Inhibitory-Excitatory Unbalance Mechanism after Traumatic Brain Injury

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EDITORIAL

Neuropsychologic impairments after traumatic brain injury (TBI) involve personality, memory, executive functions, visual spatial functions, language, emotion, and attention [1-4]. Reduced information processing speed is one of the most fundamental cognitive deficits, which may be essential in producing the attentional and memory disturbances [5-7]. This issue is famous in popular contact sports including American football [8-11], boxing [12,13] and rugby [14,15]. Very mild head trauma like heading actions at soccer has no relation to neuropsychological problem [16,17] while more severe brain injury including repeated concussion induces severe cognitive dysfunctions. It is important for not only sports players but also so many other TBI patients to be diagnosed and treated appropriately because the deficits make their and their familial outcome strongly exacerbated [18,19]. However to date there is no effective medication against those symptoms because the mechanism is not entirely clear.

Clinically it is difficult to predict them by conventional computed tomography (CT) and magnetic resonance imaging (MRI) because they do not necessarily have relation to injured brain region though some region can worsen them [20]. White matter estimation as to directly fornix, corpus callosum or internal capsule and indirectly ventricle size on MRI is useful for severe injury in relation to cognitive outcome [21]. However their changes induced by atrophy are results from severe TBI and not predictive. Therefore recent functional brain imaging methods are expected to detect the deficits correctly and chronologically. They include single-photon emission computed tomography (SPECT), positron emission tomography (PET) and functional MRI (fMRI). It is revealed that functional anterior neuronal circuit which is consist of dregulate cortex (Cg) and dorsolateral prefrontal cortex (PFC) takes a significant role as to post-TBI neuropsychologic symptom [22-24].

Recent transcranial magnetic stimulation (TMS) with EEG study reveals hyper excited cortex in PFC after TBI, which may indicate excessive glutamate accumulation that leads to NMDA-mediated excitotoxicity and excess in GABA-mediated inhibition that contributes to lasting cognitive deficits inhibitory [25-28]. Interestingly these two molecular mechanisms seem to be contradictory. However the time course, chronological histology and physiologic interaction between excitotoxicity and excessive inhibition are not shown. In PFC cortical inhibition related to GABA is transiently absent after TBI and changes in plasticity mechanism may occur [29]. The inhibitory-excitatory unbalance may be induced at that time.

GABAergic interneurons in cortex are divided into at least four categories: (1) Parvalbumin (PV) cells, which are fast-spiking cells, (2) somatostatin cells, which are late-spiking cells, (3) calretinin (CR) and/or vasoactive intestinal polypeptide (VIP) cells and (4) cholecystokinin cells (almost negative for VIP/CR) [30,31]. In cortical plasticity PV cells take important role compared to other types while they are most frequent GABAergic interneurons in cortex. It is possible that some of PV cells may change their character after TBI and take significant role in changed inhibitory-excitatory balance or plasticity.

REFERENCES


