Imperceptible Stimuli and Sensory Processing Impediment

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Weak sensory stimuli can fully escape conscious perception and yet evoke minute electroencephalography (EEG) responses (*1*), indicating at least partial cortical processing of such "subliminal" input. We used functional magnetic resonance imaging (fMRI) during imperceptible electrical finger stimulation to characterize the extent and nature of associated cortical processing (*2*). No fMRI activation could be identified; i.e., subliminal stimuli did not elicit any significant positive blood-oxygenation-level–dependent (BOLD) signal change. In contrast, statistical parametric maps for negative T contrasts (Fig. 1A) revealed distinct BOLD signal decreases, sharply localized at the hand area of the contralateral primary somatosensory cortex (S1), in the secondary somatosensory cortex (S2), and in the supplementary motor area (SMA). As either imperceptible stimuli or no stimuli at all were presented and, consequently, subjects could not detect the stimulus sequencing, cognitive response modulations, e.g., from mere stimulus anticipation (*3*), can be excluded.

Possible interpretations of negative BOLD signal changes include a focal "deactivation," e.g., a reduced baseline activity mediated by local inhibitory interneurons or due to diminished input from distant projection neurons (*4*). The first notion is supported by cortical deactivations after rapid-rate subthreshold transcranial magnetic stimulation (*5*), which activates predominantly local inhibitory interneurons, possibly causing a net reduction of spontaneous excitatory synaptic activity.

The impact of subliminal stimuli on the detectability of near-threshold test stimuli was thus analyzed in a psychophysiological experiment (*2*): The interpulse delay between a subliminal and a subsequent test pulse was set to 30 ms, far beyond the refractory period of peripheral nerve fibers, yet still within the period of intracortical disynaptic inhibition triggered by single thalamocortical spikes (*6*). The detectability of intermingled near-threshold stimuli was reduced during subliminal stimulation (Fig. 1B). Therefore, the focal cortical fMRI signal decrease, which was induced by subliminal stimuli comparable to those used in the psychophysiological study, reflected a functionally effective inhibition in the somatosensory system. This was corroborated by a second fMRI experiment where the BOLD activation evoked by clearly detectable near-threshold supraliminal stimuli was reduced (predominantly in contralateral S1) when subliminal stimulation was added (*2*).

Because inhibitory interneurons in S1 receive monosynaptic thalamocortical input and provide for highly efficient and synchronized low-threshold intracortical feed-forward inhibition (*6*–*8*), we propose that the net cortical deactivation, caused here by weak subliminal stimuli, reflects a threshold nonlinearity that generally protects the cortex against spurious activation by functionally equivocal channel "noise." In more general terms of thalamocortical circuitry, such critically poised trigger level for intracortical feed-forward inhibition could serve as a screen against cortical network activation by sporadic thalamocortical spikes that might occur spontaneously without being driven by an external stimulus.

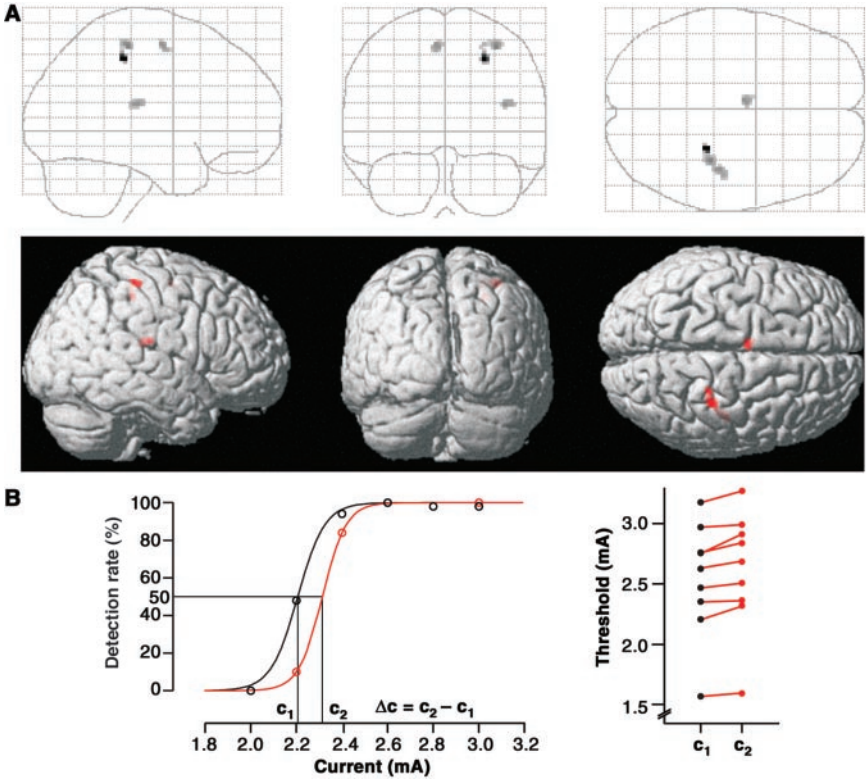

Fig. 1. (**A**) Subliminal stimuli "deactivate" somatosensory cortices (fMRI): S1 (X = 26, Y = –34, Z = 48), S2 (X = 40, Y = –24, Z = 18), and SMA (9) (X = –6, Y = –8, Z = 58); random effects analysis, $P < 0.001$ uncorrected for multiple comparisons due to a strong a priori hypothesis. (**B**) (Left) Characteristic individual detection curves for near-threshold stimuli [logistic fit; with (red) and without (black) subliminal stimulation]. (Right) Somatosensory thresholds are increased ($P < 0.02$, paired Student t test) during additional subliminal stimulation (c_2) by 2.6 % (0.065 mA ± 0.015 mA; mean ± SEM) compared with control (c_1).

References and Notes

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Materials and Methods
Fig. S1

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