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Mechanism of Brain Atrophy in Alcoholics

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Introduction: Morphological abnormalities of extensive nerve cell death have been mainly observed in cerebral cortex, hippocampus and cerebellum with a long-term use of alcohol.**Objectives & Aims:** We try to to investigate the mechanism of the brain atrophy in alcoholics. **Methods:** We reviewed consecutive CT examinations in 170 patients and 17 controls (from 23 to 85 years old) with symmetrical intracranial ventricular system and normal density of the white and gray matter. **Results:** There was a significant difference ($p<0.05$) in the BCI between controls and patients of all ages. There was no significant difference of the BCI in ages between 20s, 30s and 40s of patients. And there was a significant difference ($p<0.05$) in each generation between the young ages (20s, 30s and 40s) and the older ages (the 50's, 60s, 70s and 80s). There was no significant difference of the BCI between the generation of 20s, 30s and 40s. But the significant difference ($p<0.05$) between the young and the older generations was observed. And the generation of 50s is considered as the start point of the physiological brain atrophy. We consider that the spasmodic drinking might be due to the abnormal excitation of neural circuit. It has been reported that there is a correlation between the progression of brain atrophy and the seizure frequency and its lateralization in temporal lobe epilepsy. **Conclusions:** We hypothesized that the spasmodic drinking might be caused by abnormal excitation of neural circuit and the higher the frequency, the more brain atrophy progresses.

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