Regional cerebral metabolism was examined with PET in 16 patients with extrapyramidal degenerative diseases (7 chorea, 7 Parkinson disease and 2 dystonia) manifesting involuntary movements. F-18 FDG, O-15 H2O and O-15 O2 were used in 11, 7 and 5 studies, respectively.

Among 7 patients with chorea, 6 showed decreased rCMRGlc values in the bilateral striatum, ranging from 4.00 to 5.81 mg/min/100ml. In the remaining patient, areas of hypometabolism were confined only to the bilateral caudate nuclei. Abnormal areas other than the basal ganglia were also observed in 6 patients, and 3 of them showed decreased rCMRGlc values in the entire brain.

Two of 4 patients with hemiparkinsonism showed hypometabolism in the unilateral caudate nuclei on the contralateral side in the reference to the clinical symptoms. However, in the remaining 5 patients with Parkinson disease, no abnormalities were seen in the caudate nuclei.

In 2 patients with dystonia, areas of hypometabolism were observed not only in the basal ganglia but also in the other sites.


The reduction in benzodiazepine receptors associated with Purkinje cell degeneration in "nervous" mutant mice have been reported, and the results have been interpreted as an indication of the localization of benzodiazepine receptors on Purkinje cells. And it has been described that the most conspicuous change in the cerebellar cortex of the autopsied brain of the patients with the olivo-ponto-cerebellar atrophy (OPCA) is loss of Purkinje cells. So we performed the positron emission tomographic study of benzodiazepine receptors in 5 OPCA patients and 6 healthy male, age-matched volunteers and compared the brain kinetics between two groups.

The results indicated that benzodiazepine receptor binding was normal or slightly increased in the cerebellum in the OPCA patients compared with the controls. The mechanism underlying the reduction of Purkinje cell without the reduction of benzodiazepine receptors in the OPCA patients is not unknown, but may be explained by the hypothetical compensatory mechanism, in which benzodiazepine receptors in the intact cell in the cerebellum increases.