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GABAergic neuronal somata decrease at sites of focal epilepsy in monkey neocortex.

Previous studies have indicated that a loss of GABAergic terminals occurs at epileptic foci. The present study was undertaken to investigate if this loss is associated with a loss of GABAergic neuronal somata. Seven adult monkeys (m. mulatta) received alumina gel injections to the pre-central gyrus of the left cerebral hemisphere to produce epileptic foci. Four of these monkeys were chosen for further quantitative study. One was sacrificed prior to seizure onset ("pre-seizure"), one had seizures for three days ("acute"), and two had a seizure record of one month ("chronic"). Sections of tissue from the epileptic cortex and from the contralateral, non-epileptic cortex were processed for GAD immunocytochemistry at the light microscopic level. Quantitative analysis revealed that a loss of GAD-positive neuronal somata ranging from 24 to 52% occurred at epileptic foci for all monkeys. This decrease was significant ($p < .01$) for the two chronic monkeys. There was also a slight decrease in GAD-positive neurons 1 cm distal to the focus ("parafocus") in the chronic monkeys, but not in the acute or pre-seizure animals. GAD-positive somal areas were calculated for all four monkeys. These data were graphed as percentages of total cells in various size ranges at the focus and contralateral cortex. Small GAD-positive somata (50-150 μm^2) were more severely decreased in number at epileptic foci than larger ones (200-250 μm^2). As an experimental control, an additional monkey was given a surgical lesion in area 4 of one cerebral hemisphere. It did not display seizure activity prior to sacrifice and did not show a loss of GAD-positive neurons proximal to the control lesion. The results of this study indicate that a loss of GAD-positive neuronal somata is associated with a loss of GAD-positive terminals at epileptic foci, and that this loss may be more specific for small GABAergic neurons.

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