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SENSORY TRANSDUCTION OF WEAK ELECTROMAGNETIC FIELDS: ROLE OF GLUTAMATE NEUROTRANSMISSION MEDIATED BY NMDA RECEPTORS

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Abstract—Subliminal electromagnetic fields (EMFs) triggered nonlinear evoked potentials in awake but not anesthetized animals, and increased glucose metabolism in the hindbrain. Field detection occurred somewhere in the head and possibly was an unrecognized function of sensory neurons in facial skin, which synapse in the trigeminal nucleus and project to the thalamus via glutamate-dependent pathways. If so, anesthetic agents that antagonize glutamate neurotransmission would be expected to degrade EMF-evoked potentials (EEPs) to a greater extent than agents having different pharmacological effects. We tested the hypothesis using ketamine which blocks N-methyl-D-aspartate (NMDA) receptors (NMDARs), and xylazine which is an α_2-adrenoreceptor agonist. Electroencephalograms (EEGs) of rats were examined using recurrence analysis to observe EEPs in the presence and absence of ketamine and/or xylazine anesthesia. Auditory evoked potentials (AEPs) served as positive controls. The frequency of observation of evoked potentials in a given condition (wake or anesthesia) was compared with that due to chance using the Fisher's exact test. EEPs were observed in awake rats but not while they were under anesthesia produced using a cocktail of xylazine and ketamine. In another experiment each rat was measured while awake and while under anesthesia produced using either xylazine or ketamine. EEPs and AEPs were detected during wake and under xylazine ($P < 0.05$ in each of the four measurements). In contrast, neither EEPs nor AEPs were observed when anesthesia was produced partly or wholly using ketamine. The duration and latency of the EEPs was unaltered by xylazine anesthesia. The afferent signal triggered by the transduction of weak EMFs was likely mediated by NMDAR-mediated glutamate neurotransmission.

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Abbreviations: %D, percent determinism; %R, percent recurrence; ABR, analysis of brain recurrence; AEPs, auditory evoked potentials; atc, amplitude threshold coefficients; coif, Coiflet; Db, Daubechies; EEGs, electroencephalograms; EEPs, EMF-evoked potentials; EMFs, electromagnetic fields; NMDA, N-methyl-D-aspartate; NMDAR, NMDA receptor; XK, xylazine and ketamine.

Key words: cerebellum, evoked potentials, ketamine, electromagnetic fields, nonlinear analysis, xylazine.

INTRODUCTION

Electromagnetic fields (EMFs) with intensities below those that produce conscious awareness of the fields triggered nonlinear evoked potentials in humans and animals (Marino et al., 2003a; Carrubba et al., 2007; Frilot et al., 2009), and increased glucose metabolism in the rat hindbrain (Frilot et al., 2009, 2011). Sensory transduction of low-intensity EMFs by some fish species can be explained on the basis of field-induced forces on the gates of ion channels in neuronal membranes (Kolomytkin et al., 2007), and a similar mechanism might explain electroreception in humans (Marino et al., 2009). The location of the mammalian electroreceptor cell appears to be in the head (Marino et al., 2003a,b). One possibility is that human electroreception is a previously unrecognized function of sensory neurons in facial skin, which synapse in the trigeminal nucleus and project to the thalamus by means of a glutamate-dependent pathway (Lazarov, 2013).

Anesthesia in rabbits produced by a cocktail of ketamine and xylazine blocked EMF-evoked brain potentials (EEPs) (Marino et al., 2002). The drugs were individually capable of producing unconsciousness, but whether they blocked the EEPs individually or cooperatively was unknown. Ketamine antagonizes glutamate neurotransmission mediated by the N-methyl-D-aspartate (NMDA) receptor (NMDAR) (Papich, 2010). Xylazine is an agonist of presynaptic and postsynaptic α_2-adrenoceptors (Bayer Healthcare, 2013), but has no known effects on glutamate signaling. If the effect of the anesthetic cocktail on EEPs was mediated by pharmacological action at NMDARs, a possible consequence would be that ketamine but not xylazine affects the production of EEPs.

Our goal was to further understand the sensory pathway activated by EMFs. We hypothesized that EEPs in rats would be blocked by a cocktail of ketamine and xylazine, but that xylazine alone had essentially no effect on the occurrence or dynamical characteristics of EEPs. We planned to interpret such findings as evidence that the afferent signal triggered by the transduction of weak EMFs was mediated by NMDARs, the major targets of ketamine in the brain.

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EXPERIMENTAL PROCEDURES

Animals

Female Sprague–Dawley rats (300 g) were used in two independent experiments. In the first ($N = 10$), evoked potentials triggered by EMFs were measured while the rats were awake, and while under anesthesia produced by intraperitoneal injection of a cocktail of xylazine (10 mg/kg) and ketamine (75 mg/kg). The anesthesia lasted 40–60 min; a typical experimental session lasted 27 min (Fig. 1b). One animal died during anesthesia, leaving nine available for analysis. In the second experiment 11 additional rats were studied, each of which was measured three times: while awake; after injection with ketamine (75 mg/kg); after injection with xylazine (10 mg/kg). The duration of anesthesia produced by the agents separately was 30–50 min. In both experiments a sound stimulus was used as a positive control. All injections were done under light anesthesia using isoflurane. The chronological orders of treatment (whether awake or under anesthesia) and stimulus application (field or sound) were counterbalanced (Fig. 1c). Repeated measurements on the same rat were made at least 3 days apart. The rats were restrained for measurements during wake. Each rat had been accommodated to the restraint device by means of daily practice sessions that continued until the device was accepted with no manifestations of discomfort. All animal-related procedures were approved by the institutional animal care committee.

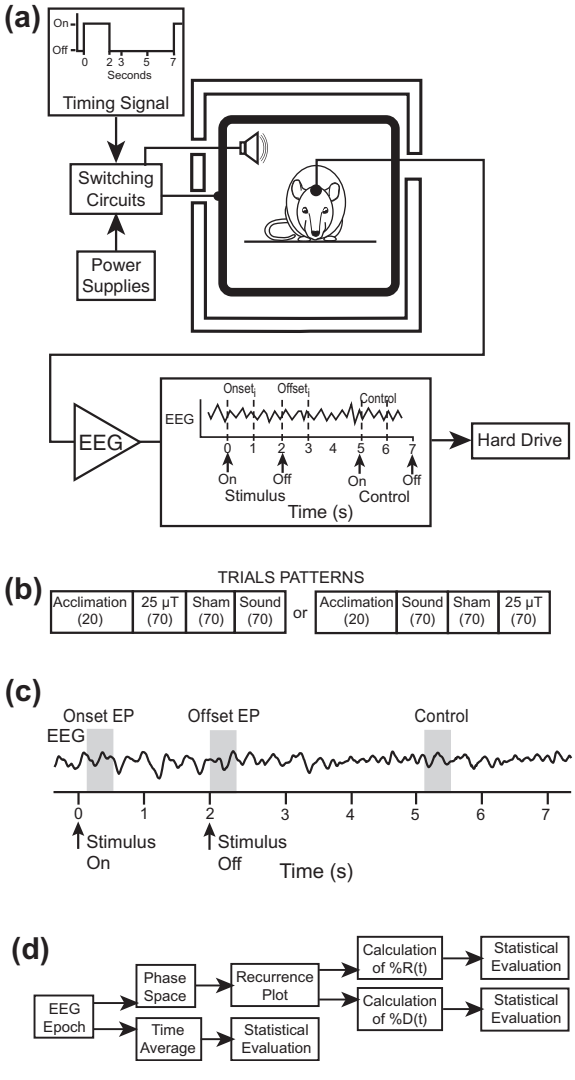

Fig. 1. Application and detection of magnetic fields. (a) Schematic representation of the experimental system. (b) Organization of the 7-s trials in an experimental session (total duration, 27 min). (c) Locations of the onset and offset evoked potentials (EPs) in each pair). (d) Nonlinear and linear analyses for detecting stimulus-induced changes in the EEG.

Stimuli

A 60-Hz magnetic field having a root mean square strength of 2.5 G was applied in a darkened room along the direction parallel to the long axis of the rat's body (Fig. 1a). The field was generated by passing a computer-timed current through a square coil (120 turns, 66 cm side length), and was uniform to within 10% throughout the region occupied by the rats. The sinusoidal current was obtained from a well-regulated power supply (harmonic content < 60 dB). The background 60-Hz magnetic field (present when no experimental magnetic field was applied) was 0.1 mG. The equipment that controlled the magnetic field and recorded the EEG was housed outside the room where the rats were exposed to the field and therefore could not produce auditory or visual clues to the rats. The lack of awareness of EMF or non-EMF stimuli was verified by a determination of the absence of behavioral responses when the equipment was turned on or off. The sound stimulus was a binaural 424-Hz tone having a sound pressure of 65 dB at the location of the rat.

Electroencephalogram acquisition

The electroencephalogram (EEG) was recorded using Ag/AgCl electrodes (Tyco, Mansfield, MA, USA) placed on top of the head and in the middle of the back. Electrode impedances (measured before and after each experiment) were $< 100\ k\Omega$. The signal was amplified (Nihon Kohden, Irvine, CA, USA), analog filtered to pass 0.3–75 Hz, sampled at 300 Hz (National Instruments, Austin, TX, USA), and analyzed offline.

To permit detection of both onset and offset EMF evoked potentials (EEPs) (Fig. 1c), the field was applied for 2 s with an inter-stimulus off period of 5 s. The EEG was divided into consecutive 7-s intervals (trials) with field onset at $t = 0$ and field offset at $t = 2$ s; the portion of the inter-stimulus off period between 5 and 7 s served as a control. Trials containing artifacts (as assessed by visual inspection) were discarded ($< 5\%$ of all trials). The artifact-free trials were digitally filtered between 0.5–35 Hz after removal of the first 30 ms in the EEG signal to eliminate circuit-switching transients (Carrubba et al., 2007).

During each EEG measurement session a rat underwent three blocks of trials (Fig. 1b). The data from the second block (no stimulus applied) were analyzed as a negative control (sham exposure). All results were determined using data from at least 50 trials.

EEG conditioning using wavelets

The EEGs recorded under anesthesia contained strong respiratory and electrocardiographic signals that were not present in EEGs recorded from awake rats. Removal of the respiratory and electrocardiographic signals was necessary to permit detection of the relatively weak evoked potentials, and amplitude-thresholded wavelet analysis was employed for this purpose (Unser, 1996). Briefly, a wavelet transform algorithm (Matlab, MatWorks, Natick, MA, USA) decomposed the EEG into a series of wavelet functions derived from a common generating function (the mother wavelet). During preliminary studies we considered the standard Daubechies (db) and Coiflet (coif) functions as possible mother wavelets. Using three rats, the functions were systematically evaluated at scaling levels of 2–7 and amplitude threshold coefficients (atc) of 1–1.5. We determined that a coif3–atc1 wavelet effectively filtered the physiological artifacts that occurred in the EEGs recorded under ketamine anesthesia, and that a db7–atc1.2 wavelet was similarly effective for removing the artifacts in EEGs recorded using xylazine, either alone or in a cocktail with ketamine. The wavelet filters were applied prospectively to the other respectively anesthetized rats.

Data analysis

Analysis of brain recurrence (ABR), a nonlinear method for quantifying the law-governed (non-random) activity in the EEG was used to detect the evoked potentials produced by magnetic fields and sound (Carrubba et al., 2007); a detailed description of the procedure is given elsewhere (Carrubba et al., 2006). Briefly, the first 100 ms of each EEG epoch of interest (see below) was embedded in a 5-dimensional phase space using a time delay of 5 points, and the corresponding recurrence plot was generated (Eckmann et al., 1987). Euclidean scaling was used to measure distances in phase space, and states were regarded as near if they were within 15% of the largest distance between any two states. The plots were quantified using: (1) percent recurrence (%R), defined as the number of recurrent points in the plot divided by the total number of points in the recurrence matrix; (2) percent determinism (%D), defined as the fraction of the points in the recurrence plot that formed diagonal lines (Zbilut and Webber, 2006). The process was repeated using a sliding window of 1 point in the EEG, yielding the time series %R(t) and %D(t), which were smoothed using a 100-ms, step-1 averaging window; the smoothed time series were analyzed statistically for the presence of evoked potentials (Fig. 1d) (see below).

Statistics

The evoked-potential latency range in %R(t) and %D(t) for rats was previously determined to occur at 200–525 ms (98 points) following presentation of a stimulus (onset or offset) (Frilot et al., 2009), which corresponded to 100–625 ms in the EEG because of the mathematical procedure used to calculate the recurrence variables (Carrubba et al., 2008). These regions were identified in each EEG trial within $t = 0.03$–1 s and 2.03–3 s; 5.03–6 s served as the control (Fig. 1c).

Paired t tests were used to compare the onset and control regions, and the offset and control regions point by point; both comparisons were performed for each of the two recurrence time series. The probability of 10 significant tests at a comparison-wise error rate of 0.05 in 98 tests is $P = 0.0249$. We planned to conclude that the rat had exhibited an evoked potential if a significant result was observed in either %R(t) or %D(t); the corresponding family-wise error rate was therefore $1 - (1 - 0.0249)^2 = 0.049$.

To determine whether the effect of the field could be detected by linear analysis of the EEG, the comparisons described above were repeated using the EEG signal directly (no embedding) (Fig. 1d). We planned to regard a change in brain electrical activity as nonlinear if it was detected only by recurrence analysis.

Contingency tables were constructed to permit assessment of whether the frequency of observation of evoked potentials in a group of rats in a given condition (wake or anesthesia) was greater than that due to chance, as evaluated using the Fisher's exact test. The tables compared the group-wide rate of occurrence of evoked potentials in the presence and absence of a stimulus (EMF or sound). We predicted that EEPs would be observed except when anesthesia was produced partly or wholly using ketamine.

RESULTS

Wavelet filtering

EEGs recorded under anesthesia were heavily contaminated by respiratory and electrocardiographic signals superimposed on the baseline brain activity (Fig. 2), which necessitated use of an automated method for removal of the artifacts prior to ABR for detecting the evoked potentials. Amplitude-thresholded wavelet filtering using coif3–atc1 effectively removed the artifacts in EEGs recorded under ketamine anesthesia (Fig. 2a). The power spectrum of the EEGs recorded using xylazine, alone or in a cocktail with ketamine, differed significantly from the spectrum produced by ketamine alone, necessitating use of a different wavelet filter. Use of a db7–atc1.2 filter effectively removed the artifacts in EEGs recorded from rats anesthetized with xylazine in the absence or presence of ketamine (Figs. 2b, c).

First experiment

A typical onset EEP is shown in Fig. 3a (left panel), which depicts the change in brain activity triggered by the field onset, as detected using %R(t). The latency interval measured from field onset was 351–444 ms, and was detected by means of point-by-point statistical comparisons between onset and control epochs (Fig. 1a). No differences in %R were seen when the

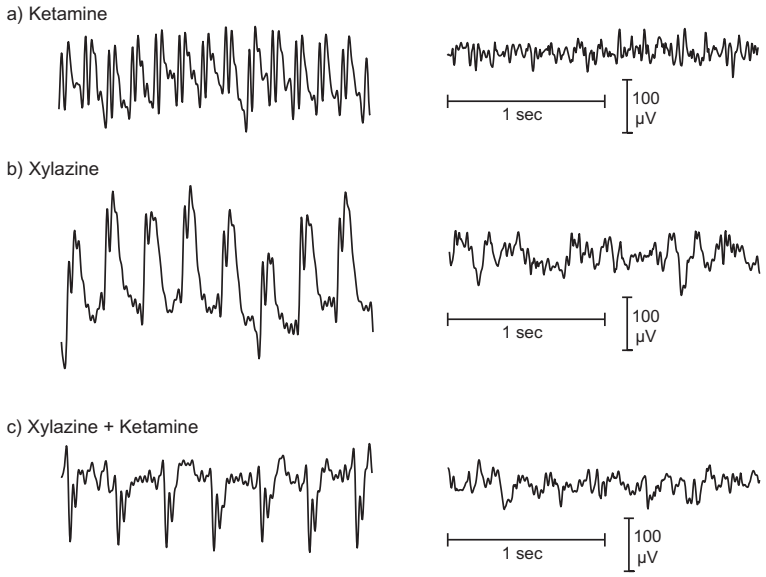

Fig. 2. Effect of wavelet filtering on EEGs from anesthetized rats. Left (right), before (after) wavelet analysis. (a) coif3-atc1 filter. (b, c) db7-atc1.2 filter.

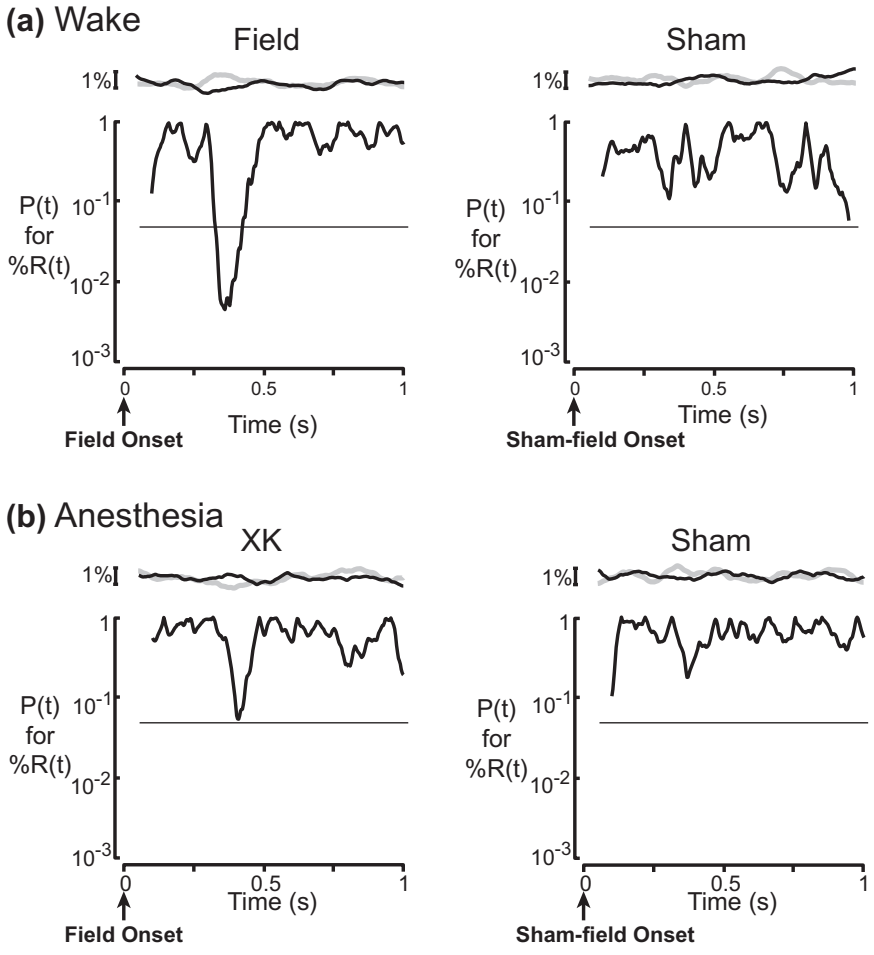

Fig. 3. Representative determination of an EMF-induced evoked potential (EEP) in a rat detected in the recurrence time series (%R(t)). (a) Awake. Average value of %R(t) for the onset (black curve) and control (gray curve) epochs, and point-by-point comparison-wise probability that the curves did not differ (paired *t*-test). An EEP was detected at 351–444 ms, which corresponded to 251–544 ms in the EEG. (b) Corresponding results under xylazine–ketamine anesthesia. Gray line, $P = 0.05$. All curves are shown after use of a 30-point smoothing window.

sham-field-onset epochs were compared with the corresponding control epochs (right panel). When the rat was anesthetized using a cocktail of xylazine and ketamine (XK), the EEP was not detected (Fig. 3b).

During wake, EEPs were seen in 100% of the rats, compared with 10% in response to sham stimulus ($P < 0.05$) (Table 1). Under anesthesia produced by the cocktail, the rate of occurrence of EPs in response to the field did not differ from the controls (22% compared with 11%). Similar results were found using sound as the stimulus. During wake the AEP detection rate was 80%, compared with 10% in the shams ($P < 0.05$); under XK anesthesia, AEPs were not detected (Table 1).

Second experiment

To determine whether the absence of evoked potentials during XK anesthesia was attributable to only one of the drugs, evoked potentials were measured while the rats were awake, and while anesthetized using xylazine alone and ketamine alone. Both EEPs and AEPs were detected during wake and while the rats were anesthetized using xylazine ($P < 0.05$); neither EEPs nor AEPs were detected above the level attributable to chance when the rats were anesthetized with ketamine (Table 2).

Role of nonlinearity

Using recurrence analysis to detect the evoked potentials, both EEPs and AEPs were observed during wakefulness and under xylazine anesthesia (Tables 1 and 2). Using time averaging, AEPs but not EEPs were observed (data not presented).

Table 1. Effect of anesthesia on the production of magnetosensory evoked potentials (EEP) in two independent experiments. In experiment no. 1, $N = 10$ rats, xylazine/ketamine cocktail used for anesthesia. In experiment no. 2, $N = 11$ rats, xylazine and ketamine employed separately for anesthesia

Field		
	Awake EEP	*$P < 0.05$ No EEP
*Field	10	0
Sham field	1	9
	XK anesthesia	
	EEP	No EEP
Field	2	7
Sham field	1	8
Sound		
	Awake AEP	*$P < 0.05$ No AEP
*Sound	8	2
Sham sound	1	9
	XK anesthesia	
	AEP	No AEP
Sound	2	7
Sham sound	1	8

Table 2. Effect of anesthesia on the production of auditory evoked potentials (AEP) in two independent experiments. In experiment no. 1, $N = 10$ rats, xylazine/ketamine cocktail used for anesthesia. In experiment no. 2, $N = 11$ rats, xylazine and ketamine employed separately for anesthesia

Field		
	Awake EEP	*$P < 0.05$ No EEP
*Field	8	3
Sham field	1	10
	Ketamine	
	EEP	No EEP
Field	2	9
Sham field	1	10
	Xylazine	
	EEP	No EEP
*Field	7	4
Sham field	0	11
Sound		
	Awake AEP	*$P < 0.05$ No AEP
*Sound	7	4
Sham sound	1	10
	Ketamine	
	AEP	No AEP
Sound	3	8
Sham Sound	1	10
	Xylazine	
	AEP	No AEP
*Sound	6	5
Sham sound	0	11

Table 3. Latency and duration data

Stimulus	Awake	Xylazine
(a) Latency (msec)		
Field	320 ± 94 (18)	373 ± 109 (7)
Sound	292 ± 77 (14)	263 ± 101 (6)
(b) Duration (msec)		
Field	260 ± 22 (20)	264 ± 18 (7)
Sound	284 ± 71 (15)	275 ± 24 (6)

Latencies and durations

On average, the latencies of the evoked potentials were greater for the EMF stimulus compared with the sound stimulus, irrespective of the presence or absence of xylazine. The duration of the evoked potentials did not depend on either stimulus or the presence of anesthesia (Table 3).

DISCUSSION

Animals and humans detected weak low-frequency EMFs by means of sensory transduction, as determined by their ability to trigger EEPs (Marino et al., 2003a; Carrubba et al., 2007; Frilot et al., 2009). Detector-localization studies suggested that the cell which directly interacted with the field was located somewhere in the head. Retinal cells were tentatively excluded as the locus of the electroreceptor function, but polymodal sensory cells in the skin remained reasonable possibilities (Marino et al., 2003a,b). This idea was supported by observations that EMF exposure increased glucose uptake in the region of the brain stem that includes the trigeminal nucleus (Frilot et al., 2009, 2011). EEPs were blocked in rabbits that had been anesthetized using a cocktail of ketamine and xylazine (Marino et al., 2003a). The drugs can independently produce unconsciousness, but only ketamine is known to block glutamate neurotransmission, the primary neurotransmitter for signaling from the trigeminal nucleus to the thalamus (Lazarov, 2013). We hypothesized that ketamine alone mediated the blockage of EEPs in animals anesthetized with an XK cocktail. To support this hypothesis we sought evidence that EEPs occurred only in the absence of ketamine.

When rats were studied while awake, EEPs were observed in two independent experiments (Tables 1 and 2), as expected based on an earlier study (Frilot et al., 2009). Under anesthesia, EEPs were blocked in the presence but not the absence of ketamine. In the first experiment (Table 1), anesthesia was produced using an XK cocktail and EEPs were not observed. In the second experiment, using only xylazine, EEPs were observed at essentially the same rate as when the animals were awake (Table 2). In contrast, using ketamine, EEPs were not observed at a rate greater than that found using a sham-field stimulus (Table 2). Thus the EEPs were blocked by an anesthetic agent that acted primarily by antagonizing NMDA receptors, but not by an agent that stimulated α_2-adrenoreceptors. We interpreted these results as evidence that the afferent pathway triggered by sensory transduction of the EMF consisted at least partially of glutamate neurotransmission via NMDA receptors.

Several considerations supported the interpretation. First, the NMDAR is a major glutamate receptor in the cochlear nuclei located in the brain stem (Petralia et al., 2000). Consequently, if inhibition of glutamate neurotransmission was responsible for blocking the EEPs, a similar effect would be expected with respect to AEPs. Indeed, essentially the same results occurred when sound was employed as the stimulus (Tables 1 and 2). Second, the latency and duration of the EEPs and the AEPs observed under xylazine did not differ from their comparable values when the rats were awake (Table 3). These results suggested that both afferent pathways were unaffected by xylazine, further suggesting that blockage of evoked potentials associated with the cocktail was mediated only by ketamine. Third, we showed previously that XK did not block visual evoked potentials in rabbits (Marino et al., 2003a). There are at least seven different neurotransmitters in retinal ganglion cells (Karten et al., 1984), which decreased the likelihood that ketamine could block all inputs to the visual cortex, possibly explaining why visual evoked potentials were not blocked by ketamine (Leopold et al., 2002; Marino et al., 2002).

The interactions between the cardiac, respiratory, and electroencephalographic signals in rats under XK depend on the depth of the anesthesia, which changes continuously while the rats are unconscious (Musizza et al., 2007). We did not take this variability into account, but rather regarded all moments under anesthesia as more or less equivalent for the purpose of affecting the generation of evoked potentials. An additional study limitation stems from reports that the known pharmacological properties of ketamine are not specific for NMDA receptors but rather can be diverse (Schnoebel et al., 2005; Papich, 2010), depending on various factors. The possibility remains that other processes caused or contributed to the blockage of evoked potentials by ketamine.

The presence of physiological artifacts materially complicated the task of detecting evoked potentials in anesthetized rats. EMF-induced evoked potentials were detected in 18 of 20 rats while they were awake, but only 7 of 11 rats under xylazine. This lower detection rate was probably a result of our choice of wavelet filter, which was chosen primarily to minimize false positive results during sham exposure, because our statistical objective was to demonstrate that the rate of true positives did not differ from that of awake animals. There are many techniques for removing artifacts from EEGs, some of which may be superior to wavelets under some conditions (Daly et al., 2013). We did not pursue that issue.

The present results can be integrated with those of earlier studies, leading to a proposed model for EMF detection (Fig. 4) that is more detailed than the initial version of the model (Becker and Marino, 1982). Environmental-strength EMFs are detected by sensory cells in the vicinity of the epidermal–dermal junction in facial skin. Biophysical detection of low-frequency EMFs involves a direct force on a channel gate caused by an electric field, either applied externally or resulting from an applied magnetic field as a consequence of Faraday's law. The detection model is thermodynamically consistent with field strengths known to exist in the basal epidermis as a result of environmental EMFs, and with the known response times of membrane ion channels (Marino et al., 2009). It can be shown that the model is consistent with detection of frequencies as high as several kHz. The force alters the channel mean open time, resulting in local receptor potentials that produce afferent signaling in the trigeminal nerve, either generation of action potentials or modification of ongoing afferent signaling, depending on the type of the sensory cell. The afferent signal triggers glutamate-mediated neurotransmission in the trigeminal nucleus leading to thalamic projection of the signal. EMF-induced evoked potentials are not

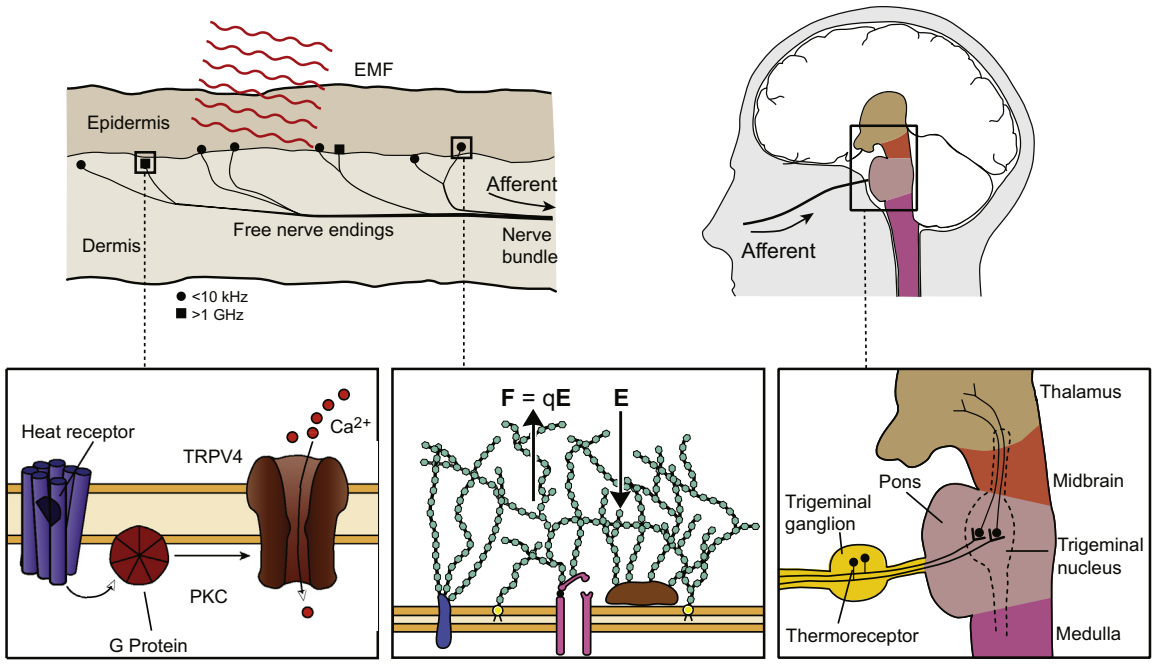

Fig. 4. Model for the detection of environmental-strength electromagnetic fields (EMF) at low frequencies (< 10 kHz). The electric (E) component of the field induces forces on charge glyco groups attached to gates of ion channels in neurons. Thermodynamic modeling of the process showed that 1 μV/m can alter the mean open time of typical channels (Kolomytkin et al., 2007). At high frequencies (> 1 GHz) heat generated by the EMF is the physical stimulus transduced by the nervous system.

consciously detected in animals or humans, suggesting the absence of cortical projections even in awake animals. High-frequency fields such as those produced by cell phones (1 GHz) are likely detected by warmth detectors in the skin. Gigahertz fields easily penetrate the epidermis and produce temperature changes on the order of 0.1 °C. Skin sensory cells are known to contain TRP channels capable of responding to temperature changes as low as 0.01 °C, thereby triggering signals to the thalamus by way of the trigeminal nucleus.

In summary, the afferent signals triggered by the transduction of weak EMFs and detected as EEPs using recurrence analysis of the EEG were likely mediated by NMDA receptors in the trigeminal nucleus.

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