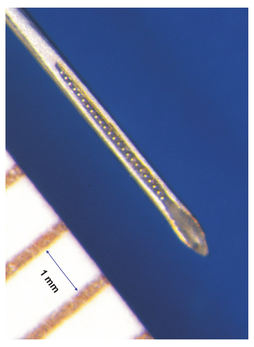
CORTICAL GATING OF AUDITORY INFORMATION PROCESSING IN SLEEP

Grand L.[1,2], Dombovari B.[1,2], Csercsa R.[1], Wittner L.[1], Magony A.[1,2], Karmos G.[1,2], Ulbert I.[1,2]

[1.] Institute for Psychology, Hungarian Academy of Sciences, Budapest, Hungary
[2.] Peter Pazmany Catholic University, Budapest, Hungary

Laminar electrode

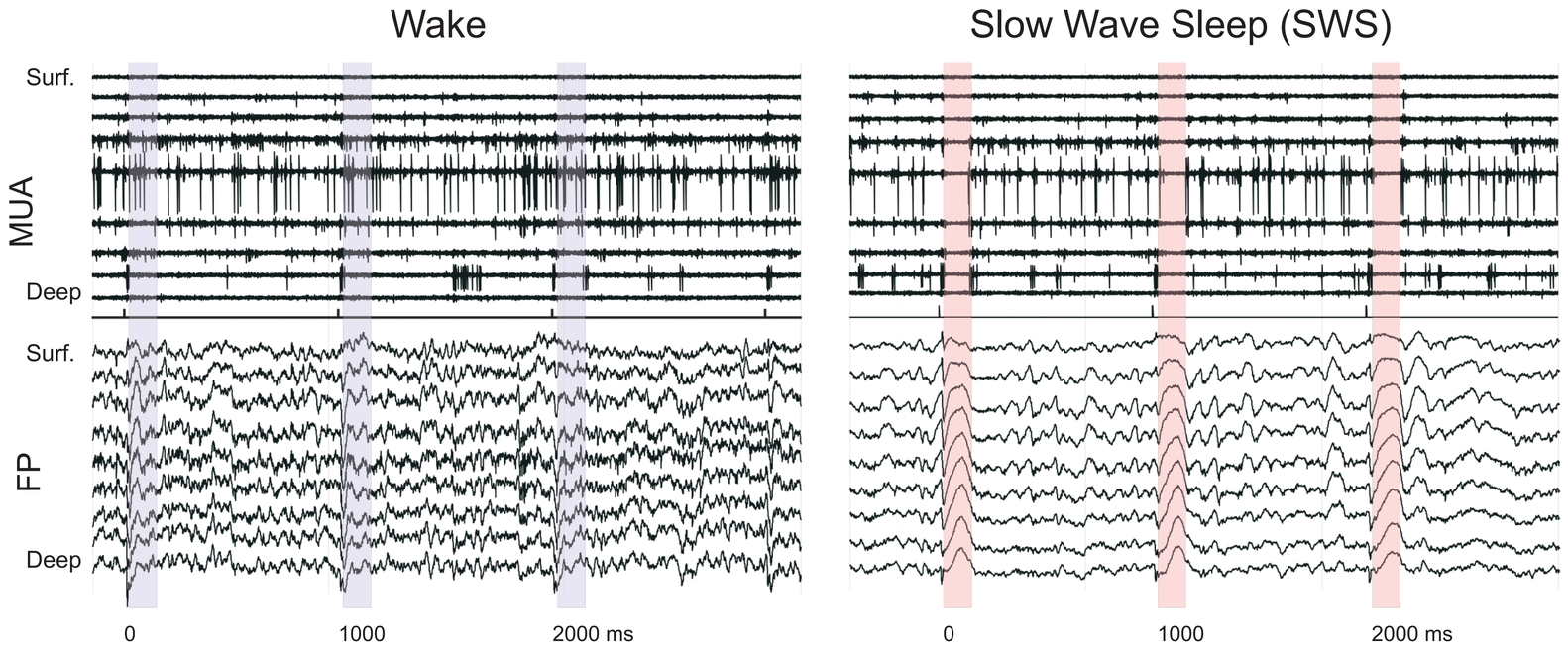


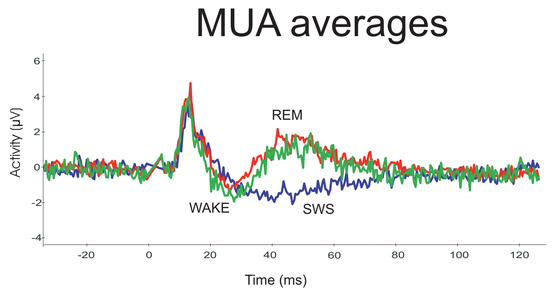
INTRODUCTION

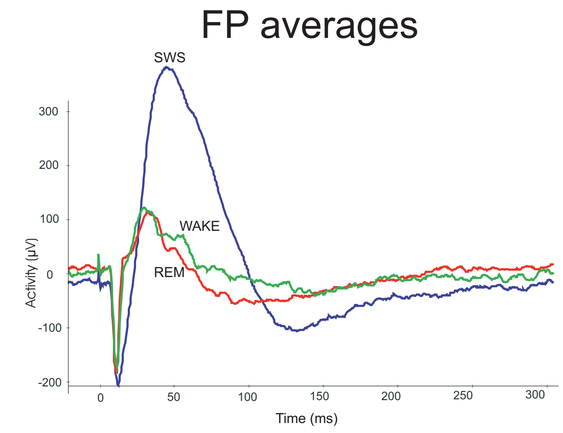
In sleep, we are unaware most of the ongoing auditory signals. Thalamic and cortical processes are responsible for filtering out unnecessary acoustic inputs to the cortex in sleep. Here we show that sound evoked cortical hyperpolarization may be responsible for gating of cortical communication and thalamic input, in the natural non-REM sleep of the cat. This mechanism shares the extracellular signatures of down-states of the cortical slow oscillations (SO) observed under ketamine anesthesia.

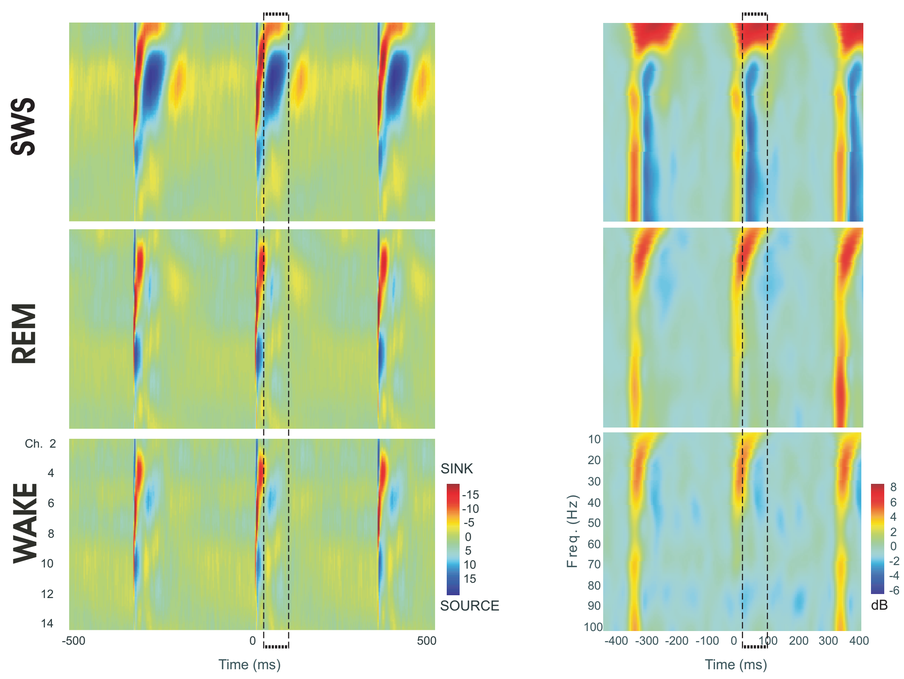
METHODS

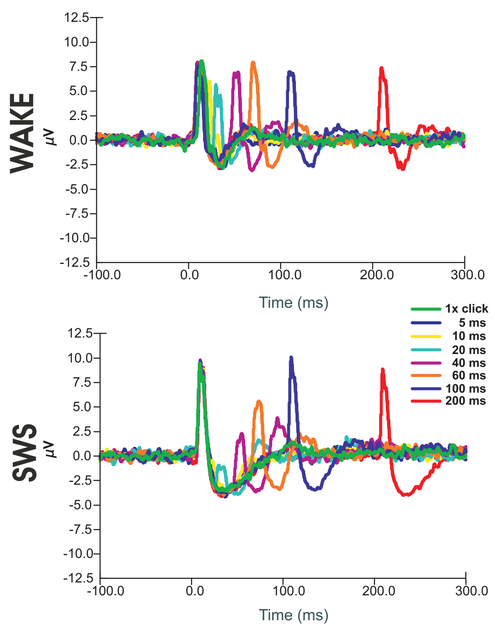
Cats were implanted with laminar multielectrodes into auditory cortex in addition to epidural, hippocampus, EOG, EMG electrodes and bone conductor to deliver click stimulus. In natural non-REM sleep, clicks evoked a depth positive component (40-140ms) after the initial transient response. The positivity was not present in natural REM sleep, or awake. Animals were then anesthetized with ketamine, to produce stable SO.

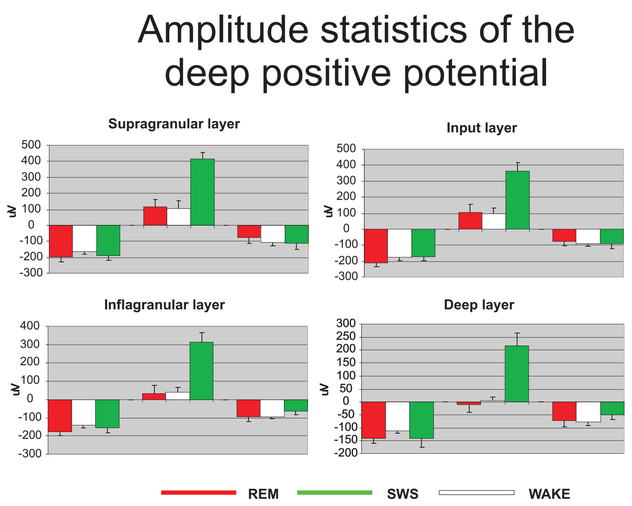


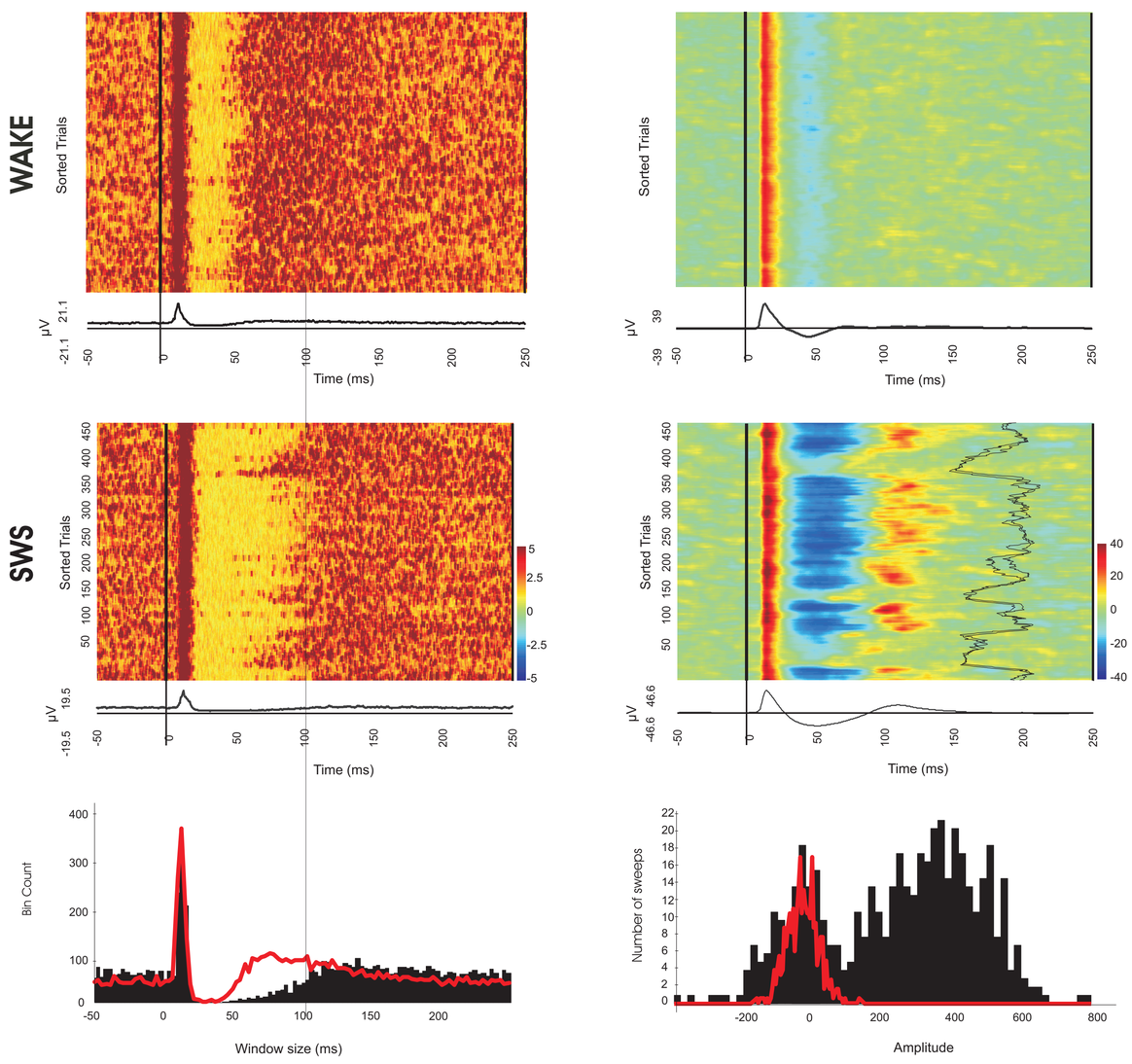


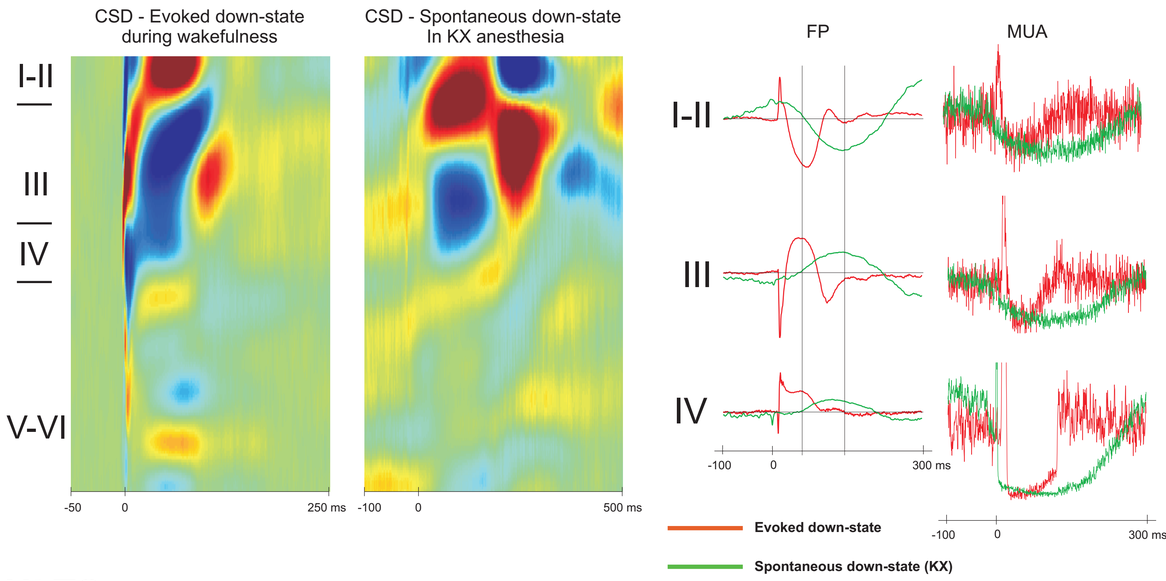












RESULTS

Current source density, multiple unit activity and time-frequency analysis showed similar signatures of the depth positive evoked component in natural non-REM sleep, and during the down-state of SO under ketamine anesthesia. Both events were characterized by oscillatory power (20-120ms, 1-200Hz) and firing rate decrease in all layers of the cortex. Prominent middle layer hyperpolarizing currents were also present in both cases, however, the average duration of the 'down'states were twice as long (200ms) as the evoked positivity.

CONCLUSIONS

Based on the similarities between down-states and hyperpolarization, we hypothesize that similar cellular and membrane mechanisms might be in action during these events. Evoked cortical hyperpolarization serves as an important sleep protecting mechanism via literally 'shutting down' cortical information processing for a brief period of time and pushing the membrane potential away from the firing threshold.

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E-mail: grand@cogpsyphy.hu