance from the lateral septal region (McLennan & Miller, 1974*b*). These data confirm our previous suggestion that two distinct mechanisms are responsible for regulating the response sequence of the medial neurones; one exerting a direct inhibitory input which synchronizes the firing of a number of the cells while the other serves either to maintain or to eliminate the rhythmical bursting and is dependent on the input transmitted by way of the lateral septum.

In view of these data it is probable that the patterns of discharge exhibited by medial septal neurones are a consequence of hippocampal neuronal activity rather than being pace-makers of the hippocampal rhythm as has been postulated by earlier investigators (Stumpf, 1965). According to the current hypothesis, medial septal neurones are subject to a tonic influx of activity arising in the mesencephalic reticular formation which is also relayed to the hippocampus and results in either a synchronized or desynchronized response pattern. The output from the hippocampus carried by the fimbrial pathway activates the relay neurones in the lateral septal collateral system and, depending on the level of activity in this system, produces either a burst discharge or an irregular firing pattern in medial septal neurones. The regulatory role played by the hippocampus is further emphasized by the results demonstrating that section of the fimbrial pathway eliminates the spontaneous bursting discharge in the medial septum. This procedure presumably has the effect of opening the loop which originates in the medial septum and projects to the hippocampus from whence it is relayed through the lateral septum and back into the medial septum (Raisman, 1966). When this occurs the transmission of information in the feed-back loop from the hippocampus is blocked and the recurrent mechanism in the lateral septum is no longer activated. As a result, the medial septal neurones do not receive a phasic inhibitory input from the lateral relay neurones, and will therefore discharge in an irregular pattern reflecting only the tonic activity ascending from the mesencephalic reticular formation.

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