PEMF in the treatment of PCS

Inflammatory response following TBI or concussion is a crucial issue. It is signaled by a rapid rise in the levels of interleukins such as interleukin, interferons, and tumor necrosis factor alpha [1,2]. The cytokine IL-1β is an essential mediator in the neuroinflammatory response and is upregulated minutes after a neuronal insult or TBI / concussion [3,4,5,6] and has a nine-fold increase four hours after injury. Inhibition or neutralization of these deleterious secondary injury mechanisms generally results in neuroprotection. Many studies relate to TBI respectively ischemic accidents, but it is considered verified that IL-1 beta occurred equally by an head injury [7,8]. This is of considerable relevance because a mild traumatic brain injury does not predict acute postconcussion syndrome (PCS), since there is a high rate of acute PCS in both mTBI and non-brain injured trauma patients. PCS was not found to be specific to mTBI. The use of the term PCS may be misleading as it incorrectly suggests that the basis of PCS is a brain injury [9,10].

PEMF on Post-Concussive Syndrome (PCS)

For some time scientists have known that PEMF can reduce inflammation after soft tissue injuries [11,12,13] and inflammation -induced edema [14]. Noteworthy, PEMF also can modulate cytokine production. In an orthopedic study PEMF reduced IL-1 alpha activity by 10-70 % respectively 10- 80 % in human cells [15]. Or the release of anti-inflammatory cytokines in tendon cells treated with PEMF for 8 and 12 h was significantly higher in comparison with untreated cells, while the production of pro-inflammatory cytokines was not affected [16]. And the finding as well that PEMF-treatment reduces the IL-1ß concentration by around 50 % in the wound exudates after surgery shows a link to the inflammatory response [17].

Regarding TBI / concussion PEMF treatment attenuated IL-1ß levels up to 10-fold in cerebrospinal fluid within 6 hours after contusive injury and also significantly suppressed IL-1ß within 17 - 24 hours after penetrating injury [18]. This is also remarkable, because neutralization of IL-1ß results in significant reduction of TBI-induced brain necrosis, suggesting that IL-1ß contributes to neuronal death (Sprague Dawley rats) [19,20].

We also know that PEMF affects neurogenesis and neural recovery under normal or pathological conditions [21] . PEMF exposure also accelerated the recovery of the blood-brain barriere after brain injury [22]. Although the protective role of PEMF in brain trauma has been indicated, the biological mechanism to excitotoxicity at the cellular and molecular levels that could explain the therapeutic effects of PEMF have not been clearly indentified .

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