Fates of the Pyramidal Neurons in Neocortex After Callosotomy: Retrograde Neuronal Label with Two Fluorescent Tracers

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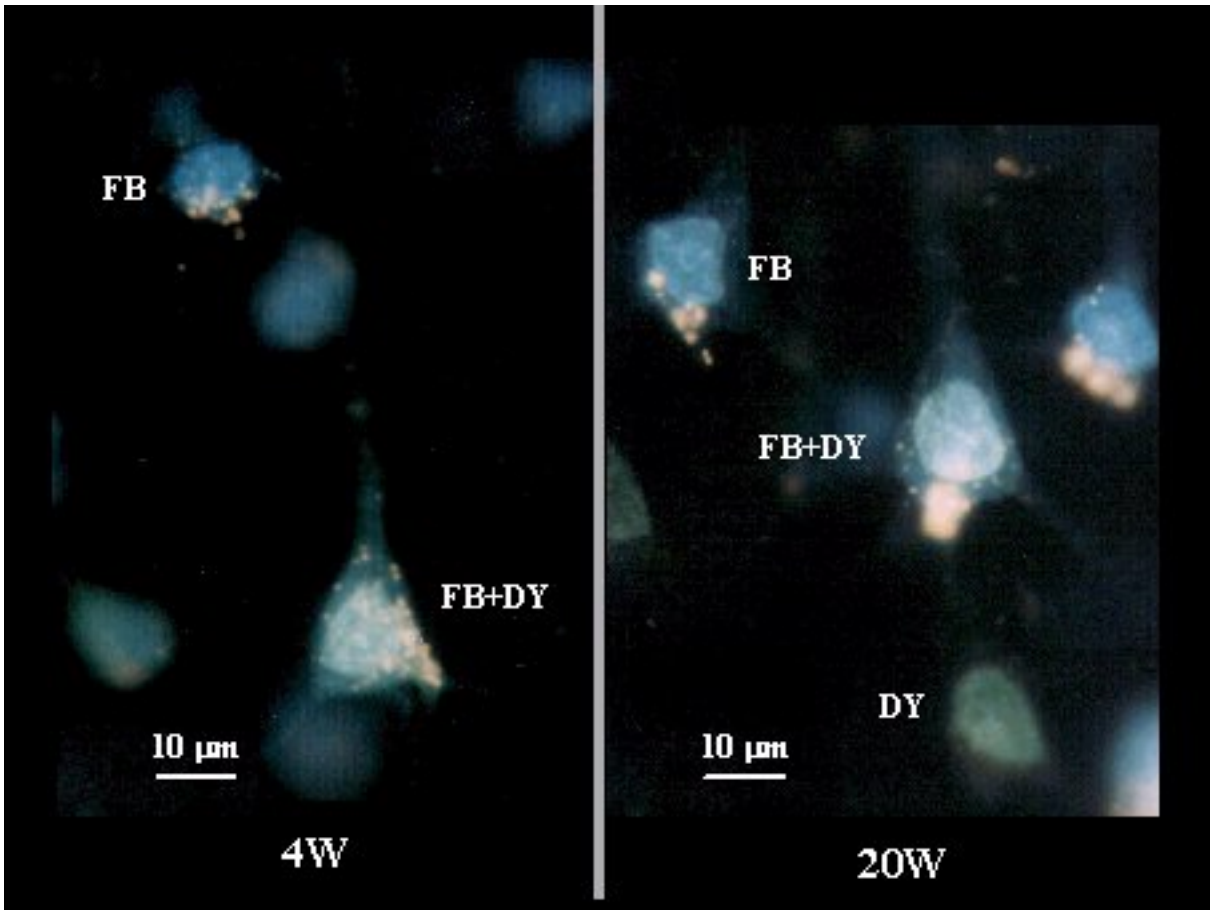
We studied the fates of the neocortical pyramidal neurons after callosotomy by using retrograd eneuronal labeling with two fluorescent tracers, fast blue(FB) and diamidinoyel1ow(DY). All procedures were performed under sodium pentobarbital deep anaesthesia. Male sibling Wistar rats underwent the first procedure at age 2 months; injection of 7% solution of FB unilaterally throughout the frontoparietal cortex via microsyringe with a 22G needle. Two Weeks later, the rats were divided at random into two groups and underwent the second procedure; total cal1osotomy or sham operation. Four, 8, and 20 weeks later, each rat underwent the third procedure; inection of 2% DY into the primary motor area opposite where the FB was injected. Five days later, they were perfused transcardiacally with physiologic saline followed by 10% neutral formalin. The brain was immediately removed from the skul1. After fixation and cryoprotection, the brain was frozen, and serial coronal sections were cut. Sections were thawed in ice cold distilled water and immediately mounted on slides. They were then cleared in xylene and coverslipped with DPX. Sections were assessed under an Olympus fluorescence microscope equipped with filter system U, providing excitation light of wavelength 360nm. Representative slices of the somatosensory area were photographed.



FB did not migrate from the labeled neurons for a considerably long time. False positive staining within surrounding glia cells was scarcely observed. The number of FB 1abeled callosal neurons was relatively small and revealed patchy distribution. Degeneration and disappearance of labeled pyramidal neurons caused by callosotomy was not observed. There was no big diffrence between callosotomy and sham operation with regard to morphology, distribution, and lamination of FB 1abeled pyramidal neurons in the neocortex on the side opposite where FB was injected. Double labeling of neurons with FB and DY was observed. This demonstrated that some of the FB labeled pyramidal neurons that once had callosal axons retained physiologic activities, even though their callosal axons were transected, because they showed evidence of retrograde axonal transport of DY via other collaterals from the ipsilateral primary motor area.

Transection of the corpus callosum in intractable generalized epilepsy would be expected to prevent generalization of epileptic discharges. Some successful cases have been

reported in which, after callosotomy, epileptic discharges localized unilaterally, and clinical seizures decreased in frequency, duration, and severity. The details of neuropathologic mechanisms of callosotomy have not been investigated. We had surmised that after callosotomy, neurons whose axons were cut might degenerate and disappear immediately. But we could recognize from our results that these neurons retained their morphology and their physiologic activity.

We concluded that after callosotomy, those neurons that once had axons to the opposite side of neocortex via the corpus callosum do not disappear immediately but maintain physiologic activity. Thus it might be assumed that these neurons may themselves be involved in reorganization of local neuronal circuits.
