

Cortico-subcortical synchronization in the chloralose-anesthetized cat

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Abstract

The spontaneous and paroxysmal cerebral cortical synchronized activity was used as reference to study the cortical impact exerted on subcortical neurons. The sensorimotor cortical synchronized activity spread down to subcortical structures receiving direct cortical input, including neuronal populations that originate descending rubrospinal, tectospinal and reticulospinal motor axons, and to a somatosensory relay station, the cuneate nucleus. Lesion of the pyramidal tract abolished the cortically induced synchronization of the activity of contralateral cuneate nucleus neurons.

Keywords: sensorimotor cortex; synchronized activity; subcortical structures; cat

Abbreviations: CN, cuneate nucleus; ECoG, electrocorticogram; MCx, primary motor cortex; mRN, magnocellular red nucleus; NRGc, nucleus reticularis gigantocellularis; PT, pyramidal tract; SC, superior colliculus; SN, substantia nigra; SSCx, primary somatosensory cortex

The neocortex and the thalamus form an oscillatory network modulated by ascending inhibitory and excitatory influences arising mostly in the brainstem. While the cerebral cortical oscillatory activity during different behavioral states and anesthesia has been preferentially studied in conjunction with reciprocal thalamic rhythmicity⁷ (and references therein), few studies have dealt with the problem of coherent activity between ensembles of neurons at multiple levels of the brain.^{3, 5, and 8} Since the long-axoned neocortical neurons present oscillatory activity,⁶ it is expected that these neurons would transmit the major cortical rhythms to their targets. Accordingly, we decided to study whether the cerebral cortex may impose the patterns of its activity on subcortical structures targeted by corticofugal fibers. Spontaneous sensorimotor cortical field potentials and electrically induced paroxysmal activity were correlated with the extracellular activity of different subcortical structures.

A total of 12 cats of either sex (2.2–4.5 kg) were anesthetized (α -chloralose, 60 mg/kg, i.p.), artificially respired and routinely subjected to a bilateral pneumothorax. A craniotomy was performed over the sensorimotor cortex, and a set of six bipolar electrodes mounted in a tower and lowered to 1.5 mm deep to electrically stimulate the precruciate primary motor (MCx) and postcruciate primary somatosensory (SSCx) cortices to induce paroxysmal cortical discharges and to observe their impact on subcortical structures. A concentric bipolar electrode was positioned at 1 mm depth in the lateral tip of the cruciate sulcus to differentially record the depth electrocorticogram (ECoG) activity between the inner (active) and the outer (reference) leads, separated by 500 μ m. Tungsten recording electrodes (1–5 M Ω resistance) were lowered ipsilaterally into the inferior laminae of the superior colliculus (SC), the magnocellular red nucleus (mRN) and the substantia nigra (SN), and contralaterally into the nucleus reticularis gigantocellularis (NRGc) and the middle main cuneate nucleus (CN). Contrary to the SN, the SC, mRN, NRGc and CN are known to receive direct sensorimotor corticofugal inputs. Thus, recording of the activity from the basal ganglia structure served to compare the corticofugal effects exerted on an indirectly targeted subcortical site with directly influenced structures. The inferior laminae of the SC were identified by cutaneous stimulation of the contralateral forelimb and by electrically stimulating the contralateral medial bulbar reticular formation, the mRN by electrical stimulation of the contralateral dorsolateral funiculus at the cervical level,¹ the NRGc by stimulating the cervical ventromedial funiculus,² the NC by cutaneous stimulation of the distal ipsilateral forelimb and electrical stimulation of the contralateral medial lemniscus, and the SN by recording neuronal activity immediately below the medial lemniscus at Horsley–Clarke coordinates A2, L4.5.

The ECoG and extracellular activity from each subcortical structure were simultaneously recorded spontaneously and after inducing paroxysmal activity within the sensorimotor cortex. Electrical stimulation between both leads (separated by 500 μm) of each bipolar cortical stimulating electrode at 50–100 Hz for 1–2 s (0.05 ms pulse duration) induced cortical synchronized activity that outlasted the stimulation by 5–25 s. The paroxysmal postdischarges initiated at frequencies of 7–20 Hz, and gradually increased in amplitude and slowed in frequency to reach 0.5–6 Hz before ending abruptly. In five animals, the pyramidal tract (PT) was approached ventrally and electrolytically lesioned to study whether the synchronous activity observed in the cortex and the cuneate depended on the integrity of this tract.

The results were homogeneous in all the animals. With the exception of the SN, the subcortical structures tested showed coherent oscillatory activity related to cortical synchronized activity, both spontaneous and electrically induced, as observed in the ECoG. Figure 1 shows coherent oscillatory activity between spontaneous cortical spindles and bursting discharges in the SC, NRGc and mRN, but not in the SN. Note that, under chloralose anesthesia, the ECoG intra-spindle activity presented frequencies of 3–8 Hz.

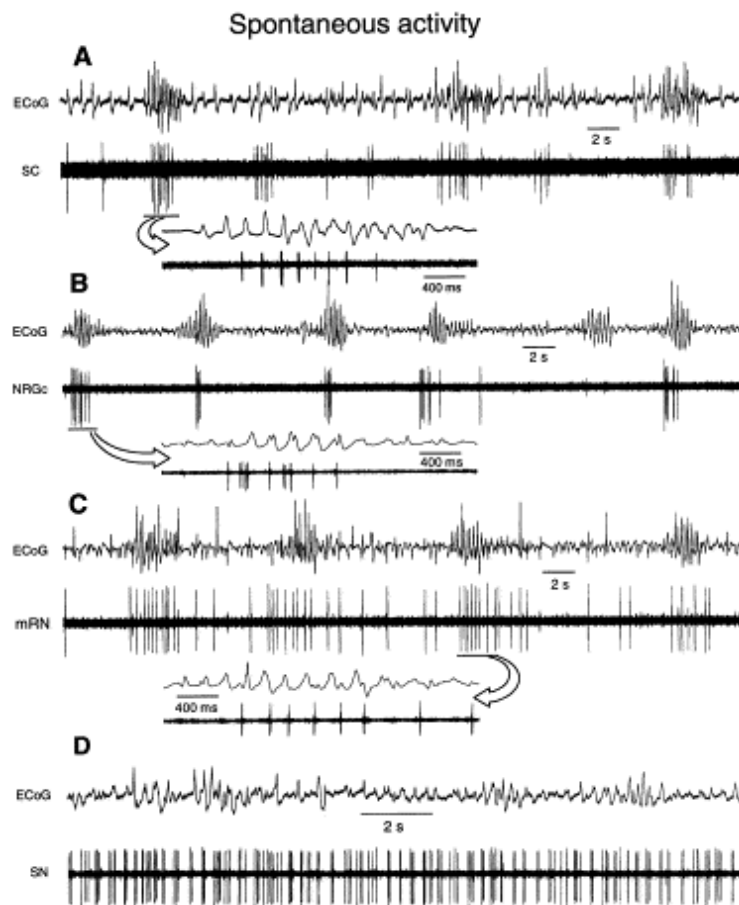


Fig. 1. The synchronized cortical activity spreads down to directly contacted subcortical structures. The spontaneous ECoG activity imposed its rhythmic patterns on the extracellular activity of the SC (A), the NRGc (B) and the mRN (C), but not on the activity of the SN neurons (D). In A–C, the selected portions marked by horizontal bars are expanded below.

Electrically induced sensorimotor cortical paroxysmal activity also spread down to the SC, NRGc and mRN, but not to the SN (Fig. 2), even when stimulating cortical premotor areas (this was done in three animals in an attempt to influence the SN activity). Different cortical sites showed preferences for each structure. Thus, SSCx stimulation induced coupled activity in the SC but only occasionally in the NRGc and mRN, whose activities were clearly coupled to the postdischarges induced by MCx stimulation (Fig. 2). However, SSCx and MCx tetani were equally effective in synchronizing the cuneate activity (Fig. 3). The synchronization observed in subcortical neurons reflected the cortical hypersynchronization. The subcortical paroxysmal bursting discharges were time locked to the “spike” component of cortical field

potentials, while silenced firing was related to the “wave” component, in particular at the end of the paroxysms when they reached spike-and-wave discharges at 0.5–6 Hz (Figs 2A–C, 3A). The synchronizing process increased progressively, the subcortical discharges lagged behind the onset of the “spike” in the ECoG, and both cortical and subcortical paroxysmal activities terminated abruptly and synchronously. This indicates that none of the tested subcortical structures possesses the internal circuitry necessary to generate epileptic-like activity by itself. Stimulation of the ipsilateral cortex drove SC and mRN neurons, while stimulation of the contralateral cortex drove NRGc and CN neurons, and none of the subcortical structures were bilaterally driven from the cortex.

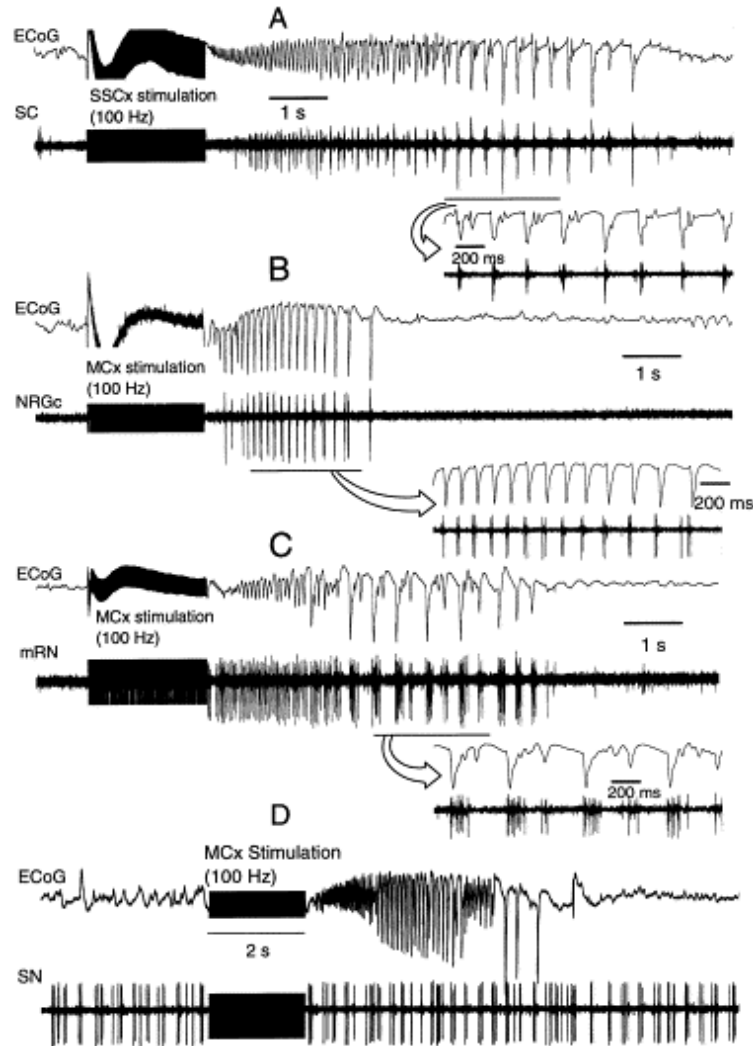


Fig. 2. The paroxysmal activity induced in the sensorimotor cortex spreads down to subcortical structures which originate descending motor axons. Electrical tetani (100 Hz for 2 s) applied to the SSCx or to the MCx induced epileptiform activity which was transmitted to the SC (A), the NRGc (B) and the mRN (C), but not to the SN (D). The portions marked by horizontal lines in A–C are expanded below.

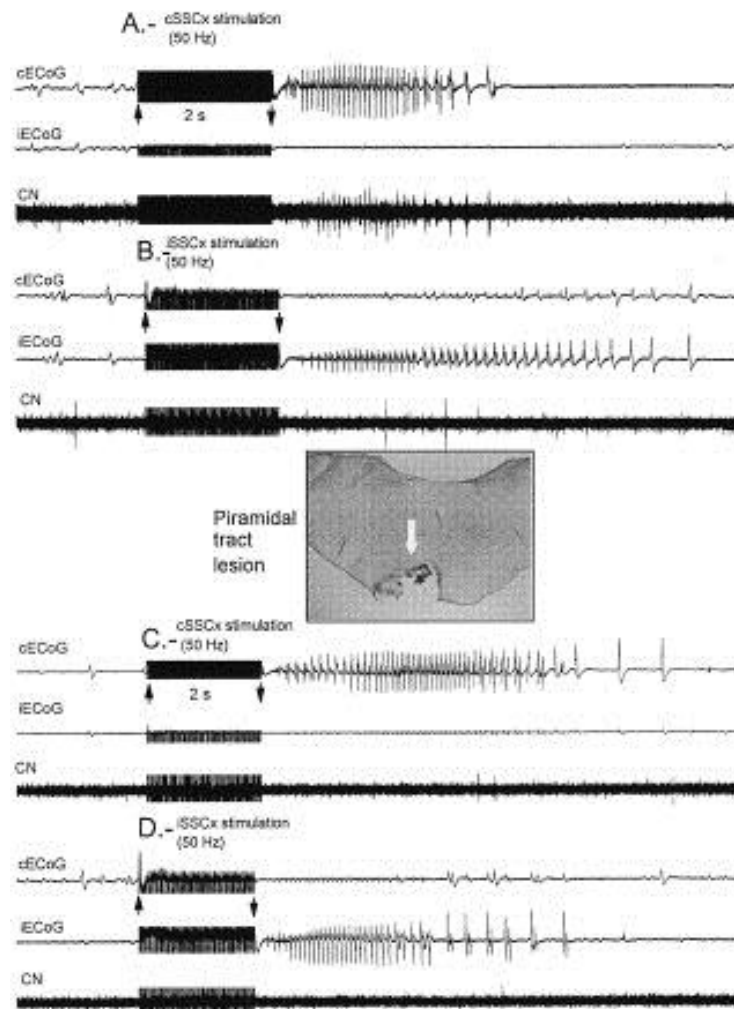


Fig. 3. The cortical paroxysmal activity is transmitted to the contralateral CN through the ipsilateral PT. Electrical stimulation (50 Hz for 2 s) of the contralateral primary somatosensory cortex (cSSCx) induced ictal discharges (cECoG) transmitted to the CN (A). The same stimuli applied to the ipsilateral cortex also induced paroxysmal activity (iECoG) that was not transmitted down to the CN (B). Electrolytic lesion of the PT (inset, the lesion denoted by a white arrow) prevented the transmission of the contralateral corticofugal-induced paroxysmal activity to the CN (C), and the ipsilateral cortex remained ineffective (D).

Finally, PT lesion abolished the cortically induced rhythmic discharges in the CN, as shown in Fig. 3, illustrating that contralateral cortical stimulation was effective in driving the cuneate neurons (Fig. 3A) which were not affected by stimulation of the ipsilateral sensorimotor cortex (Fig. 3B). Electrolytic lesion of the PT (inset in Fig. 3), while maintaining the recording cuneate electrode in place, avoided the spread of the cortical ictal activity to the contralateral CN (Fig. 3C), which corroborates that the cortico-dorsal column nuclei fibers course through the PT.⁴

Our data demonstrate that the cerebral cortex spreads its patterns of activity to the subcortical neurons to which it directly projects. The exerted effects are easily detected when ensembles of corticofugal neurons discharge synchronously both during spindle and delta rhythms or after electrically induced epileptiform discharges. Although the paroxysmal discharges represent a physiopathological process, they are of considerable help in illustrating corticofugal interactions. Under normal circumstances, however, particular ensembles of cortical neurons tend to be activated by common afferent and recurrent inputs influencing specific subcortical neuronal populations that originate descending motor tracts (rubrospinal, tectospinal, reticulospinal), as well as sensory relay stations like the CN. This would imply that the cerebral cortex is able to co-ordinate the activity among different descending motor systems and, at the same time, to contribute to select the feedback sensory ascending information generated by the movement itself.

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