Introduction. Electrical stimulation of the cerebral cortex is typically delivered through intracortical electrodes. In the context of brain-machine interfaces (BMIs), intracortical stimulation has emerged as a method to deliver "sensory" feedback signals directly into the brain, potentially bypassing an injured afferent pathway, or to induce activity-dependent cortical plasticity which could "re-wire" an injured cortical area. Epidural or subdural electrocorticography (ECoG) electrodes are less invasive alternatives to intracortical electrodes for recording brain potentials and they can also be used to deliver electrical cortical stimulation (ECS). However, the effects of ECS on the properties of the electrodes themselves, as well as on cortical activity have not been documented. **Methods**. In three nonhuman primates with subdural or epidural implants over sensorimotor cortex, repetitive, bipolar, single-pulse (0.2ms each phase) ECS was delivered for 30 minutes, at stimulation rates 2-5 Hz and at intensities at 80% of motor response threshold (MRT). MRT for a given electrode was defined as the minimum current intensity at which single pulses consistently evoked muscle activity (EMG recordings) or movement (wrist torques) of the contralateral arm or hand. Before and after stimulation, we determined, at the stimulated site (Cstim) and at sites at various distances from it: (1) electrode impedance, using impedance spectroscopy, a measure of the effectiveness of the electrode-tissue interface, (2) MRTs, a measure of cortical excitability, and (3) ECoG signal power at different frequencies, obtained from 10-20 minute-long recordings while the monkey was seated quietly in its chair, a measure of spontaneous cortical activity. **Results**: (1) ECS produced a drop in electrode impedance that persisted for 24-48 hours; the drop was specific to Cstim in epidural ECS, but also appeared in surrounding electrodes in subdural ECS (figure panel 1). (2) ECS caused a reduction in cortical excitability that manifested as an increase in MRT; that change was maximal at Cstim and became smaller at increasing distances from Cstim. MRT returned to pre-stimulation levels after 24 hours (panel 2). (3) ECS produced subsequent suppression of spontaneous ECoG power at low frequencies (<35 Hz) and enhancement at high frequencies (>40 Hz). Those signal changes, in all frequencies, showed a similar spatial distribution as those of MRT, and disappeared within 24 hours following stimulation (panels 3a, 3b). Changes in impedance, MRT and signal power were more pronounced with subdural ECS but their time-course was not different than those with epidural ECS. **Conclusion**. Repetitive ECS for as short as 30 minutes, at stimulation rates and intensities compatible with some BMI applications, produces changes in the electrode-tissue interface, cortical excitability and spontaneous cortical activity that outlast the duration of ECS and, while maximal at the stimulated site, also involve neighboring sites.