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 decrease 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.

Effects of clonazepam to the brain kinetics of carbon-11 labeled Ro15-1788. To date, the clinical pharmacological studies have been performed measuring the blood concentration of drugs. However, the recent development of positron emission tomography and the appropriate ligands labeled with positron emitter has made it possible to assess the effects of various drugs to the neuro-receptors in the brain.

We investigated the effects of clonazepam which had been taken orally to the brain kinetics of carbon-11 labeled Ro15-1788 in 6 male volunteers and compared the physiological effects of clonazepam. When the subjects had taken clonazepam (30-50 ug/kg), the initial brain uptake of carbon-11 labeled Ro15-1788 was not altered but the wash-out of carbon-11 became faster and the radioactivity in the brain was reduced at the later time of the study, compared with the control experiment. After the PET study, the event-related potential was measured and the prolongation of the latency of P300 was observed when the subjects had taken clonazepam. And the reduction of brain uptake of carbon-11 at 30 min after injection and the prolongation of P300 was correlated well. The result suggest that the benzodiazepine receptor occupancy in the brain is a good indicator of physiological response of benzodiazepines including clonazepam.