Dedicated Neuronal Ensembles in the Cerebral Cortex

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Abstract

Although continuous activity and stimulus responses are combined in neural activity in the sensory cortex, it is still unknown whether these reflect the same underlying dynamics or different processes. In the current study, we demonstrate in mice that the neuronal assemblages elicited by sounds in the auditory cortex and thalamus during awake are unique to the stimulus and different from the assemblies seen in continuous activity. In contrast, evoked assemblies in the cortex are indistinguishable from continuing assemblies when subjected to three different anesthetics. In the thalamus, they continue to be different, nonetheless. This dynamic state change brought on by anesthesia is accompanied by a substantial remapping of sensory responses.

Introduction

It has long been known that, in the absence of stimuli from their specific sensory modality, the circuits of sensory areas in the cerebral cortex exhibit tremendous continuous activity. Uncertainty exists regarding the function of this continuous activity and how it relates to triggered sensory reactions. Initial anesthetic investigations in the visual cortex of numerous mammals have revealed strong similarities between patterns of continuous activity on the mesoscopic scale and sensory responses, pointing to the possibility that ongoing activity is a replay of sensory responses. In the primary auditory cortex of rats and guinea pigs, similar findings have been made.

Recent recordings in awake mice's visual cortex, however, have demonstrated that continuing cortical activity in wakefulness is closely related to both the animal's state of arousal and the patterns of its facial motor activity. The visual thalamus exhibits changes in response to arousal as well. This shows that continuing cortical dynamics in the awake state are more than just a replay of previous sensory activity, even though the direction of causality between behavioral and cortical observables is still unknown. Similar groups of neurons are recruited by both continuous and evoked activity, but they correspond to activity patterns that exist in orthogonal neuronal dimensions, which is consistent with our observation.

These contradictory findings in various physiological situations imply that anesthesia causes a significant alteration in neuronal dynamics that extends beyond cortical networks. Strong effects of anesthesia on cortical neuronal integration, as well as cortico cortical and, to a lesser extent, thalamocortical connections, corroborates this theory. Field potential recordings and single-cell analyses of cortical neurons during anesthesia and alertness show altered sound response and a lower signal-to-noise ratio. The lack of spatially defined data from these research on thalamocortical population activity patterns makes it impossible to tell if the resemblance between evoked and spontaneous population patterns is a side effect of anesthesia or a more widespread phenomenon. Throughout wakefulness and three different types of anesthesia, we examined continuous and sound-evoked activity in sizable populations of mouse auditory cortex neurons as well as axonal terminals from the auditory thalamus in the current work. We found that during awake, the cortex produces discrete evoked and continuing cell assemblies that facilitate accurate encoding of a variety of sounds. On the other hand, during anesthesia, ongoing and evoked activity patterns merged into one. As a result, even though there were particular sound responses in the anaesthetized condition, sound representations were significantly diminished and dissimilar from those seen in the waking state.

We have noted unique continuous and evoked assemblies in thalamocortical axons during wakefulness, and in the current investigation, the two types of assemblies remained distinct under anesthesia. Under anesthesia, this suggests a functional disconnect between the cortex and its thalamic inputs, whereas the presence of discrete, continuous cortical cell assemblies that are sound-specific appears to be a hallmark of awake perception.

Our findings demonstrate that during wakefulness, sensory inputs and ongoing activity interact with specific neuronal assemblies in the cortex, whereas under anesthesia, sensory responses produce assemblies that also manifest in continuing activity. This discovery explains earlier findings that were inconsistent and came from the two distinct states. At mesoscopic and cellular levels, evoked responses in continuing neuronal assemblies in anaesthetized animals had been seen to occur again. This was in contrast to recent observations showing continuing activity in awake animals is primarily orthogonal to evoked responses. In the current study, we compare thalamic activity along with the spontaneous and evoked cell assemblies in both states.

We discovered that although sound stimuli under anesthesia activate stereotyped cortical cell assemblies that are already active during spontaneous activity, despite the fact that thalamic inputs to the cortex differ between spontaneous and evoked activity. In contrast, when an animal is awake and aware of sounds, sound stimuli cause sound-specific cell assemblies to be activated.

One key finding is that in the anaesthetized cortex, the cell assemblies evoked appear to interact with cell assemblies that are already active during spontaneous activity. This is because both spontaneous and evoked activities originate from the same small set of cortical cell assemblies, but in the awake cortex, much larger and more varied sets of assemblies are visible. Previous research that suggested the anaesthetized auditory cortex had a reduced dimensionality of sound responses may be explained by the limiting of dynamics during anesthesia. It is interesting to note that even if sensory information collapses during anesthesia, it does not completely vanish as evidenced by sound classification performance that is well above chance levels and the retention of the response profiles by over half of the neurons.

This is consistent with the widespread finding that anesthesia does not completely abolish stimulus-specific patterns19. Although we were unable to directly evaluate this aspect, earlier publications suggest that activity patterns in the anaesthetized state follow recognizable functional maps, such as the ton topic map in the auditory cortex or the contour orientation maps in the visual cortex of carnivores. The spatial scope of the stereotyped population events that dominate cortical dynamics during anesthesia may be constrained by these maps, which correspond to anatomically hardwired circuits. In humans, anesthetic resting-state activity likewise exhibits a reduction in complexity and follows large-scale anatomical connection networks.

This could also explain why the main sensory cortex already experiences a loss of perception during an aesthesia, as evidenced by our findings. Our findings support the notion that newly formed assemblies resulting from sensory inputs will spread throughout the awake cortex, but the mechanisms underlying this selective propagation are currently unknown. However, earlier models have suggested that asynchronous states may serve as the substrate for this selective propagation.

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