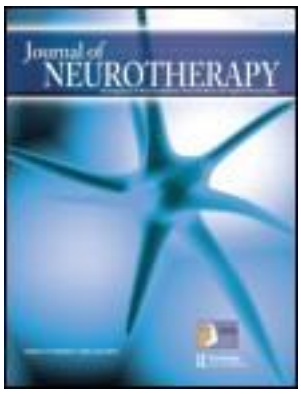

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Introduction: Blood Flow Hemoencephalography

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Introduction: Blood Flow Hemoencephalography

Tim Tinius, PhD

This special issue of the *Journal of Neurotherapy* entitled *New Developments in Blood Flow Hemoencephalography* is a presentation of new literature on a technique developed directly from the field of neurotherapy or neurofeedback. The technology to provide instant real time feedback of electrical changes in the brain has long been established. Dr. Toomim has spent the last several years creating and developing this technology called blood flow Hemoencephalography or HEG. Neurofeedback with this technique gives clinicians and researchers another method to measure, quantify and, most importantly, teach a person to change a measure of brain functioning via self-monitoring. I remember my first introduction to this technique at the 1997 Society for the Study of Neuronal Regulation (SSNR) Conference in Snowmass, Colorado. Immediately, I wondered "Is this really a valid and reliable measure of brain functioning?" Fast forward seven years to this special issue and my questions are answered. This special issue brings you a variety of studies that explain the HEG technology, describe the validity and reliability of the technology, describe many applications of HEG and most importantly, show this technique can be applied to a wide variety of central nervous system symptoms that many clinicians in the International Society for Neuronal Regulation (ISNR) see on a daily basis.

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A commonality that I see among the persons receiving treatment described in these special issue articles is a problem of underlying arousal. A change in arousal and function can be seen in athletes who have sustained a concussion during a sport. A recent review of the literature on concussion or mild traumatic brain injury in sports suggested that after a concussion axonal fibers are stretched and become swollen, beaded or varicose (Echemendia & Julian, 2001). The neurochemical and metabolic changes after a concussion begin within the first hour of insult and continue for up to 10 days post injury as the neurons are alive, but exist in a vulnerable state (Hovda et al., 1990). This vulnerability is characterized by an increase in the demand for glucose (fuel) and a reduction in cerebral blood flow (fuel delivery) and as a result, the neurovascular system is rendered unable to respond to demands for the energy required to return to normal neurochemical and ionic environments (Hovda et al., 1999). The duration of decreased cerebral blood flow may be a cardinal factor in predicting outcome of persons with mTBI (Hovda et al., 1999). There is a strong relationship between the concentration of Ca^{2+} and regional control of cerebral blood flow as those regions of the brain exhibiting Ca^{2+} flux show reduced cerebral blood flow after traumatic brain injury (Wojtys et al., 1999). Although the exact mechanism of Ca^{2+} flux after a concussion remains in question, it is quite clear that cerebral concussion may not, in and of itself, produce extensive neuroanatomic damage, but the surviving cells are placed in a state of vulnerability best characterized in terms of a metabolic dysfunction (Wojtys et al., 1999). The cascade of events leading to this dysfunction is multidimensional, resulting initially in acute periods of hyperglycolysis, followed by a chronic period of metabolic depression (Wojtys et al., 1999). In general terms, this dysfunction may be thought of as a breakdown in the harmony between energy demand, production, and delivery of oxygen (Wojtys et al., 1999). This energy crisis after traumatic brain injury was documented years ago (Jaggi, Obrist, & Gennarelli, 1990). In recent studies, changes in cerebral blood flow (CBF) measured on days one through five post-injury correlate positively with long-term neurological outcome (Kelly et al., 1997). Further, older age and more severe injury (as defined by pupillary abnormalities and incidence of evacuated subdural and intracerebral hematomas) are the most important clinical factors associated with low CBF during this period. These findings suggested the longer the duration of reduced flow (determined by the number of observations in which CBF was less than 33 ml/100 g/minute) the worse the outcome (Kelly et al., 1997). The importance of these findings is clear as clinical guidelines for concussion management describe

changes in blood flow to account for the symptoms after concussion (Wojtys et al., 1999).

When I read the physiology and cellular changes from the concussion and sports research, I immediately notice that the guidelines do not talk about active treatment. The current treatment of concussion is passive treatment of medication monitoring, no treatment, removal of activity, or education (Wojtys et al., 1999). After I read the articles in this special issue, I concluded that HEG neurofeedback may have promise as an active treatment in the alleviation of symptoms in athletes with a concussion. Research suggests that athletes with a concussion experience a blood flow crisis, but now there is technology that could assist in the resolution of this crisis and alleviate symptoms very soon after a concussion. Future research with the use of blood flow HEG neurofeedback in the clinical population of the concussed athlete can be a key to the development of HEG neurofeedback or neurotherapy. As you read this special issue on blood flow Hemoencephalography (HEG), I would strongly suggest that you develop research ideas, collect research data, and publish the results of clinical populations where this technology was used and shown to be effective.

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