ULTRA-SLOW OSCILLATION (0.025 Hz) TRIGGERS HIPPOCAMPAL AFTERDISCHARGES IN WISTAR RATS

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Abstract—Oscillations in neuronal networks are assumed to serve various physiological functions, from coordination of motor patterns to perceptual binding of sensory information. Here, we describe an ultra-slow oscillation (0.025 Hz) in the hippocampus. Extracellular and intracellular activity was recorded from the CA1 and subiculum regions in rats of the Wistar and Sprague–Dawley strains, anesthetized with urethane. In a subgroup of Wistar rats (23%), spontaneous afterdischarges (4.7 ± 1.6 s) occurred regularly at 40.8 ± 15.7 s. The afterdischarge was initiated by a fast increase of population synchrony (100–250 Hz oscillation; “tonic” phase), followed by large-amplitude rhythmic waves and associated action potentials at gamma and beta frequency (15–50 Hz; “clonic” phase). The afterdischarges were bilaterally synchronous and terminated relatively abruptly without post-ictal depression. Single-pulse stimulation of the commissural input could trigger afterdischarges, but only at times when they were about to occur. Commisural stimulation evoked inhibitory postsynaptic potentials in pyramidal cells. However, when the stimulus triggered an afterdischarge, the inhibitory postsynaptic potential was absent and the cells remained depolarized during most of the afterdischarge. Afterdischarges were not observed in the Sprague–Dawley rats. Long-term analysis of interneuronal activity in intact, drug-free rats also revealed periodic excitability changes in the hippocampal network at 0.025 Hz. These findings indicate the presence of an ultra-slow oscillation in the hippocampal formation. The ultra-slow clock induced afterdischarges in susceptible animals. We hypothesize that a transient failure of GABAergic inhibition in a subset of Wistar rats is responsible for the emergence of epileptiform patterns. © 1999 IBRO. Published by Elsevier Science Ltd.

Key words: oscillation, afterdischarges, strain, inhibition, hippocampus, subiculum.

Oscillatory activity is one of the basic patterns of neurons and neuronal networks. Population activity of cooperatively active neurons can be best characterized by recording extracellular field activity (electroencephalogram, EEG). The scalp EEG contains various rhythmic patterns in the delta, theta, beta and gamma frequency ranges. Cortical oscillations slower than those in the delta range have also been described.

Extreme forms of neuronal oscillations are present in epileptic afterdischarges. During a single stimulation-induced afterdischarge in the hippocampal formation, oscillatory patterns range from 1 to 400 Hz and the various frequency bands alternate in a predictable manner. Epileptic patterns may arise from genetic factors or because of acquired damage to a selective population of neurons. Although several rodent models are available for studying the genetic influence on thalamocortical oscillations, inherited factors affecting neuronal excitability in the hippocampal formation are poorly understood. Here, we report that, in a subgroup of Wistar rats, spontaneous afterdischarges occur periodically at 40-s intervals.

EXPERIMENTAL PROCEDURES

The experiments were performed on 73 Wistar (HsdBrHan : WIST; National Laboratory Animal Center, University of Kuopio, Kuopio, Finland) and 45 Sprague–Dawley (Hilltop, Scottsdale, PA, U.S.A.) male rats (180–260 g). All efforts were made to minimize animal suffering, to reduce the number of animals used and to utilize alternatives to in vivo techniques. The recording methods were described in detail earlier. In brief, rats were anesthetized with an intraperitoneal injection of urethane (1.2–1.5 g/kg). For recording, each rat was positioned in a stereotaxic apparatus. For extracellular recording, a 1-mm-diameter opening was drilled at AP −4.0 mm and ML 4.0 mm on the left side of the brain after removing the scalp. The dura mater was removed and a 60-µm insulated tungsten wire or a tetrode, consisting of four 12.5-µm Nichrome wires, was advanced at a 30° angle for 1.8 mm to be located in the CA1 pyramidal layer. The exact location of the recording electrode tip was determined by observing evoked field responses to commissural stimulation and spontaneous multi-unit discharges of complex spikes (500–6000 Hz). Another opening, of approximately the same size, was prepared at AP −1.3 mm and ML 1.0 mm, for the placement of an extracellular stimulation electrode in the ventral hippocampal commissure. The stimulation electrode consisted of two parallel 100-µm insulated stainless steel wires. The electrode was first advanced just above the right fimbria–fornix, to a depth of 3 mm, and then in steps of 100 µm while continuously monitoring CA1 field evoked responses (0.1–6000 Hz). Electrical stimulation consisted of 0.2-ms pulses at 40–500 μA delivered every 5 s. Once a typical CA1 evoked field response with a population spike was obtained, the stimulating electrode was fixed to the skull with dental acrylic. The opening was covered by a mixture of paraffin and paraffin oil (50%/50%).

An intracellular micropipette was also placed into either the hippocampus (ipsilateral or contralateral) or the subiculum (ipsilateral). Intracellular recordings were obtained with sharp electrodes pulled on a Sutter pipette puller (Sutter Instrument Company, Novato, CA, U.S.A.) from 1-mm-diameter glass pipettes. Electrodes were filled with 1 M potassium acetate and 2% biocyan, resulting in a resistance of 80–130 MΩ. The electrodes were advanced to the brain through a small opening in the dura, which was cut with a sharp syringe needle. After advancing the electrode by 200 μm, the brain surface was covered with a warm paraffin/paraffin oil mixture and the electrode was then advanced close to the recording depth. To obtain neurons for recording, the electrode was moved in 2-μm steps by a piezoelectric device.
stepping motor (Burleigh Instruments, Fishers, NY, U.S.A.). Once a stable recording with a resting membrane potential more negative than $-55 \text{ mV}$ was obtained, the cellular characteristics of the neuron were defined by applying 200- or 600-ms duration polarizing pulses ($0.8$ to $0.8 \text{ nA}$, in steps of $0.1$ or $0.2 \text{ nA}$) every 5 s. Synaptic responses were characterized by delivering 0.2-ms-long electrical stimulation pulses to the fimbria–fornix at 40-$\mu\text{A}$ steps, up to current levels twice the threshold of the extracellular population spike in the CA1 pyramidal layer. Spontaneous intracellular activity, together with extracellular activity, was recorded at different polarization levels. For evoking theta oscillation, the tail or the hindleg was pinched with tweezers. Intracellular signals were recorded with Axoclamp-2B (Axon Instruments, Foster City, CA, U.S.A.) in current-clamp mode. Extracellular signals were first amplified 20 times with a high-impedance preamplifier (a.c. coupled at 0.1 Hz). Both intra- and extracellular activity was recorded at different polarization levels. For evoking theta oscillation, the tail or the hindleg was pinched with tweezers.

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After data collection, biocytin was injected through a bridge circuit using 500-ms depolarizing pulses at $0.8$–$1.8 \text{ nA}$ at 1 Hz for 10–45 min. The duration of biocytin injection and the current levels were adjusted according to the condition of the neuron.

At least 2 h after the last cell injection, the rats were given a lethal injection of urethane and perfused through the heart. The recorded cells were anatomically identified using procedures described previously.34 Data were analysed off-line with AxoScope (Axon Instruments, Foster City, CA, U.S.A.), Mini Analysis (Synaptosoft, Leonia, NJ, U.S.A.), Stranger (Biographics, Winston-Salem, NC, U.S.A.) and Origin (Microcal Software, Northampton, MA, U.S.A.) or custom-made programs.

Extracellular unit data were collected in two drug-free animals (Sprague–Dawley). These two animals were part of a study published previously.10 The methods of implantation, recording and separation of unitary activity were described in that paper. In brief, electrical activity was recorded with either wire tetrodes or silicon electrode arrays. For the examination of slow network oscillation, we recorded CA1 area unit activity continuously for up to 3 h (amplification: $\times 5000$–$10,000$; 5 kHz low-pass filtering; 10 kHz sampling frequency; 16-bit sampling resolution; DataMax, RC Electronics, Santa Barbara, CA, U.S.A.). Data were analysed off-line using a personal computer running under

Fig. 1. Spontaneous afterdischarge in the CA1 pyramidal layer of the hippocampus. (A) The afterdischarge was initiated by a large-amplitude field potential (20–60 ms duration, filled arrow), followed by a low-amplitude, high-frequency (100–250 Hz) “tonic” phase and a period 3- to 6-s large-amplitude beta frequency oscillation (“clonic” phase). (B) Power spectrum computed for the whole afterdischarge period. Note the peaks at 0.8 Hz (clipped) and 16 Hz and higher harmonic frequencies (32 and 48 Hz). (C) The wide-band signal, in this and subsequent figures, is a.c. coupled at 0.1 Hz and low-pass filtered at 6000 Hz with a fourth-order elliptical filter. Unit activity was separated by additional high-pass filtering at 500 Hz. Note the relationship between field potential and multiple unit activity. Lower trace: instantaneous frequency of beta oscillation, defined as the reciprocal of the intervals between the positive peaks of the field beta oscillation. The oscillation frequency steadily declined from 18 to 10 Hz. (D) The afterdischarges recurred regularly at an ultra-slow frequency (42-s intervals, corresponding to 0.025 Hz). The first of the afterdischarges is the one shown in A and C on expanded time-scales. Negativity is down in this and subsequent figures for both extra- and intracellular recordings.
the LINUX operating system. Signals were pass-band filtered (0.8–5 kHz) digitally and units were separated with custom-made programs.

RESULTS

Ultra-slow oscillation in the hippocampus

Hippocampal activity in the anesthetized rat consists of various irregular field potentials and oscillations.\(^{20,21,34,35,42,61,62}\) Deep anesthesia is characterized by large-amplitude, low-frequency oscillation (0.5–1 Hz, slow delta waves; Fig. 1A), associated with phase-locked discharges of multiple unit activity. Superimposed on this slow rhythm, lower amplitude waves in the gamma (30–100 Hz) and beta (10–30 Hz) are typically present, similar to the delta-modulated gamma oscillation in the neocortex.\(^{8,56}\) The delta waves are often grouped into 3- to 5-s-long periods separated by relative quiescence for 2–5 s, giving a slower rhythm at 0.1–0.2 Hz.\(^{50}\) In the lightly anesthetized rat or in response to strong sensory stimulation (e.g., hindleg or tail pinching), faster rhythmic waves (2–6 Hz, referred to as “theta” oscillation in the hippocampus) replace the large-amplitude slow waves.\(^{31,42,62}\)

In addition to the above rhythms, we observed a much slower oscillation in time-compressed recordings (Fig. 1D, Fig. 3D). This ultra-slow rhythm was especially evident in a subgroup of Wistar rats with spontaneously occurring afterdischarges (see below). The interval between the increased excitability periods ranged from 14 to 70 s (mean = 40.8 ± 15.7 s S.D.; n = 17; or 0.025 Hz). The ultra-slow oscillation did not disappear after sensory stimulation, even though the state change was clearly reflected by the suppression of delta oscillation and the appearance of theta waves (Fig. 2). Further support for the stability of the ultra-slow oscillation comes from stimulation experiments. Afterdischarges could be triggered by single-pulse stimulation of the commissural afferents (see below). Commissural stimulation was applied at 10-s intervals. This period was about one-fourth of the interval between spontaneous sequences and about twice the length of individual afterdischarges (Fig. 3D). Stimuli applied 10–20 s after the afterdischarge occasionally evoked a short, “abbreviated” afterdischarge. Stimuli delivered at a later time-points were ineffective, but pulses delivered at about the time when the occurrence of the spontaneous event was expected could trigger an afterdischarge.

After observing the ultra-slow oscillation in the anesthetized Wistar rats, we questioned whether this rhythm also exists in the awake rat. Because population behavior in hippocampal networks is reliably predicted by the activity of interneurons,\(^{10}\) we examined the autocorrelograms of three hippocampal interneurons in two rats. The examined sessions lasted for 1–3 h, most of which time the animals spent in sleep. The autocorrelograms of one interneuron from each rat are shown in Fig. 4. The wide time distribution of the activity around time zero and the additional peak at 25- to 45-s intervals indicate the presence of the ultra-slow network oscillation in intact, drug-free rats as well. Taken together, the findings indicate that excitability in the hippocampal formation fluctuates at an ultra-low frequency of 0.025 Hz.

Spontaneous hippocampal afterdischarges in rats of the Wistar strain

Spontaneous afterdischarges were observed in 17 of the 73 Wistar rats (23%), but in none of the animals of the Sprague–Dawley strain. The afterdischarge could be divided into initial “tonic” and secondary “clonic” phases. The onset of the afterdischarge was marked by a rapid increase of the background unit discharge, culminating in a large synchronous population discharge of many neurons. This “population discharge” was associated with a large-amplitude sharp field potential (50–100 ms) of positive polarity in the pyramidal layer. The spike event was followed by a low-amplitude fast field oscillation, associated with unit discharge (“tonic” phase). The initial frequency of the fast oscillation was 250 Hz, which gradually slowed to 100 Hz (e.g., Fig. 5). Cross-correlation of the fast field oscillation with the multiple unit discharges revealed that unit activity was phase locked to the negative peaks of the local field (not shown).

The “tonic” phase (<0.5 s) was followed by a longer, large-amplitude field oscillation. The average duration of this “phasic” oscillation period was 4.73 s (S.D. = 1.56 s; n = 17). The phasic pattern consisted of either a long period of beta frequency oscillation (e.g., Fig. 1B, C) or a mixture of gamma and beta frequency waves (Fig. 3A). In both cases, the initially faster frequency slowed down considerably by the end of the afterdischarge, from 16–22 to 8–10 Hz (Figs 1C, 5). When slow delta waves were present prior to the onset of the spontaneous afterdischarge, delta and beta frequency oscillations coexisted (Fig. 1A, C). The afterdischarges terminated rather abruptly without any obvious “post-ictal” suppression of the background activity, typical of the
stimulation-induced “kindling” type seizures. Multiple unit activity was phase locked by the large-amplitude beta oscillatory field (Figs 1C, 3B, C). Faster time resolution of the individual beta waves revealed a sharp negative spike-like component, superimposed on the positive waves (arrows in Fig. 3B and C). This field component likely reflected the summed activation of action potentials of nearby neurons, because it coincided with the most synchronous part of the multiple unit discharges.

Intracellular recording from either CA1 pyramidal neurons or subicular cells confirmed the above conclusions deduced from the cross-correlation between field activity and multi-unit discharges. The “tonic” phase was associated with a large (5–25 mV), steep depolarization of the membrane, associated with a barrage of action potentials (Fig. 5). In the “clonic” phase, the action potentials occurred on the rising phase of the beta frequency oscillation of the membrane potential (Fig. 5).

Although the duration of individual afterdischarges was rather constant, shorter events were also observed (Fig. 6). In these events, the duration and shape of the “tonic” phase were remarkably similar and the variability was due to the shortening of the “clonic” phase. The “tonic” phase, but not the “clonic” phase, could occur in isolation (Fig. 6A), indicating that the “tonic” phase was a prerequisite for the induction of the beta frequency oscillation.
The spontaneous afterdischarge occurred virtually simultaneously in both hippocampi, as revealed by simultaneous recording of intracellular activity and extracellular patterns in both hemispheres (Fig. 7). Simultaneous recordings from both the CA1 region and subiculum indicated that the afterdischarge also spread to the subiculum (Fig. 9).

As indicated above, afterdischarges could also be triggered by single-pulse stimulation of the commissural input. The stimulation, however, was effective only in animals which showed spontaneous afterdischarges and at times when the spontaneous events were about to occur. The evoked afterdischarges differed from the spontaneous events only in the “tonic” phase. In contrast to the initial spike-like event and fast oscillation, the “clonic” phase was initiated by a large evoked response, followed by suppression of neuronal activity (Fig. 3B).

Spontaneously occurring afterdischarges were associated with a relatively steady depolarization in intracellularly recorded neurons (n = 3 CA1; n = 10 subiculum). The steady depolarization was especially prominent in subicular pyramidal cells. Hyperpolarization of the neuron increased the amplitude of the beta frequency oscillation (Fig. 8), indicating that a major part of the oscillation was brought about by depolarizing potentials. In two cases, varying the membrane potential by steady current injection into the neuron revealed a potential at the beginning of the “tonic” phase, which reversed in polarity between 60 and 75 mV (Fig. 8A, B). Because this voltage range corresponds to the equilibrium potential of chloride ions, we suggest that this early potential is mediated by GABA_A receptors. In the remaining neurons, this early hyperpolarizing potential was not observed. Instead, the onset of the spontaneous or evoked afterdischarge was reflected by a step-like depolarization of the membrane potential. When commissural stimulation was applied between the spontaneous afterdischarge events, it evoked a hyperpolarization or an excitatory postsynaptic potential followed by a hyperpolarization. In contrast, the same stimulation evoked a steady depolarization when it triggered an afterdischarge (Fig. 9).

**DISCUSSION**

The novel findings of the present experiments are (i) the presence of an ultra-slow oscillatory pattern (0.025 Hz) in the hippocampal formation, and (ii) the occurrence of spontaneous afterdischarges in a subgroup of rats of the Wistar strain. These spontaneous afterdischarge episodes were associated with a relatively steady depolarization in intracellularly recorded neurons (n = 3 CA1; n = 10 subiculum). The steady depolarization was especially prominent in subicular pyramidal cells. Hyperpolarization of the neuron increased the amplitude of the beta frequency oscillation (Fig. 8), indicating that a major part of the oscillation was brought about by depolarizing potentials. In two cases, varying the membrane potential by steady current injection into the neuron revealed a potential at the beginning of the “tonic” phase, which reversed in polarity between 60 and 75 mV (Fig. 8A, B). Because this voltage range corresponds to the equilibrium potential of chloride ions, we suggest that this early potential is mediated by GABA_A receptors. In the remaining neurons, this early hyperpolarizing potential was not observed. Instead, the onset of the spontaneous or evoked afterdischarge was reflected by a step-like depolarization of the membrane potential. When commissural stimulation was applied between the spontaneous afterdischarge events, it evoked a hyperpolarization or an excitatory postsynaptic potential followed by a hyperpolarization. In contrast, the same stimulation evoked a steady depolarization when it triggered an afterdischarge (Fig. 9).
triggered by the ultra-slow cyclic increases of network excitability.

Ultra-slow network rhythm in the hippocampus

Hippocampal networks are known to sustain various intermittent population events and oscillatory patterns at various frequencies, including rhythms in the delta, theta, beta, gamma and ultra-fast bands (cf. Ref. 24). In the anesthetized rat\textsuperscript{21,24} and hippocampal slices perfused with CsCl,\textsuperscript{63} slower oscillations (0.2–1 Hz) have also been observed.

Slow rhythms have also been described in the thalamo-cortical system. Steriade \textit{et al.}\textsuperscript{50} defined a slow neocortical oscillation in nitrous oxide or ketamine/xylazine (0.6–1 Hz) or urethane (0.3–0.4 Hz) anesthetized cat. Because the slow rhythm survived complete damage of the ipsilateral thalamus, the authors concluded that the slow rhythm arises from neocortical networks.\textsuperscript{49} Subsequently, the slow rhythm was also described in the scalp EEG of healthy humans during slow-wave sleep using Fourier spectral analysis.\textsuperscript{1} In a genetic study of spike-and-wave epilepsy, periodic recurrence of the epileptic pattern has been described in the rat at 10- to 30-s and 15- to 30-min intervals.\textsuperscript{17} The period of the former oscillation corresponds to the ultra-slow rhythm observed in the present study. Importantly, cyclic changes of cortical excitability at this ultra-slow pace have also been observed in human epileptic patients. Terzano \textit{et al.}\textsuperscript{54,55} described cyclic variation of sleep spindles and spike-and-wave patterns in petit mal patients. Our findings in intact, drug-free rats (Sprague–Dawley strain) revealed that epileptic patterns are not a prerequisite of the ultra-slow oscillatory pattern. Because the pattern was also observed in the intact rat, one may hypothesize that the periodic occurrence of afterdischarges in a subgroup of Wistar rats was a consequence of a physiological fluctuation of network excitability. The periodically enhanced excitation simply triggered afterdischarges in the seizure-susceptible brain (see below).

On the basis of the above findings, one may hypothesize that the ultra-slow oscillation emerges in neocortical networks and is relayed to the hippocampal formation by the entorhinal cortex. Alternatively, the slow rhythm may reflect a dynamic interaction between local blood supply and neuronal activity.\textsuperscript{14} Imaging studies of cortical activity often report rhythmic changes of light reflection and/or blood oxygenation at periods slower than 5 s. To date, it is not clear whether the primary effect in these experiments derive from the autonomic nervous system or cortical neuronal activity. Whereas the present findings cannot refute either of the above scenarios, support for the primary role of the hippocampal neuronal network in the emergence of ultra-slow oscillation may come from \textit{in vitro} observations. Bath application of carbachol or activation of metabotropic glutamate receptors can induce periodically enhanced excitatory events in hippocampal slices at intervals of 20–60 s.\textsuperscript{5,12,27,36,53} Although the continuous presence of a drug in these studies is critical for the continued presence of the hyperexcitable events, these studies suggest that the hippocampal network alone may be sufficient for controlling the intervals between the occurrence of hyperexcitatory events.\textsuperscript{44} Both the mechanism and the physiological relevance of the ultra-slow oscillation remain subjects for future exploration.

Spontaneous afterdischarge episodes in Wistar rats

In approximately 25% of the Wistar rats, but in none of the Sprague–Dawley animals, spontaneous afterdischarges were observed.
regularly triggered by the ultra-slow oscillation. The pattern of the afterdischarge was similar to the complex partial seizures observed in the human hippocampus,\(^{13}\) including an initial short “tonic” phase followed by a longer epoch of “clonic” pattern. Unlike the stimulation-induced afterdischarges, used in kindling studies,\(^{38,43}\) the afterdischarge was not followed by post-ictal depression of neuronal activity. A possible explanation for this difference is the shorter duration of the afterdischarge observed in the Wistar animals (\(<5\) s) compared to the stimulation-induced epileptiform patterns (\(>15\) s).\(^{6,25}\) The spontaneous afterdischarge terminated rather abruptly and EEG then returned immediately to the normal background pattern. The occurrence of the “tonic” phase was a prerequisite for the induction of the “clonic” phase, because the high-frequency bursts could occur in isolation, but the “clonic” pattern occurred without the initial population bursts only when the afterdischarge was induced by electrical stimulation. Thus, the synchrony of neuronal activity in the “tonic” phase appeared to determine the length of the “phasic” afterdischarge. In slice and model experiments, weak electrical stimulation induced only a short high-frequency oscillation,\(^{60}\) whereas strong stimuli induced a longer afterdischarge. Similarly to our \textit{in vivo} observations, the frequency of the clonic pattern gradually decelerated from the gamma to the slower frequency beta range.\(^{58,59}\)

The striking difference in the occurrence of afterdischarges between rats of the Wistar and Sprague–Dawley strains suggests that the epileptiform patterns are genetically determined. Wistar rats have an unusually high incidence of generalized spike-and-wave patterns,\(^{8}\) and have been used for genetic selection studies.\(^{28}\) In another genetic study, the F2 descendents of Fischer 344 and Brown–Norway rats had a very high incidence of generalized spike-and-wave patterns. Importantly, the temporal recurrence of spike-and-wave patterns was similar to the intervals of spontaneous afterdischarges observed in the present study.\(^{17}\) One might argue, therefore, that the afterdischarges described here were simply induced by the spread of neocortical spike-and-waves to the otherwise intact hippocampal formation. However, simultaneous recordings from the neocortex and hippocampus revealed that thalamocortical rhythms only exceptionally invade the hippocampal formation in rats.\(^{72}\) Conversely, physiological and anatomical changes have been described in both the thalamocortical and limbic systems of mutant mice.\(^{16,37}\) Our intracellular observations also indicate that pathophysiological changes within the hippocampal formation might be important for the occurrence of

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**Fig. 8.** Voltage dependence of the magnitude of intracellular potential fluctuation during spontaneous afterdischarges in a CA1 pyramidal cell. (A) Note that the amplitude of beta oscillation increased with increasing hyperpolarization of the membrane. Resting membrane potential was \(-69\) mV. Letters B and C mark an initial hyperpolarization (at rest) and the peak of the “tonic” phase. (B) The amplitude plot of event B in A as a function of membrane potential. Note the amplitude minimum and phase reversal between \(-65\) and \(-75\) mV, suggesting that this early component was, at least partially, mediated by GABA\(_A\) receptors. Component B increased linearly in the depolarizing direction. (C) The amplitude plot of event C in A as a function of membrane potential. Component C decreased linearly in the depolarizing direction.
spontaneous afterdischarges. Although single-pulse stimulation evoked large inhibitory postsynaptic potential in pyramidal neurons, such evoked inhibitory postsynaptic potentials were conspicuously absent when the stimulus triggered an afterdischarge. Thus, the failure of GABAergic inhibition by an undisclosed mechanism might be responsible for the enhanced excitability in a subgroup of Wistar rats.

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**Note added in proof**—After this paper was accepted, we found that slow oscillations have also been described in other brain areas [Albrecht D., Royl G. and Kaneko Y. (1998) *Neurosci. Res.* 32, 219–223; Ruskin D. N., Bergstrom D. A., Kaneko Y., Patel B. N., Twery M. J. and Walters J. R. (1999) *J. Neurophysiol.* 81, 2046–2055].