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PET Imaging of Mild Traumatic Brain Injury and Whiplash Associated Disorder

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Chronic whiplash-associated disorder

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Chronic whiplash-associated disorder

We read the Article from Zoe Michaleff and colleagues (July 12, p.133)[1] on different physiotherapeutic regimens in chronic whiplash-associated disorders (cWAD) with great interest. Any treatment concept must be a trial-and-error process as long as the mechanism of action is not understood. How can an injury caused by a low velocity accident trigger such a broad variety of symptoms? The many positron and single-photon emission tomography studies of the brain and cervical soft tissue in patients with cWAD are inconclusive in terms of the mechanism of action of this disease. They only show indirect effects—i.e. the reaction to the trauma (musculoskeletal inflammation or hypoperfusion of the posterior parietal occipital region), but not the origin.[2,3]

There are three main hypotheses regarding the origin: first, is the hypothesis that whiplash does not exist. The second is the nociceptive-vascular hypothesis. According to Moskowitz and Buzzi,[4] there is a widespread effect on local vasoactive peptides and the cranial vascular system, caused by stimulation of pain-sensitive afferents in the trigeminal system. The third hypothesis is the mid-brain hypothesis. Vállez García and colleagues[5] reported that there is a mismatch between aberrant information from the neck muscles and the vestibular and visual systems, which is integrated in the mesencephalic periaqueductal gray and adjacent regions.

The fact that there is no accepted concept or proof for what causes the symptoms leads to endless discussion about this condition. Moreover, as long as we treat only some aspects of the various symptoms of this syndrome there will be no substantial treatment effect either.

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