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Evoked oscillations in unit recordings from the thalamo-cortical auditory system: an aspect of temporal processing or the reflection of hyperpolarized brain states?

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Minireview

Abstract. Since the beginning of the 90's several lines of research have brought new insights concerning the temporal aspects of sensory processing. Initial observations showing that sensory stimuli can trigger evoked, or induced, oscillations has generated a large number of studies where, explicitly or implicitly, the large-scale rhythmic activity of thalamo-cortical neurons was viewed as a key factor to synchronize neurons responding to different dimensions of a given stimulus, and therefore to solve the so-called "binding problem" (for reviews see Eckhorn 2000, Singer 1990, 1999). This line of research contrasts with the rate coding concept which has been used over the last 40 years to describe the functional properties of neurons in sensory systems and the plasticity of sensory systems both during development and in adulthood. We review here results obtained in the thalamo-cortical auditory system. After a characterization of the oscillatory events, we determined their origins by inactivating each component of the thalamo-cortical loop. Then we compared, within the same animals, the oscillations obtained in anesthetized and unanesthetized conditions. Our data strongly suggest that to clarify the functional roles of evoked oscillations, and more generally of any aspects of temporal coding, we need to study them on awake animals.

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FIRING RATE AND TOPOGRAPHIC MAPS: TWO CORNERSTONES IN SENSORY PHYSIOLOGY

Since the very first studies performed in the early sixties, the firing rate was usually considered as the unique code for the transmission of information within the central nervous system. According to the rate coding description, the more a neuron is firing action potentials at presentation of a given stimulus, the more efficiently it is suspected to process information concerning this stimulus. This rational has been used to unravel the functional properties of neurons in all sensory modalities. Using modulation of firing rate as metric, ocular dominance and orientation selectivity were described in the visual system (Hubel and Wiesel 1962, 1963), frequency tuning in the auditory system (Erulkar et al. 1956, Hind et al. 1960) and the selectivity of cortical neurons for precise zones of the somatosensory epithelium (Powell and Mountcastle 1959). In addition, analyzing the distribution of neuronal selectivity across cortical and subcortical areas revealed the topographic organization maintained from the periphery up to primary sensory cortices in the visual, auditory and somatosensory modality.

Subsequently, rate coding was used to describe sensory plasticity, both at the single cell and at the map level. At the single cell level, an increased firing rate at presentation of a given stimulus was always considered as unambiguous signature of a more efficient processing for this stimulus. At the map level, it was assumed that the larger is the number of neurons responding to a stimulus in a given brain region, the more efficient is the processing of that stimulus over the whole region. Therefore, extensions of topographic maps were always considered as benefit for processing a particular stimulus. In a way, this view is justified when looking at evolution. When a species makes extensive use of a particular region of the sensory epithelium for its daily behavior, the amount of cortical tissue responding to this peripheral region is dramatically expanded. Rodents make extensive use of their vibrissae in their daily behavior, and the rodent barrel cortex, whose counterpart is the hand representation in primates, extend on large territories. In the auditory system, the cortical area responding to the bat vocalization frequency occupies 50% of its primary auditory cortex, whereas the human auditory cortex contains large territories responding to 500-2,000 Hz, the frequency range the most represented in speech. However, considering that experience-induced plasticity replicates, in a short-time scale, the adaptive selection operating through evolution, is a seductive but may be a too simplistic view. Without denying the importance of cortical maps in sensory processing (Kaas 1997), an important literature stresses the fact that other factors than the firing rate and the size of cortical areas underlie the neural code and ultimately behavioral performance.

THE MULTIPLE FACETS OF NEURAL CODING: TEMPORAL ASPECTS OF NEURONAL DISCHARGES

No doubt that, today, most of the neuroscientists will accept the idea that firing rate is only one of the actors allowing information to be encoded in the central nervous system. It is not possible to develop here the various facets by which neurons might code information (for reviews see Eggermont 1998, 2001), but a large number of studies, particularly in the auditory system have pointed out that important aspects of sensory coding might be missed by focusing exclusively our attention on firing rate. However, it is first necessary to consider that behind the words "temporal coding" several non-exclusive levels of temporal organization can be envisioned. These different levels correspond to "who" is considered as sender and receiver of information: a single neuron, a small group of neurons, or a large population of cells.

At the single cell level, temporal coding can mean that the exact time of occurrence of the action potentials, and/or the succession of the interspike intervals, give an accurate representation of a stimulus. At the level of small cell assemblies, temporal coding can be expressed by the short-time scale coordinations of neuronal discharges as assessed by cross-correlograms. At the level of large populations of cells, synchronization of neuronal populations that respond to different parameters of a given stimulus (the carrier frequency, the frequency modulation, the first- and second-order amplitude modulation of a sound) can also be part of the neural code. As oscillations triggered at presentation of acoustic stimuli are, probably, the easiest way to synchronize large neuronal populations, oscillations might help solving the "binding" problem, i.e., the integration of different sensations in a single percept (for reviews see Engel et al. 2001, Kreiter and Singer 1996). In the following paragraphs, we will review results on large scale oscillations in the thalamo-cortical auditory sys-

tem and we will emphasize the fact that looking at these aspects of temporal coding in anesthetized animals gives very little, if any, information on how sensory processing operates in awake animals.

EVOKED OSCILLATIONS IN THE THALAMO-CORTICAL AUDITORY SYSTEM

Since their original description (Eckhorn et al. 1988, Freeman and Van Dijk 1987, Gray and Singer 1989), stimulus-evoked neuronal oscillations have been an intense field of research. Initially, these oscillations have been studied in the visual system where their frequency was often found to be in the gamma range (30-90 Hz). In the visual cortex, they were mostly described as non stimulus-locked ("induced oscillations"). Originally, these high-frequency oscillations were obtained under anesthesia (mainly under NO/O_2 anesthesia: Eckhorn and Obermueller 1993, Engel et al. 1990, 1991a,b, Ghose and Freeman 1992, Gray et al. 1989, 1990) and their presence in undrugged animals was questioned when some laboratories failed to detect them in these conditions (Bair et al. 1994, Tovée and Rolls 1992, Young et al. 1992). Nonetheless, several subsequent studies performed in awake animals described oscillations in proportions equal or superior to those obtained in anesthetized animals (Friedmann-Hill et al. 2000, Gray and Viana Di Prisco 1997, Kreiter and Singer 1992, Maldonado et al. 2000).

In the auditory modality, relatively few studies have described evoked oscillations, and among them, a clear dichotomy exists between those using spiking activity (multiunit or single unit recordings) and those using local field potentials (LFP). Most of the studies using unit activity reported low frequency oscillations. For example in auditory cortex, long latency responses, or "afterdischarges", with an inter-burst frequency of about 10 Hz were observed at presentation of tones (Maldonado and Gerstein 1996, Sally and Kelly 1988) or of trains of clicks (Eggermont 1992) under various anesthetic conditions (urethane, ketamine, pentobarbital). Also, rhythmic discharges of about 8-12 Hz have long been described in the auditory thalamus: they were named "cyclical activity" by Galambos and colleagues (1952), "reverberatory responses" by Aitkins and colleagues (1966), and more recently "rhythmic responses" by Bordi and Ledoux (1994). Lastly, in the auditory sector of the reticular nucleus, both acoustic stimuli and electrical stimulation of the inferior colliculus can trigger rhythmic bursts discharges at about 8 Hz (see Figs. 1 and 3 in Shosaku and Sumitomo 1983). In contrast, LFP recordings performed by Barth and colleagues systematically revealed high frequency "induced" gamma oscillations in the auditory cortex of halothane anesthetized rats (Barth and MacDonald 1996, Franowicz and Barth 1995, MacDonald and Barth, 1995). These oscillations were already present during spontaneous activity and had a complex temporal decay after a brief (1 ms) click presentation: they disappeared at stimulus presentation, re-appeared 300 ms later and intensified 600-800 ms after stimulus onset. High frequency induced oscillations were also recently reported from LFP recordings obtained in auditory cortex of ketamine anesthetized monkeys (Brosch et al. 2002).

In a set of experiments, we have studied the oscillations triggerred in the thalamo-cortical auditory system by acoustic stimuli. Multi-unit activity was simultaneously recorded in auditory cortex, auditory thalamus (medial geniculate body, MGB) and in the auditory sector of the reticular nucleus (auditory RE). Both stimulus-locked oscillations and non stimulus-locked oscillations were detected under anesthesia, but, as the stimulus-locked ones were the most prominent, experiments were designed to provide a better description of this particular type of oscillations. After assessing some of the factors that influence their occurrence and their characteristics (Cotillon et al. 2000), we have looked for their origins by inactivating each component of the thalamo-cortical loop (Cotillon and Edeline 2000). Finally, we have compared, in the same animals, the oscillations (stimulus locked and non stimulus-locked) observed under various anesthetics conditions and those obtained during waking, slow-wave sleep and paradoxical sleep (Cotillon-Williams and Edeline 2003). All experimental procedures were performed in conformity with national (JO 887-848) and European (86/609/EEC) legislations on animal experimentation, which are similar to those described in the "Guidelines for the Use of Animals in Neuroscience Research of the Society of Neuroscience".

CHARACTERISTICS OF RELIABLE STIMULUS-LOCKED OSCILLATIONS

From a database of 265 recordings obtained under urethane anesthesia and exhibiting clear "on" evoked responses, we observed reliable evoked oscillations in

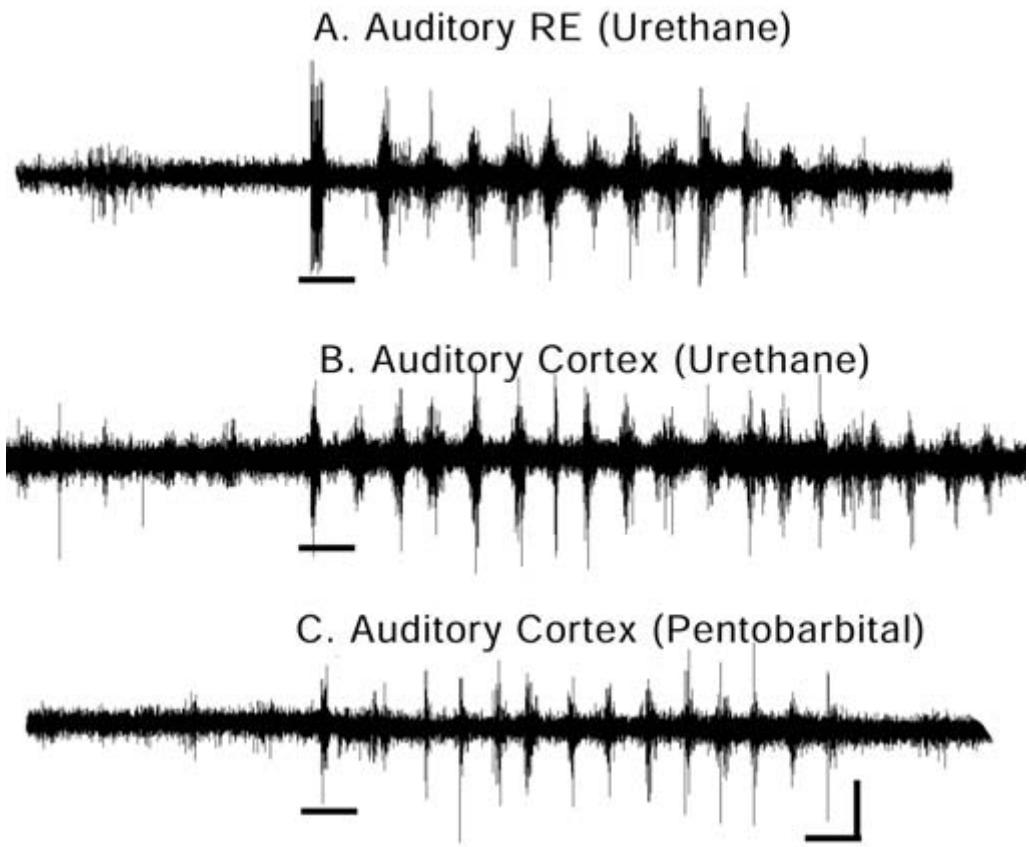

Fig. 1. Examples of oscillations obtained from the auditory sector of the reticular nucleus (A) and from the auditory cortex (B and C) under urethane anesthesia (A and B) and pentobarbital anesthesia (C). In (A), (B) and (C), the traces (50 kHz sampling rate) display the raw neuronal activity. Note that the "on" tone-evoked response (bars of 100 ms below the traces) is followed by rhythmic discharges for several hundreds of milliseconds. Scale bars = 100 ms, 150 μV

28% of the cases in the auditory cortex, in 16.5% of the cases in the auditory thalamus and 42% in the auditory RE. Fig. 1 provides examples of multiunit recordings exhibiting such oscillations under two different anesthetics. The term "reliable" refers to the fact that evoked oscillations were obtained for at least 50% of the stimulus presentations over periods of about 60 minutes. In contrast, "labile" oscillations correspond to recordings exhibiting oscillatory patterns in less than 50% of the stimulus presentations (see Cotillon et al. 2000 for details). All subsequent descriptions of tone-evoked oscillations correspond to data obtained from recordings exhibiting reliable oscillations.

As shown in Fig. 2, stimulus-locked oscillations were so reproducible at stimulus presentation that they can be directly observed on peri-stimulus time histograms (PSTHs). Analyzing the frequency of these evoked oscillations revealed that, in the three investigated structures, the frequency range was between 5 and 15 Hz; only a few recordings obtained in the auditory RE exhibited oscillations above 15 Hz (Fig. 3). Interestingly, we observed that the oscillation frequency was clearly function of the animal's temperature. For a set of recordings (n = 8), tone-evoked oscillations were obtained at physiological temperature (37-38°C), then at lower temperature (35-36°C). As illustrated in Fig. 4, lowering the temperature reduced the frequency and increased the duration of these oscillations, but it did not change their probability of occurrence. In contrast, the interstimulus interval was a key factor controlling the occurrence of these oscillations (Fig. 4E1-E4): they were never observed for inter-tone intervals (ITI) shorter than 1 second, and only one case was obtained with an ITI of 1second (Cotillon et al. 2000).

As in many cases, at least two recordings were collected within the same structure, and at least in two structures, we were able to determine whether these oscillations occurred simultaneously in the thalamo-corti-

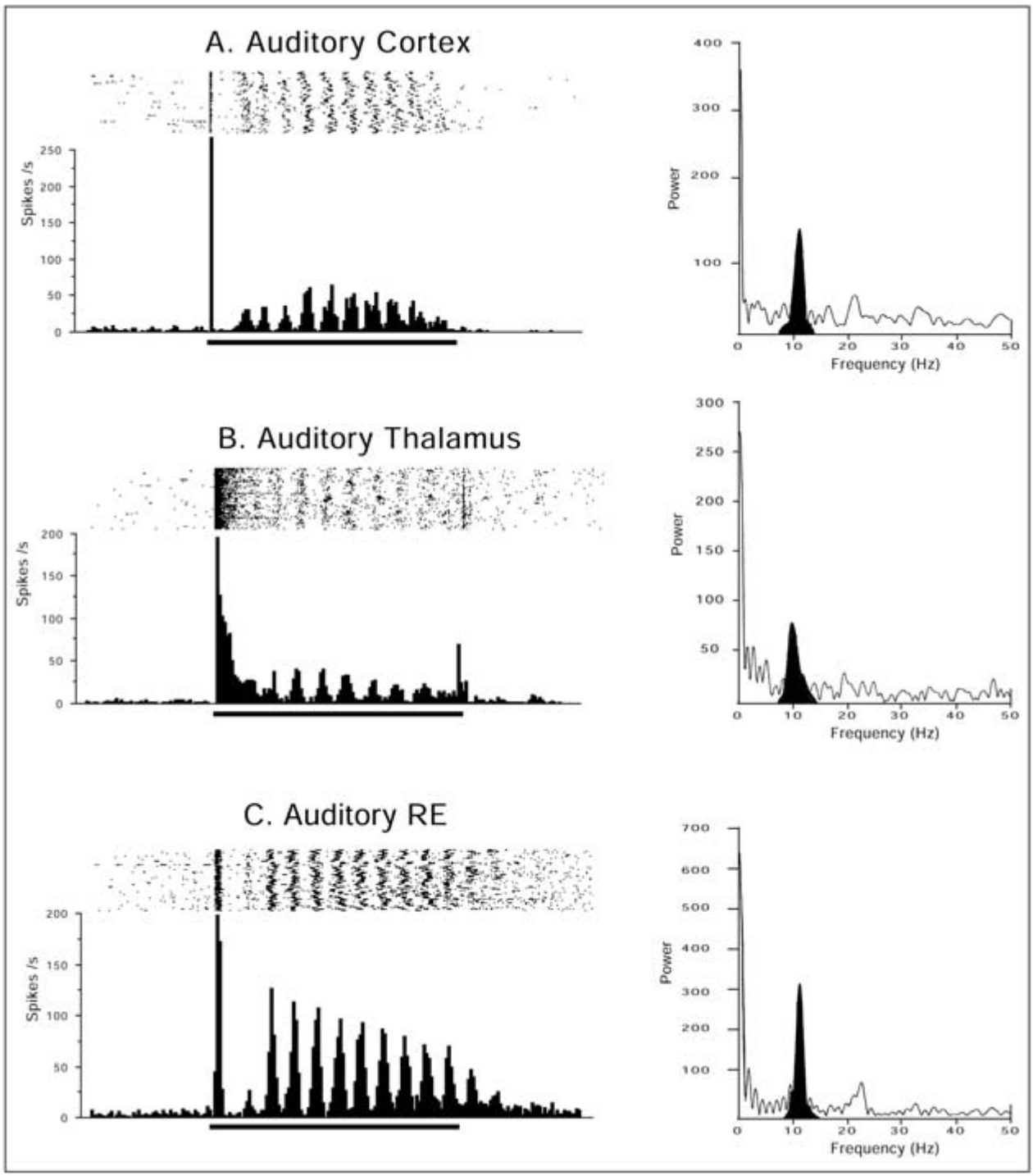

Fig. 2. Peri stimulus time histograms (PSTHs) and corresponding power spectra of "reliable" tone-evoked oscillations. Left: the PSTHs (10 ms bin size) present evoked responses averaged over 70 tone presentations. The horizontal bar under the histograms indicates the tone period (1 second). The first large peak in PSTHs is the "on" evoked response. It is followed by regular peaks which are separated by about 100 ms and occurred for several hundreds of milliseconds ((A) 800 ms, (B) 650 ms, (C) 1,100 ms). The rasters show that the oscillations result from a precise timing of the discharges after tone onset, which is very reproducible from one trial to another. Right: each corresponding power spectrum exhibits a clear main peak which gives the oscillation frequency ((A) 11.5 Hz, (B) 10 Hz, (C) 11 Hz). The area of the main peak delimited by a filter (black area) quantifies the oscillation strength (Ppeak).

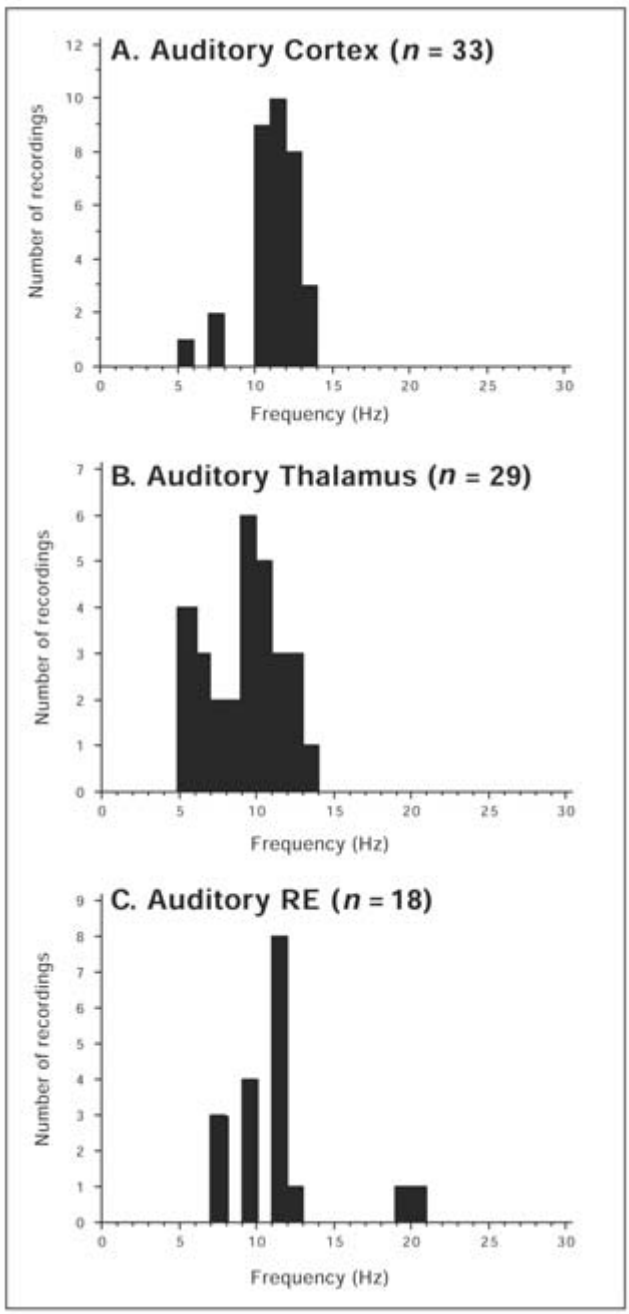

Fig. 3. Frequency range of stimulus-locked oscillations. Each histogram represents the distribution of the main peak frequency obtained from the power spectra (bin width = 1 Hz). (A) In auditory cortex, the frequency range of stimulus-locked oscillations is 5-13 Hz (mean frequency = 10.7 Hz, median = 11 Hz). (B) In auditory thalamus, the frequency range of stimulus-locked oscillations is 5-13 Hz (mean frequency = 8.8 Hz, median = 9 Hz). (C) In auditory RE, the frequency range of stimulus-locked oscillations is 7-20 Hz (mean frequency = 11.1 Hz, median = 11 Hz), but most of the oscillations are between 7 and 12 Hz.

cal loop and if they displayed the similar frequencies. Within a given structure, pairs of neighboring electrodes (200-300 μm apart) displayed simultaneous oscillations with similar frequency (± 0.5 Hz). In contrast, the probability of observing oscillations from pairs of electrodes located in two different structures was not above the chance level (see Table IV in Cotillon et al. 2000), in particular we never found simultaneous oscillations while recording in the two auditory cortices during binaural stimulation.

ORIGINS OF STIMULUS-LOCKED OSCILLATIONS

Surprisingly, whereas the origins of spontaneous oscillations have been the subject of a large literature (for reviews see McCormick and Bal 1997, Steriade et al. 1993, 1994), the origins of stimulus-evoked oscillations remain still largely unexplored. A set of experiments was carried out to determine the implication of each structure of the thalamo-cortical auditory system (auditory cortex, MGB and auditory RE) in the generation of stimulus-locked oscillations. In separate experiments, each structure was inactivated and its involvement in the generation of evoked oscillations was inferred from the evolution of oscillations in the two other structures. Inactivation was performed by injection, or local application, of muscimol (0.1 or 0.05 μl), a $GABA_A$ agonist which has been shown to have effects lasting up to several hours (Hikosaka and Wurtz 1985). As it is known (but not always recognized) that injection of inactivating agents could lead to uninterpretable results, special care were taken to make sure that the obtained results reflected physiological effects and not potential artifact because of extensive drug diffusion. For example, autoradiographic studies (involving both classical measures by optical density and also direct quantification of the radioactivity by a β-imager) were conducted to determine the extent of muscimol diffusion after injection of various volumes in the auditory reticular nucleus (Edeline et al. 2002). We also showed, in these pilot experiments, that abrupt decreases in spontaneous activity in the MGB were good indicators of drug diffusion at the recordings locus. These experiments, and modeling of muscimol diffusion (Dubois and Edeline, unpublished results), allowed to define periods during which observation of modifications in oscillatory patterns cannot be suspected to result from diffusion of the drug at the recording locus.

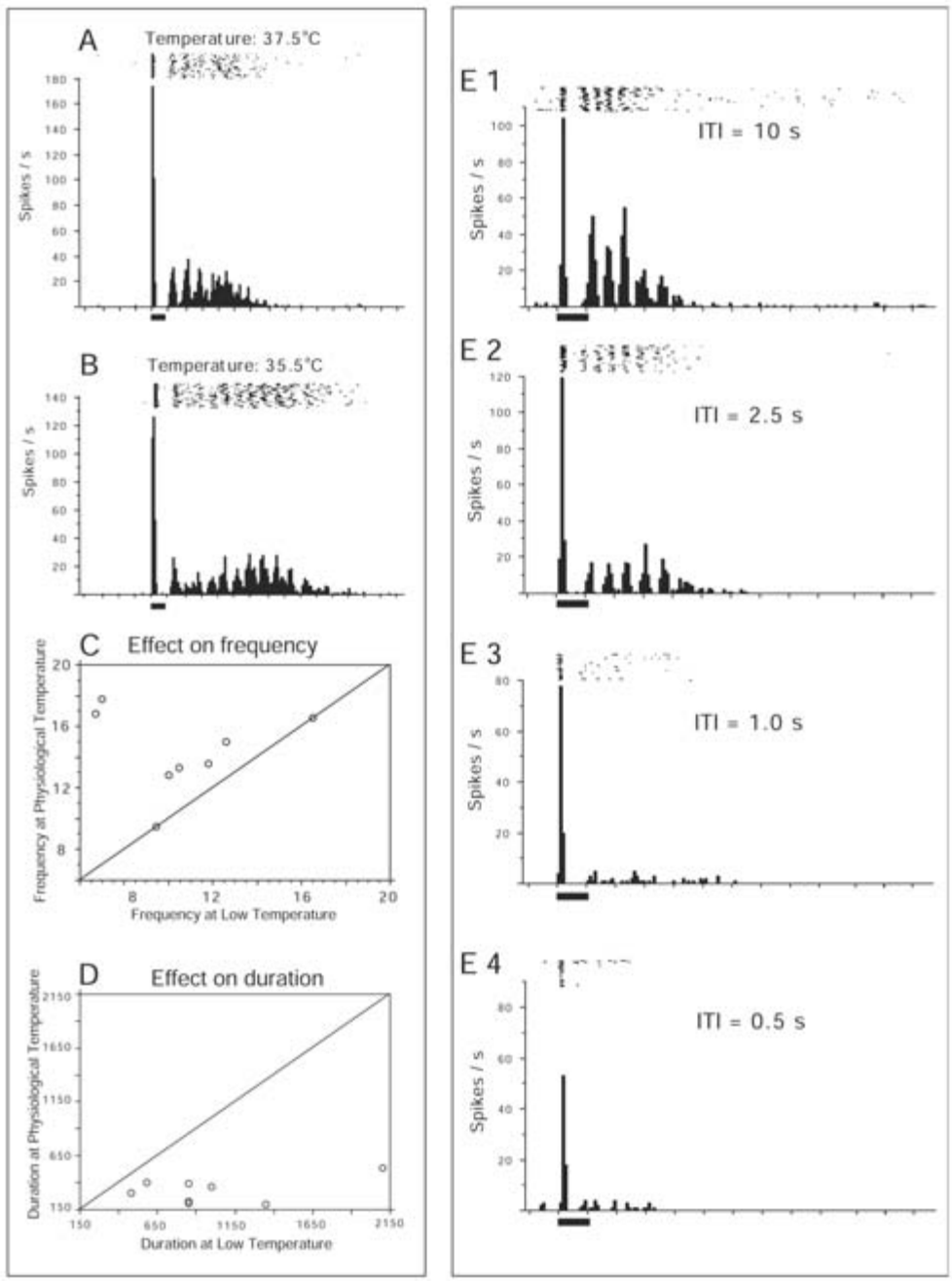

Fig. 4. Factors controlling the occurrence of stimulus-locked oscillations. (A) At 37.5°C, the PSTH presents only 3 peaks after the "on" evoked response (oscillation duration = 700 ms, oscillation frequency = 12.8 Hz). (B) At 35.5°C, PSTH presents at least 7 peaks. The duration of the oscillation is longer than in A (1,000 ms) and the oscillation frequency is lower (10 Hz). X axis ticks 100 ms. (C), (D) Scattergrams showing that lowering the animal's temperature affects the frequency and the duration of tone-evoked oscillations. For each recording ($n = 8$), acoustic stimuli were delivered with a 10 s inter-tone interval, both at physiological temperature (37-38°C) and at lower temperature (35-36°C). (C) Except for two recordings, the frequency of the oscillations was higher at physiological temperature than at lower temperature (t_7=2.61, P=0.03). (D) Each recording exhibits shorter oscillations duration at physiological temperature than at lower temperature (t_7=4.07, P=0.004). (E1-E4) PSTHs obtained in the auditory thalamus with different intertone intervals: 10 s, 2.5 s, 1 s, 0.5 s. For each ITI, the PSTHs (bin width = 10 ms) and the corresponding rasters were from 11 tone presentations. The horizontal bar under each histogram represents the tone period (100 ms). Both on the PSTHs and on the corresponding rasters, "reliable"oscillations can be observed for ITIs of 10 s and 2.5 s. Oscillatory patterns are no longer present for ITIs of 1 s and 0.5 s.

Once all ambiguous data were eliminated, the following set of results was obtained (for details see Cotillon and Edeline 2000). First, inactivation of the auditory sector of the reticular nucleus totally abolished evoked oscillations in the auditory thalamus and auditory cortex. Second, inactivation of auditory cortex did not affect the oscillatory patterns in the auditory RE. Third, inactivation of the auditory thalamus suppressed the oscillatory responses evoked in the auditory RE by electrical stimulation of auditory cortex (Fig. 5). Thus, we came to the conclusion that the stimulus-locked oscillations detected at three different levels of the thalamo-cortical loop indeed results from the interactions between thalamus and reticular nucleus. This singularity suggests that these low-frequency evoked oscillations share some functional properties with spontaneous spindle waves (see below for details).

EVOKED OSCILLATIONS IN DRUGGED AND UNDRUGGED ANIMALS

To the best of our knowledge, whatever the sensory modality, no study has ever tried to determine (and to quantify) to what extent oscillations obtained in a given state (e.g., anesthetized state) can also be observed in another state (unanesthetized and under a different anesthetic). We took advantage of the fact that our laboratory has long been using chronically implanted restrained animals to follow the oscillatory patterns obtained from the same recording sites under different natural states (waking, slow-wave sleep, paradoxical sleep) and under different drugs (pentobarbital, urethane and diazepam).

During several weeks, multiunit activity was collected from recording sites in auditory thalamus, auditory cortex and auditory RE. Data were analyzed only during unambiguous periods of waking (W), slow-wave sleep (SWS) or paradoxical sleep (PS). Several striking features emerged when comparing the data obtained in undrugged and drugged states. First, out of a total of 689 tests (i.e., groups of trials with an Inter-tone interval of 2.5 or 10 seconds), we could not detected a single case of stimulus-locked oscillations in undrugged conditions. In contrast, when the same recording sites were tested under pentobarbital, urethane or diazepam, evoked oscillations ranged from 24% to 66% depending on the conditions (see Table II in Cottillon-Williams and Edeline 2003). Figure 6 shows examples of stimulus-locked oscillations in auditory cortex and in auditory RE detected under pentobarbital, urethane or diazepam that were not detected in undrugged state. Second, analyzing the spike trains with autocorrelograms revealed that under the three drugs used in our study (pentobarbital, urethane and diazepam), the oscillatory patterns could be either stimulus-locked or non stimulus-locked (see Fig. 7A,B). In contrast, only non stimulus-locked were found during natural states of vigilance. Third, the non stimulus-locked oscillations obtained in undrugged state were exclusively observed during SWS, never during W and PS (see Fig. 7C).

Several interesting features also emerged from our analyses. Both in drugged and undrugged conditions, oscillations were present during periods of spontaneous activity in the same proportions as the proportions of non stimulus-locked oscillations (see Fig. 8A,B). When analyzing the relationships between spontaneous and evoked oscillations, we discovered that: (i) the power intensity of stimulus-locked and non stimulus-locked oscillations was always larger (Fig. 8C) than those of spontaneous oscillation; (ii) the power of stimulus-locked oscillations was larger than the one of non-stimulus locked oscillation; but (iii) frequency range of the 2 types of oscillations was similar to that of spontaneous oscillations (Fig. 8D). From these results, one can conclude that non stimulus-locked oscillations show characteristics that are quite similar to those exhibited by spontaneous oscillations: they can be detected in the same proportion of recordings, under the same physiological conditions (anesthesia and SWS) and exhibited the same frequency range. In drugged conditions, stimulus-locked oscillations are present in much higher proportions than non-stimulus locked oscillations. Their total absence in natural states (W, SWS and PS) indicates that they are unambiguously the signature of anesthetized or "drugged" states.

COMPARISONS WITH FINDINGS OBTAINED IN THE AUDITORY THALAMO-CORTICAL SYSTEM

As previously noted, tone-evoked oscillations in the low frequency range (about 10 Hz) have long been mentioned from single unit recordings in MGB (Aitkin et al. 1966, Galambos et al. 1952). Similar oscillations have been reported in the auditory sector of the reticular nucleus (Shosaku and Sumitomo 1983) and at the cortical level (Dinse et al. 1997, Maldonado and Gerstein 1996). Thus, the dominant frequency range observed here is

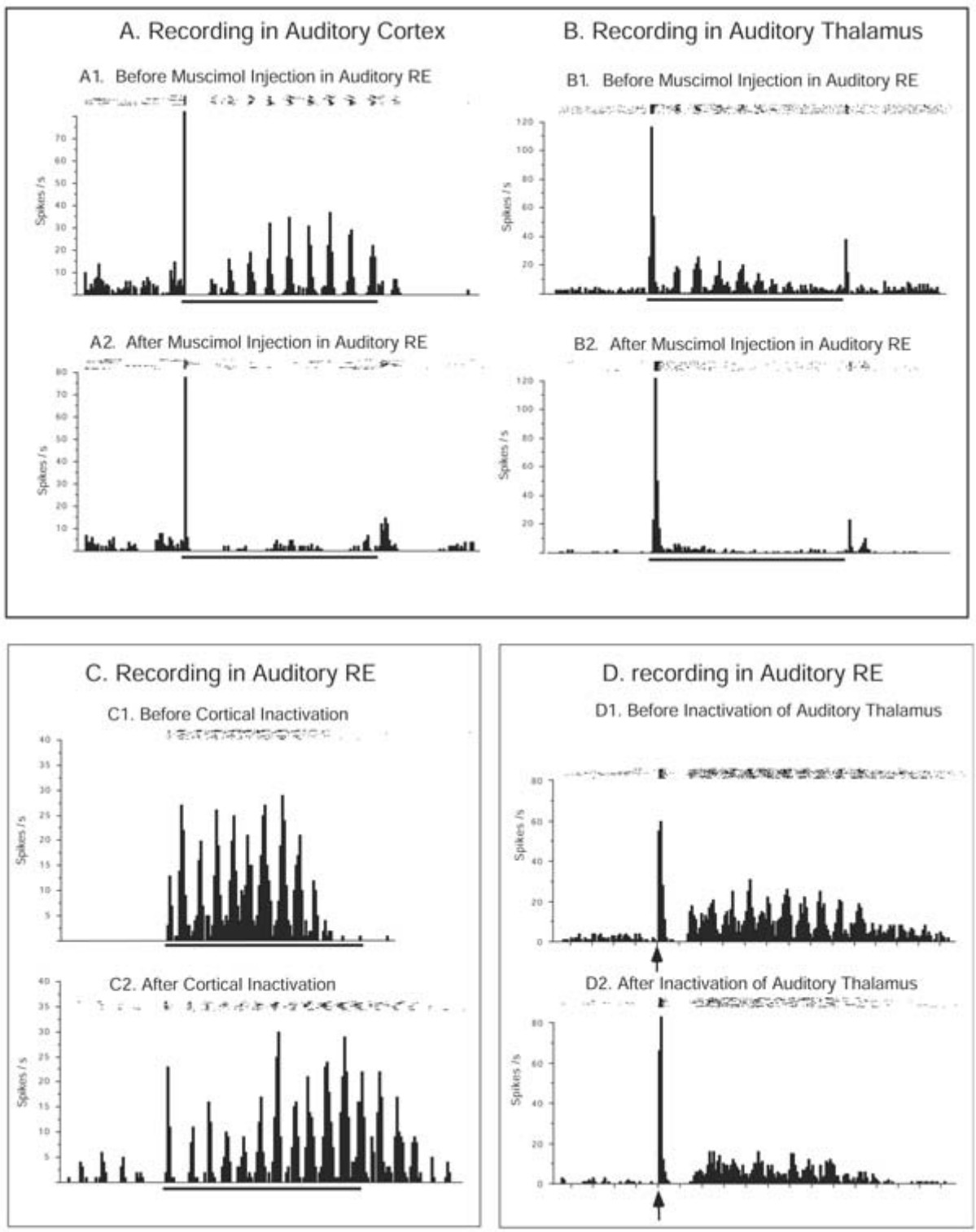

Fig. 5. Origins of stimulus-locked oscillations. (A), (B) Effect of auditory RE inactivation: in control situation, oscillations were detected from simultaneous recordings in auditory cortex and in auditory thalamus (A1, B1). After inactivation of the auditory RE by 0.05 µl of muscimol, the oscillations which normally follow the "on" phasic responses totally disappeared. (C) Effect of auditory cortex inactivation: oscillations, detected from recordings collected in the auditory RE, were still present after inactivation of auditory cortex. (D) Effect of auditory thalamus inactivation: oscillations, triggered in the auditory RE by cortical stimulation disappeared after inactivation of auditory thalamus.

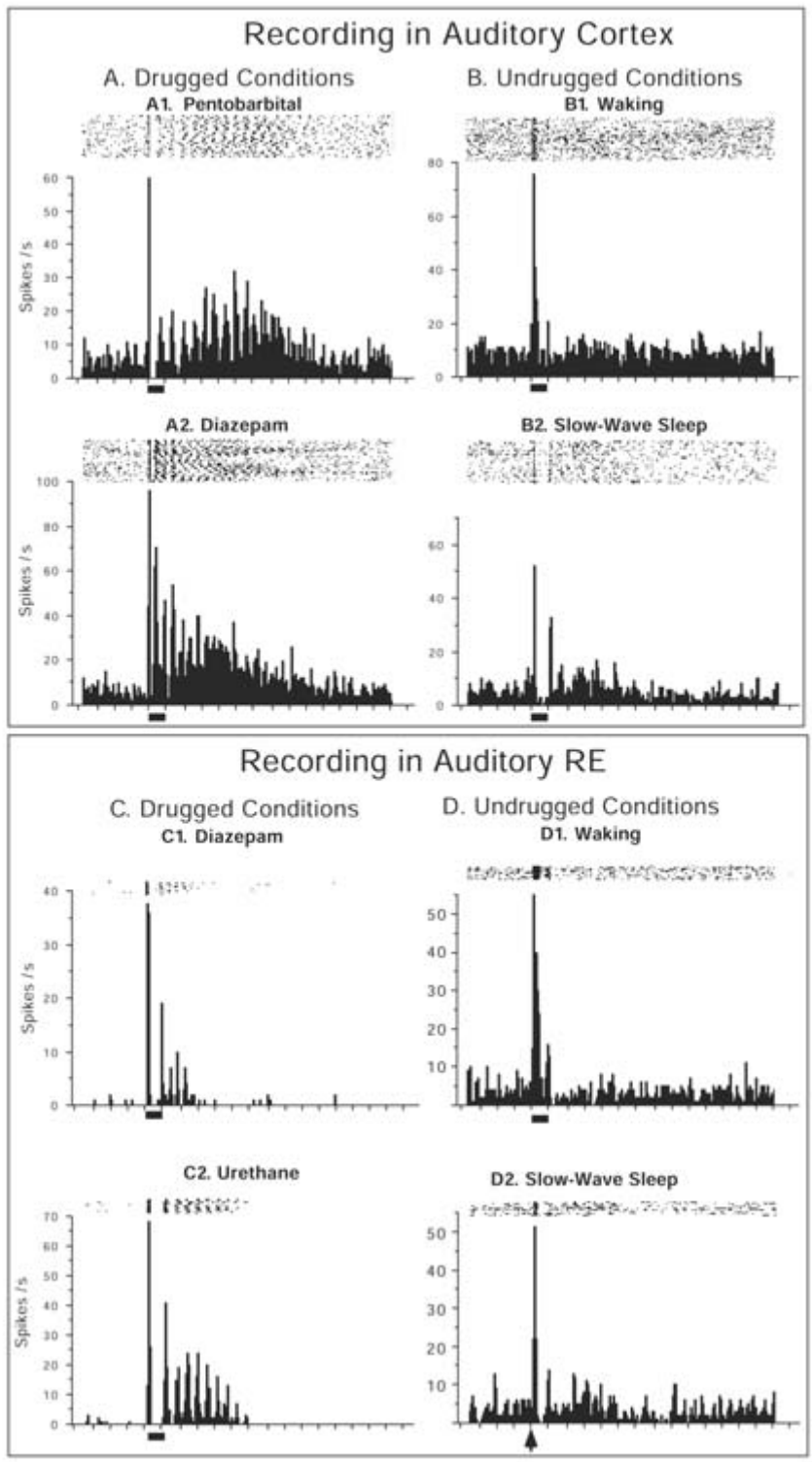

Fig. 6. Comparison of stimulus-locked oscillations in drugged and undrugged conditions. (A), (B) For this recording obtained in auditory cortex, stimulus-locked oscillations were clearly observed both under pentobarbital anesthesia and under large doses of diazepam (A1, A2). In contrast, when the same recording site was tested during waking or slow-wave sleep, no oscillation could be detected (B1, B2). (C), (D) For this recording obtained in auditory RE, stimulus-locked oscillations were clearly observed both under pentobarbital and urethane anesthesia (C1), (C2). In contrast, when the same recording site was tested during waking or slow-wave sleep, no oscillation could be detected (D1, D2).

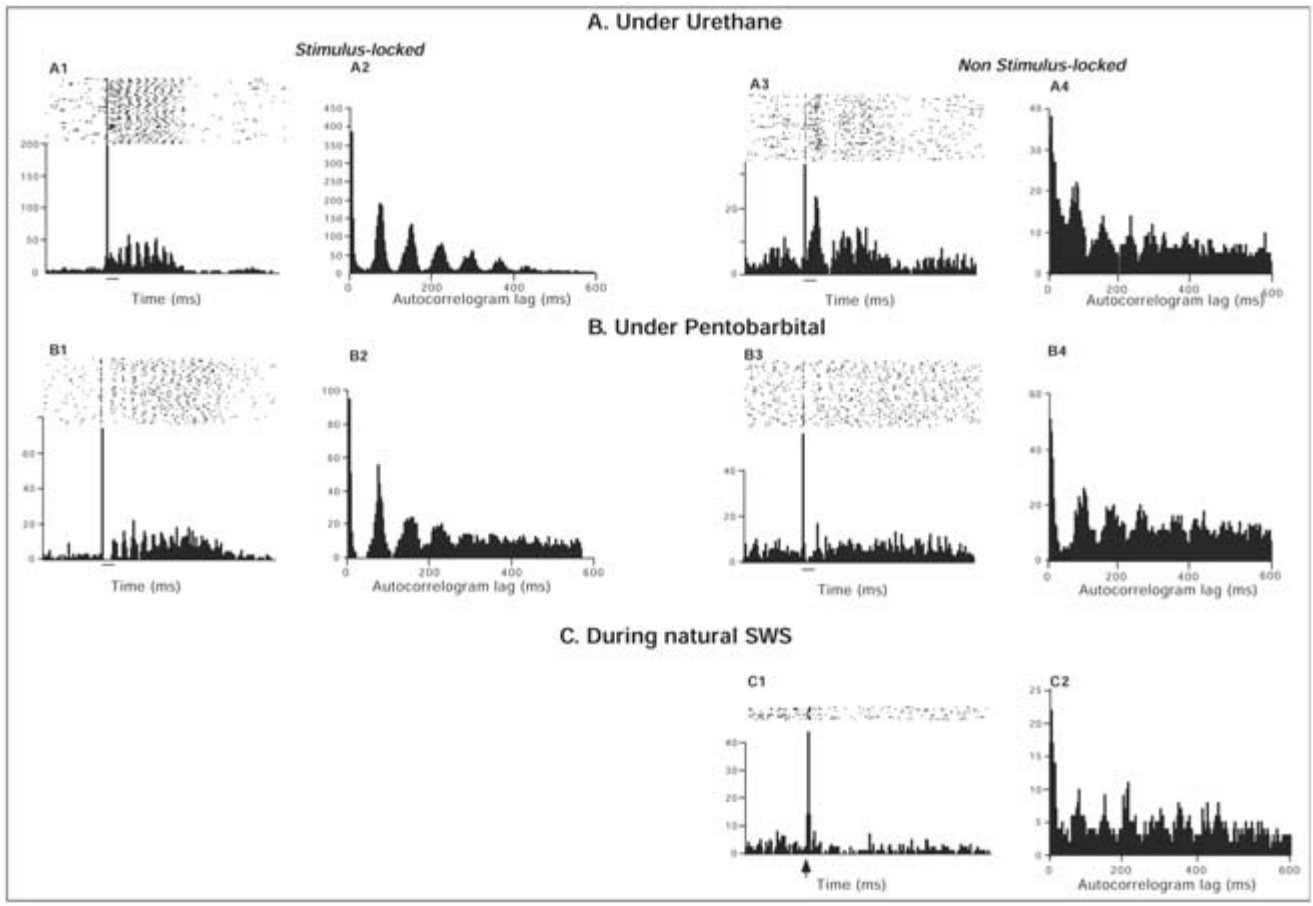

Fig. 7. Non stimulus-locked oscillations were detected in drugged conditions and during SWS. In drugged conditions (A), (B) both stimulus-locked oscillations (A1, B1) and non stimulus-locked oscillations (A3, B3) could be detected. During SWS, only non stimulus-locked oscillations were triggered at tone presentation (C1, C2). (Modified from Cotillon-Williams and Edeline 2003)

largely in agreement with previous studies performed in anesthetized animals.

In contrast with the results described here with multi-unit recordings, high frequency oscillations have been described in the auditory cortex of halothane anesthetized rats using LFP recordings (Barth and MacDonald 1996, Franowicz and Barth 1995, MacDonald and Barth 1995). However, it is crucial to mention that 40 Hz oscillations were detected only with trains of clicks presented at 40 Hz (Franowicz and Barth 1995), but not with single clicks. In fact, presentation of a single click suppressed the spontaneous 40 Hz oscillations which re-appeared few hundreds of milliseconds later (Franowicz and Barth 1995, MacDonald and Barth 1995). Observation of oscillations in LFP does not guarantee their presence in multi-unit or single-unit recordings. For example, in the visual cortex Gray and Singer (1989) described 97% of oscillations based on LFP recordings, but only 47% based on multi-unit recordings. Nonetheless, in auditory cortex Brosch et al. (2002) reported about the same percentage (76% and 60%) of high frequency oscillations using LFP and multi-unit recordings in ketamine anesthetized monkeys. These oscillations differ from those observed in rat by their frequency (40 Hz in rat vs. 45-80 Hz in monkey) and their latency (350 ms in rat vs. 100 ms in monkey). Obviously, methodological factors have to be considered when comparing the results obtained among studies. For example, Barth and colleagues as well as Brosch et al. (2002) used anesthetic agents that were not utilized in our experiments (halothane and ketamine). Also, it is important to remind that the criteria used to determine what is an oscillatory recording can lead to different results (see Young et al. 1992 for discussion).

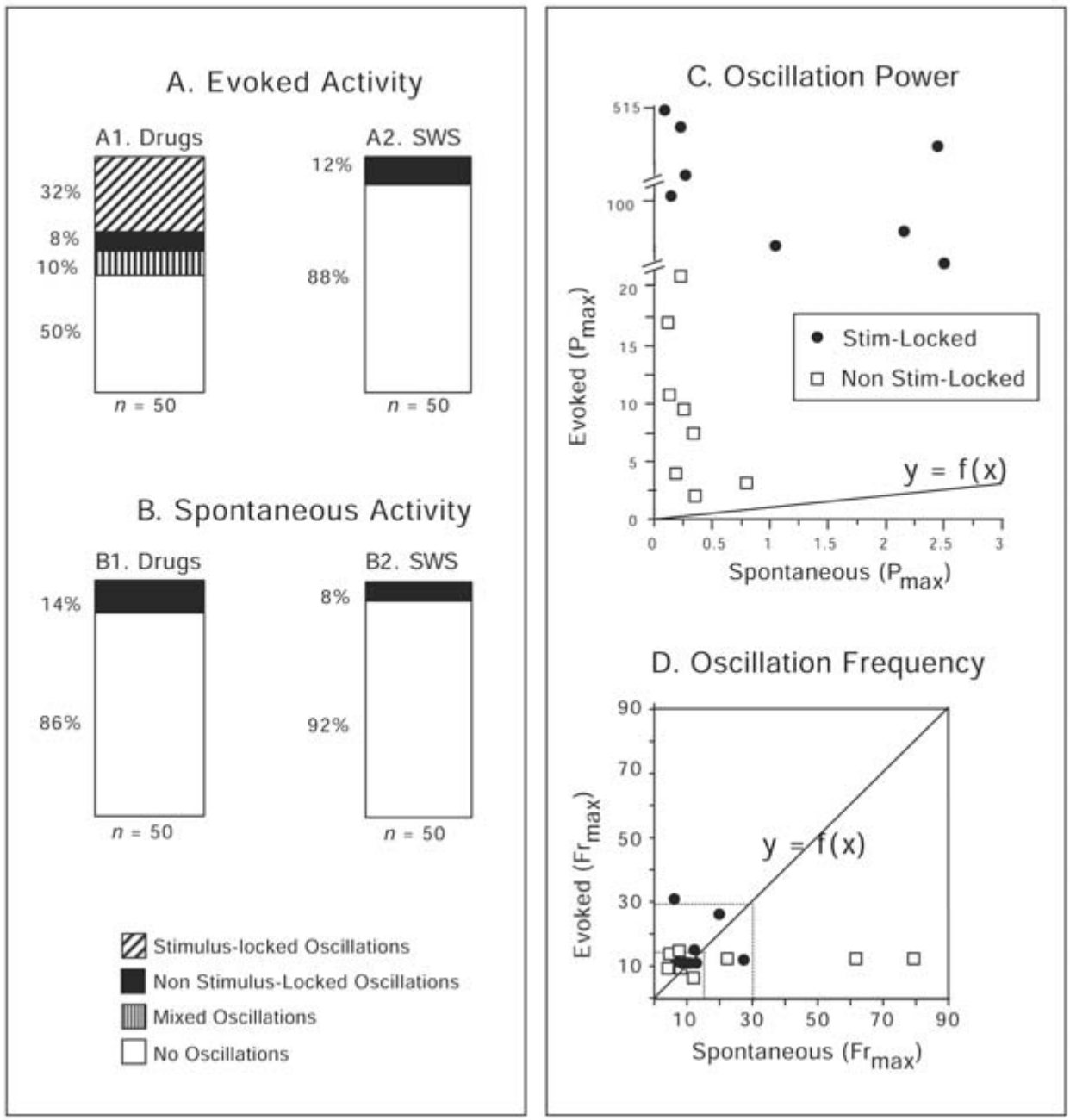

Fig. 8. Comparison of spontaneous and evoked oscillations obtained in drugged and undrugged conditions. (A), (B) Proportions of the different types of oscillations during evoked (A) and spontaneous activity (B) under drug conditions (A1, B1) and during SWS (A2, B2). (A) During evoked activity, there was a higher proportion of stimulus-locked than of non stimulus-locked oscillations under drugged conditions (A1), and only non-stimulus-locked oscillations were present during SWS (A2). Mixed "groups of trials" were composed of "trials" showing stimulus-locked and of "trials" showing non-stimulus locked oscillations. (B) During spontaneous activity, the percentage of oscillations was similar under drug conditions and SWS. (C), (D) Scattergrams illustrating the relationships between the characteristics of spontaneous and evoked oscillations under drug. (C) The power of spontaneous oscillations was systematically lower than the power of evoked oscillation. (D) The frequency of spontaneous oscillations was usually very similar to the frequency of evoked oscillations. In two cases, the spontaneous oscillations were of much higher frequency than the evoked oscillations, the reverse occurred only in one case. Both in (C) and (D), the line indicates the y = f(x) relation; the black dots represent the stimulus locked oscillation and the open squares represent the non stimulus-locked oscillations.

COMPARISONS WITH STUDIES PERFORMED IN OTHER SENSORY SYSTEMS OF ANESTHETIZED ANIMALS

Initially, most of the studies concerning stimulus-evoked oscillations have been performed in the visual cortex of anesthetized animals (for reviews see Eckhorn 2000, Engel et al. 1992, Frégnac et al. 1994, Singer 1990). They have mainly reported non stimulus-locked oscillations in the gamma range (30-90 Hz). Nonetheless, low frequency oscillations were reported in a few studies in anesthetized cat (stimulus-locked: Dinse et al. 1997, non stimulus-locked: Bringuier et al. 1992, 1997), but not in monkey. In fact, in the cat visual cortex, slow movements of visual stimuli can induce changes from low to high frequency (Kruse and Eckhorn 1996). In primate visual cortex, low frequency oscillations were found to be prominent during spontaneous activity, but they were usually replaced by higher frequencies at stimulus presentation (Eckhorn 2000, Juergens et al. 1999, Schanze and Eckhorn 1997). Actually, the proportion of low frequency oscillations in the visual cortex of cat or monkey might have been underestimated. Indeed, most of these studies were interested in high frequency oscillations and not in low frequency oscillations. In fact, an important degree of temporal overlap exists between the different frequency ranges: when power bispectra were used, 70% of the oscillatory events were found to occur simultaneously in the different frequency ranges (Schanze and Eckhorn 1997). Again, the anesthetic conditions could participate to the differences in oscillatory patterns (see Frégnac et al. 1994): in the cat visual cortex, all the studies which reported high frequency oscillations have used NO/O_2 anesthesia, whereas lower oscillations were obtained under alfathesine anesthesia (Bringuier et al. 1992, 1997). However, low frequency (stimulus-locked) oscillations have also been observed in cat anesthetized with NO/O_2 (Dinse et al. 1997).

COMPARISONS WITH PREVIOUS FINDINGS IN UNANESTHETIZED ANIMALS

Conflicting results have been obtained from awake subjects in various sensory modalities. In the cat visual cortex, all the studies using LFP, multi-unit and single-unit recordings have described high frequency oscillations (Gray and Viana Di Prisco 1997, Kruse and Eckhorn 1996, Molotchnikoff and Shumikhina 1996, 2000). Gray and Viana Di Prisco (1997) even mentioned a higher percent of oscillations in unanesthetized cat compared to anesthetized one (26.4% *versus* 16.9%). The situation is different in human and non-human primates. In monkey, some studies have found a high percentage of oscillations in the area IT (61% in Friedman-Hill et al. 2000, 71% in Eckhorn et al. 1993), whereas others did not find any (Tovée and Roll 1992, Young et al. 1992). A similar discrepancy also exists in the area MT: Kreiter and Singer (1992) reported 58% of oscillations, whereas Bair et al. (1994) did not detect any. In human, oscillations in the gamma range have often been reported on EEG recordings (Lachaux et al. 2000, Lutzenberger et al. 1995, Tallon et al. 1995, Tallon-Baudry et al. 1996, 1997, 1998). Nevertheless, cautions have to be exercised: when EEG power spectra were compared in human and monkey, visual stimulation elicited an increase in gamma range in the monkey EEG, but not in the human EEG (Juergens et al. 1999).

In the somatosensory cortex, spontaneous 30 Hz oscillations have been observed from single-unit recordings in monkeys performing a reaction-time task, but these oscillations disappeared at stimulus presentation (Ahissar and Vaadia 1990). In rat, LFP recordings have revealed an opposite pattern: spontaneous oscillations were not observed, but moving the vibrissae induced oscillations (Jones and Barth 1997).

In the auditory system, the only study looking for oscillations in awake animals was performed in the auditory cortex of mustached bat (Horikawa et al. 1994). Multi-unit and single-unit recordings did not reveal any oscillation on PSTHs and only 2% on autocorrelograms. Nevertheless, this study used a one second ITI, an interval that we found to be too short to evoke low-frequency oscillations in anesthetized rats (Cotillon et al. 2000). In human, oscillations of evoked potentials (mostly in the gamma range) have been reported at stimulus presentation (Galambos et al. 1981, Jacobson and Fitzgerald 1997, Yordanova and Kolev 1997, Yordanova et al. 1997).

In fact, a crucial factor that could explain differences between studies is to what extent the awake animal is engaged in a challenging behavioral task. In several recent studies some laboratories have stressed the fact that the attentional demand can enhance the beta activity recorded from the lateral geniculate and visual cortex (Bekisz and Wróbel 1993, 2003, Wróbel et al. 1994, for review see Wróbel 2000). Although, these results are

certainly among the most promising to link rhythmic activities and behavioral performance, they were obtained with LFP recordings not with spiking activities. In fact, when multiunit activity (MUA) and LFP were simultaneously recorded, correlations with the animal perceptive performance were found with LFP recordings not with MUA recordings (Gail et al. 2004, Woelbern et al. 2002).

POTENTIAL MECHANISMS

Important similarities were noted between low frequency stimulus-locked oscillations and spontaneous spindles: (i) they are in the same frequency range (5-15 Hz); (ii) they occur with an inter-tone (or an inter-event) interval of at least 2 seconds (Bal and McCormick 1996, Cotillon et al. 2000); (iii) they both seem to originate from interactions between thalamic relay cells and thalamic reticular cells, without a dominant contribution of the cortical level (Bal et al. 1995, Cotillon and Edeline 2000). Nonetheless, an important difference exists between these oscillations and the spindles. Stimulus-locked oscillations were rarely synchronized between the thalamic and cortical levels, as well as between the two hemispheres (Cotillon et al. 2000), whereas spindles are highly synchronized over the cortex and the thalamus (Contreras et al. 1997a), as well as between the two hemispheres (Contreras and Steriade 1996). The involvement of cortical activity was shown to explain the long-range synchronization of spindles (Contreras et al. 1996, 1997b, Destexhe et al. 1999), the absence of cortical involvement in tone-evoked oscillations could explain their lack of long-range synchronization.

Stimulus-locked oscillations were detected under different drugs acting on GABAergic receptors (pentobarbital, valium) or not (urethane). We previously suggested that this type of oscillations requires important levels of hyperpolarization (Cotillon-Williams and Edeline 2003). It is well-known that the presence of bursts, underlying the low frequency oscillations, requires an hyperpolarized state of thalamic cells (Contreras and Steriade 1996, Curro-Dossi et al. 1991, 1992, Deschęnes et al. 1984, Lo et al. 1991, Lu et al. 1992). During SWS, only modest hyperpolarizations were observed at the thalamic level (only 4 mV in the cat LGd, see Hirsch et al. 1983). One can argue that this moderate level of hyperpolarization is, however, enough for the presence of a small percentage of spontaneous spindles. Again, the cortical waves of activity, which synchronize massively the discharge of both RE and thalamic cells during spindles, can play a key role: by acting more strongly onto RE cells than onto thalamic cells (Golshani et al. 2001), spindles can be triggered by cortical activity even with relatively modest levels of hyperpolarization of thalamic cells. In contrast, sensory stimuli: (i) activates thalamic cells before the RE cells; and (ii) only activates a small population of thalamic cells (those responding to a particular frequency or to click presentation). Thus, because of the small size of the cell populations and because the triggering event starts by an excitation of thalamic cells (and not by an excitation of RE cells), the thalamo-reticular network probably needs to be more strongly hyperpolarized for the occurrence of stimulus-locked oscillations than for the spontaneous spindles.

As the spontaneous spindles, the non stimulus-locked oscillations might require only moderate levels of hyperpolarization, and as a consequence they were detected during SWS. In line with this view, the non stimulus-locked oscillations were found in the same proportion than spontaneous oscillations both in SWS and in drugged conditions. They were in the same frequency range (5-15 Hz), and, as spontaneous oscillations, they were observed in similar proportions in SWS and under drugs. Note that in drugged or undrugged conditions spontaneous and non stimulus-locked oscillations were present at a lower percentage and with a lower power than the stimulus-locked oscillations.

FUNCTIONAL IMPLICATIONS

According to this scheme, the lack of oscillations during waking and paradoxical sleep is simply the consequence of a more depolarized level during these states of vigilance. Obviously, the large literature that has involved the oscillations in cognitive functions (particularly in the visual system) will argue that the lack of oscillations during waking reflects the absence of an attentional demand, and that training the animals in behavioral tasks can reveal a higher proportion of oscillations. However, an alternative view is that the presence of evoked oscillations might not be an advantage for processing auditory information, and might even set the temporal limits of auditory perception. For example, in anesthetized cats, the ability of neurons to follow a train of clicks was inversely correlated with the duration of spontaneous oscillations (Kenmochi and Eggermont 1997). Also, a relationship was found between the fre-

quency of these oscillations and the best modulation frequency of units in the auditory cortex of anesthetized rats (Gaese and Ostwald 1995). Thus, the lack of oscillations in awake animals might, in fact, increase the temporal performance of auditory neurons and should allow detection of higher periodicity in complex stimuli. Because neuronal oscillations have often been viewed as a way to synchronize neuronal populations involved in perceptive processes, the present hypothesis clearly challenges the notion that visual perception and auditory perception rely on common mechanisms. By essence, auditory perception is temporal, and the need to rapidly respond to changes in the auditory stream is probably more crucial than in the visual system. Thus, the absence of oscillations in awake animals might allow each neuron to process the auditory inputs independently of its neighbors, and should allow a fine grain analysis of auditory messages.

Finally, we would like to point out that it is probably crucial to distinguish the rhythmic activities detected from LFP recordings from the spiking activity (MUA or single unit) analyzed at the same recording sites. We should keep in mind that neurons transmit information by action potentials. Detecting oscillations from LFP recordings does not mean that the information is transmitted *via* these oscillations, in particular when neuronal discharges do not exhibit rhythmic activity. Thus, the presence of oscillatory events, and their relations with the animal perception, might also be a function of whether or not the inputs reaching in a given brain region need to be transformed into neuronal discharges to contribute for the animal's immediate performance.

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REFERENCES

Ahissar E, Vaadia E (1990) Oscillatory activity of single unit in a somatosensory cortex of an awake monkey and their possible role in texture analysis. Proc Natl Acad Sci U S A 87: 8935-8939.

Aitkin LM, Dunlop CW, Webster WR (1966) Click-evoked response patterns of single units in the medial geniculate body of the cat. J Neurophysiol 29: 109-123.

Bair W, Koch C, Newsome W, Britten K (1994) Power spectrum analysis of bursting cells in area MT in the behaving monkey. J Neurosci 14: 2870-92.

Bal T, McCormick DA (1996) What stops synchronized thalamocortical oscillation. Neuron 17: 297-308.

Bal T, von Krosigk M, McCormick DA (1995) Role of the ferret perigeniculate nucleus in the generation of synchronized oscillations in vitro. J Physiol Lond 483: 665-685.

Barth DS, MacDonald KD (1996) Thalamic modulation of high-frequency oscillating potentials in auditory cortex. Nature 383: 78-81.

Bekisz M, Wróbel A (1993) 20 Hz rhythm of activity in visual system of perceiving cat. Acta Neurobiol Exp (Wars) 53: 175-82.

Bekisz M, Wróbel A (2003) Attention-dependent coupling between beta activities recorded in the cat's thalamic and cortical representations of the central visual field. Eur J Neurosci 17: 421-426.

Bordi F, Ledoux J (1994) Response properties of single units in areas of rat auditory thalamus that project to the amygdala. I. Acoustic discharge patterns and frequency receptive fields. Exp Brain Res 98: 261-274.

Bringuier V, Frégnac Y, Debanne D, Shulz D, Baranyi A (1992) Synaptic origin of rythmic visually evoked activity in kitten area 17 neurones. Neuroreport 3: 1065-1068.

Bringuier V, Frégnac Y, Baranyi A, Debanne D, Shulz D (1997) Synaptic origin and stimulus dependency of neuronal oscillatory activity in the primary visual cortex of the cat. J Physiol Lond 500: 751-774.

Brosch M, Budinger E, Scheich H (2002) Stimulus-related gamma oscillations in primate auditory cortex. J Neurophysiol 87: 2715-2725.

Contreras D, Steriade M (1996) Spindle oscillation in cats: the role of corticothalamic feedback in a thalamically generated rhythm. J Physiol Lond 490: 159-179.

Contreras D, Timofeev I, Steriade M (1996) Mechanisms of long-lasting hyperpolarizations underlying slow sleep oscillations in cat corticothalamic networks. J Physiol Lond 494: 251-264.

Contreras D, Destexhe A, Sejnowski TJ, Steriade M (1997a) Spatiotemporal patterns of spindle oscillations in cortex and thalamus. J Neurosci 17: 1179-1196.

Contreras D, Destexhe A, Steriade M (1997b) Spindle oscillations during cortical spreading depression in naturally sleeping cats. Neuroscience 77: 933-936.

Cotillon N, Edeline J-M (2000) Tone-evoked oscillations in the rat auditory cortex result from interactions between the thalamus and reticular nucleus. Eur J Neurosci 12: 3637-3650.

Cotillon N, Nafati M, Edeline J-M (2000) Characteristics of reliable tone-evoked oscillations in the rat thalamo-cortical auditory system. Hear Res 142: 113-130.

Cotillon-Williams N, Edeline J-M (2003) Evoked oscillations in the thalamo-cortical auditory system are present in anesthetized but not in unanesthetized rats. J Neurophysiol 89: 1968-1984.

Curro-Dossi R, Pare D, Steriade M (1991) Short-lasting nicotinic and long-lasting muscarinic depolarizing responses of thalamocortical neurons to stimulation of mesopontine cholinergic nuclei. J Neurophysiol 65: 393-406.

Curro-Dossi R, Nunez A, Steriade M (1992) Electrophysiology of a slow (0.5-4Hz) intrinsic oscillation of cat thalamocortical neurones in vivo. J Physiol Lond 447: 215-234.

Deschênes M, Paradis M, Roy JP, Steriade M (1984) Electrophysiology of neurons of lateral thalamic nuclei in cat: resting properties and burst discharges. J Neurophysiol 51: 1196-1219.

Destexhe A, Contreras D, Steriade M (1999) Neocortical excitability controls the coherence of thalamic-generated oscillations through corticothalamic feedback. Neuroscience 92: 427-443.

Dinse HR, Krüger K, Akhavan AC, Spengler F, Schöner G, Schreiner CE (1997) Low-frequency oscillations of visual, auditory and somatosensory cortical neurons evoked by sensory stimulation. Int J Psychophysiol 26: 205-227.

Eckhorn R (2000) Cortical synchronization suggests neural principles of visual feature grouping. Acta Neurobiol Exp (Wars) 60: 261-269.

Eckhorn R, Obermueller A (1993) Single neurons are differently involved in stimulus-specific oscillations in cat visual cortex. Exp Brain Res 95: 177-182.

Eckhorn R, Bauer R, Jordan W, Brosch M, Kruse W, Munk M, Reitboeck HJ (1988) Coherent oscillations: a mechanism of feature linking in the visual cortex? Multiple electrode and correlation analysis in the cat. Biol Cybern 60: 121-130.

Eckhorn R, Frien A, Bauer R, Woelbern T, Kehr H (1993) High frequency (60-90 Hz) oscillations in primary visual cortex of awake monkey. Neuroreport. 4: 243-246.

Edeline J-M, Hars B, Hennevin E, Cotillon N (2002) Muscimol diffusion after intracerebral microinjections: a re-evaluation based on electrophysiological and autoradiographic quantifications. Neurobiol Learn Mem 78: 100-124.

Eggermont JJ (1998) Is there a neural code? Neurosci Biobehav Rev 22: 355-370.

Eggermont JJ (1992) Stimulus induced and spontaneous rhythmic firing of single units in cat primary auditory cortex. Hear Res 61: 1-11.

Eggermont JJ (2001) Between sound and perception:reviewing the search for a neural code. Hear Res 157: 1-42.

Engel AK, König P, Gray CM, Singer W (1990) Stimulus-dependent neuronal oscillations in cat visual cortex: inter-columnar interaction as determined by cross-correlation analysis. Eur J Neurosci2: 588-606.

Engel AK, König P, Kreiter AK, Singer W (1991a) Interhemispheric synchronisation of oscillatory neuronal responses in cat visual cortex. Science 252: 1177-1179.

Engel AK, Kreiter AK, König P, Singer W (1991b) Synchronization of oscillatory neuronal responses between striate and extrastriate visual cortical areas of the cat. Proc Natl Acad Sci U S A 88: 6048-6052.

Engel AK, Konig P, Kreiter AK, Schillen TB, Singer W (1992) Temporal coding in the visual cortex: new vistas on integration in the nervous system. Trends Neurosci 15: 218-226.

Engel AK, Fries P, Singer W (2001) Dynamic predictions: oscillations and synchrony in top-down processing. Nat Rev Neurosci 2: 704-716.

Erulkar SD, Rose JE, and Davies PW (1956) Single unit activity in the auditory cortex of the cat. Bull John Hopkins Hosp 99: 55-86.

Franowicz MN, Barth DS (1995) Comparison of evoked potentials and high-frequency (gamma-band) oscillating potentials in rat auditory cortex. J Neurophysiol.74: 96-112.

Freeman WJ, van Dijk BW(1987) Spatial patterns of visual cortical fast EEG during conditioned reflex in a rhesus monkey. Brain Res 422: 267-276.

Frégnac Y, Bringuier V, Baranyi A (1994) Oscillatory neuronal activity in the visual cortex: a critical re-evaluation. In: Temporal coding in the brain (Eds. G. Buzsaki, R. Llinas, W. Singer, A. Berthoz and Y. Christen). Springer-Verlag, Berlin, p. 81-102.

Friedman-Hill S, Maldonado PE, Gray CM (2000) Dynamics of striate cortical activity in the alert macaque: I. Incidence and stimulus-dependence of gamma-band neuronal oscillations. Cereb Cortex 10: 1105-1116.

Gaese BH, Ostwald J (1995) Temporal coding of amplitude and frequency modulation in the rat auditory cortex. Eur J Neurosci 7: 438-450.

Gail A, Brinksmeyer HJ, Eckhorn R (2004) Perception-related modulations of local field potential power and coherence in primary visual cortex of awake monkey during binocular rivalry. Cereb Cortex 14: 300-313.

Galambos R, Rose JE, Bromiley RB, Hughes JR (1952) Microelectrode studies on medial geniculate body of cat. II.Response to clicks. J Neurophysiol 15: 359-380.

Galambos R, Makeig S, Talmachoff PJ (1981) A 40-Hz auditory potential recorded from the human scalp. Proc Natl Acad Sci U S A 78: 2643-2647.

Ghose GM, Freeman RD (1992) Oscillatory discharge in the visual system: does it have a functional role? J Neurophysiol 68: 1558-1574.

Golshani P, Liu X B, Jones EG (2001) Differences in quantal amplitude reflect GluR4- subunit number at corticothalamic synapses on two populations of thalamic neurons, Proc Natl Acad Sci U S A 98: 4172-4177.

Gray CM, Singer W (1989) Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc Natl Acad Sci U S A 86: 1698-1702.

Gray CM, Viana Di Prisco G (1997) Stimulus-dependent neuronal oscillations and local synchronization in striate cortex of the alert cat. J Neurosci 17: 3239-3253.

Gray CM, Konig P, Engel AK, Singer W (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar synchronisation which reflects global stimulus properties. Nature 338: 334-337.

Gray CM, Engel AK, König P, Singer W (1990) Stimulus-dependent neuronal oscillations in cat visual cortex: Receptive-field properties and feature dependence. Eur J Neurosci 2: 607-619.

Hikosaka O, Wurtz RH (1985) Modification of saccadic eye movements by GABA-related substances. I. Effects of Muscimol and bicuculline in monkey superior colliculus. J Neurophysiol 53: 266-291.

Hind JE, Rose RE, Davies PW, Woolsey CN, Benjamin RM, Welkes WS, Thompson RF (1960) Unit activity in auditory cortex. In: Neural mechanisms of the auditory and vestibular systems (Eds. G.L. Rasmussen and W.F. Windle). Charles C Thomas, Springfield, p. 201-210.

Hirsch JC, Fourment A, Marc ME (1983) Sleep-related variations of membrane potential in the lateral geniculate body relay neurons of the cat. Brain Res 259: 308-312.

Horikawa J, Tanahashi A, Suga N (1994) After-discharges in the auditory cortex of the mustached bat: No oscillatory discharges for binding auditory information. Hear Res 76: 45-52.

Hubel DH, Wiesel TN (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J Physiol 160: 106-154.

Hubel DH, Wiesel TN (1963) Shape and arrangement of columns in cat's striate cortex. J Physiol 165: 559-568.

Jacobson GP, Fitzgerald MB (1997) Auditory evoked gamma band potential in normal subjects. J Am Acad Audiol 8: 44-52.

Jones MS, Barth DS (1997) Sensory-evoked high-frequency (gamma-band) oscillating potentials in somatosensory cortex of the unanesthetized rat. Brain Res 768: 167-176.

Juergens E, Guettler A, Eckhorn R (1999) Visual stimulation elicits locked and induced gamma oscillations in monkey intracortical- and EEG-potentials, but not in human EEG. Exp Brain Res 129: 247-259.

Kaas JH (1997) Topographic maps are fundamental to sensory processing. Brain Res Bull 44: 107-112.

Kenmochi M, Eggermont JJ (1997) Autonomous cortical rhythms affect temporal modulation transfer functions, Neuroreport 8: 1589-1593.

Kreiter AK, Singer W (1992) Oscillatory neuronal responses in the visual cortex of the awake macaque monkey. Eur J Neurosci 4: 369-375.

Kreiter AK, Singer W (1996) On the role of neural synchroni-y in the primate visual cortex. In: Brain Theory-Biological basis and computational principles. (Eds. A. Aertsen and V. Braitenberg), Elsevier, Amsterdam, p. 201-227.

Kruse W, Eckhorn R (1996) Inhibition of sustained gamma oscillations (35-80Hz) by fast transient responses in cat visual cortex. Proc Natl Acad Sci U S A 93: 6112-6117.

Lachaux JP, Rodriguez E, Martinerie J, Adam C, Hasboun D, Varela FJ (2000) A quantitative study of gamma-band activity in human intracranial recordings triggered by visual stimuli. Eur J Neurosci 12: 2608-2622.

Lo FS, Lu SM, Sherman SM (1991) Intracellular and extracellular in vivo recording of different response modes for relay cells of the cat's lateral geniculate nucleus. Exp Brain Res 83: 317-328.

Lu SM, Guido W, Sherman SM (1992) Effects of membrane voltage on receptive field properties of lateral geniculate neurons in the cat: contributions of the low-threshold Ca2+ conductance. J Neurophysiol 68: 2185-2198.

Lutzenberger W, Pulvermuller F, Elbert T, Birbaumer N (1995) Visual stimulation alters local 40-Hz responses in humans: an EEG-study. Neurosci Lett 183: 39-42.

Maldonado PE, Gerstein GL (1996) Neuronal assembly dynamics in the rat auditory cortex during reorganization induced by intracortical microstimulation. Exp Brain Res 112: 431-441.

MacDonald KD, Barth DS (1995) High frequency (gamma-band) oscillating potentials in rat somatosensory and auditory cortex. Brain Res 694: 1-12.

McCormick DA, Bal T (1997) Sleep and arousal: thalamocortical mechanisms. Annu Rev Neurosci 20: 185-215.

Molotchnikoff S, Shumikhina S (1996) The lateral posterior-pulvinar complex modulation of stimulus-dependent oscillations in the cat visual cortex. Vision Res 36: 2037-2046.

Molotchnikoff S, Shumikhina S (2000) Relationships between image structure and gamma oscillations and synchronization in visual cortex of cats. Eur J Neurosci 12: 1440-1452.

Powell TPS, Mountcastle VB (1959) Some aspects of the functional organization of the cortex of the postcentral gyrus of the monkey: a correlation of findings obtained in a single unit analysis with cytoarchitecture. Bull John Hopkins Hosp 105: 133-162.

Sally SL, Kelly JB (1988) Organization of auditory cortex in the albinos rat: sound frequency. J Neurophysiol 59: 1627-1638.

Schanze T, Eckhorn R (1997) Phase correlation among rythms present at different frequencies: spectral methods, application to microelectrode recordings from visual cortex and functional implications. Int J Psychophysiol 26: 171-189.

Singer W (1990) Search for coherence: a basic principle of cortical self-organization. Concepts Neurosci 1: 1-26.

Singer W (1999) Neuronal synchrony: a versatile code for the definition of relations? Neuron 24: 49-65.

Steriade M, Contreras D, Curro-Dossi R, Nunez A (1993) The slow (<1Hz) oscillation in reticular thalamic and thalamocortical neurons: Scenario of sleep rythm generation in interacting thalamic and neocortical networks. J Neurosci 13: 3284-3299.

Steriade M, Contreras D, Amzica F (1994) Synchronized sleep-oscillations and their paroxysmal developments. Trends Neurosci 17: 199-208.

Shosaku A, Sumitomo I (1983) Auditory neurons in the rat thalamic reticular nucleus. Exp Brain Res 49: 432-442.

Tallon C, Bertrand O, Bouchet P, Pernier J (1995) Gamma-range activity evoked by coherent visual stimuli in humans. Eur J Neurosci 7: 1285-1291.

Tallon-Baudry C, Bertrand O, Delpuech C, Pernier J (1996) Stimulus specificity of phase-locked and non-phase--locked 40 Hz visual responses in human. J Neurosci 16: 4240-4249.

Tallon-Baudry C, Bertrand O, Delpuech C, Permier J (1997) Oscillatory gamma-band (30-70 Hz) activity induced by a visual search task in humans. J Neurosci 17: 722-734.

Tallon-Baudry C, Bertrand O, Peronnet F, Pernier J (1998) Induced gamma-band activity during the delay of a visual short-term memory task in humans. J Neurosci 18: 4244-4254.

Tovée MJ, Rolls ET (1992) Oscillatory activity is not evident in the primate temporal visual cortex with static stimuli. Neuroreport 3: 369-372.

Woelbern T, Eckhorn R, Frien A, Bauer R (2002) Perceptual grouping correlates with short synchronization in monkey prestriate cortex. Neuroreport 13: 1881-1886.

Wróbel A (2000) Beta activity: a carrier for visual attention. Acta Neurobiol Exp (Wars) 60: 247-260.

Wróbel A, Bekisz M, Kublik E, Waleszczyk W (1994) 20 Hz bursting beta activity in the cortico-thalamic system of visually attending cats. Acta Neurobiol Exp (Wars) 54: 95-107.

Yordanova J, Kolev V (1997) Alpha response system in children: changes with age. Int J Psychophysiol 26: 411-430.

Yordanova J, Kolev V, Demiralp T (1997) Effects of task variables on the amplitude and phase-locking of auditory gamma band responses in human. Int J Neurosci 92: 241-258.

Young MP, Tanaka K, Yamane S (1992) On oscillating neuronal responses in the visual cortex of the monkey. J Neurophysiol 67: 1464-1474.

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