Whiplash Syndrome- a disorder of the brain?

Jennifer Biendara BSc, Andreas Otte MD

Division of Biomedical Engineering, Faculty of Electrical Engineering and Information Technology, Offenburg University, Offenburg, Germany . andreas.otte@hs-offenburg.de

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Abstract

In the past two decades much has been published on whiplash injury, yet both the confusion regarding the condition, and the medicolegal discussion about it have increased. In this paper, functional imaging research results are summarized using MRIcroGL 3D visualization software and assembled in an image comprising regions of cerebral activation and deactivation.

Introduction

In the past two decades much has been published on whiplash injury, also some parts in the HJNM [1, 2, 3], yet both the confusion regarding the condition, and the medicolegal discussion about it have increased. The existing publications come from many different perspectives and from differing medical and non-medical disciplines: clinical, physical, mechanistic, cervical and cerebral imaging, neuropsychological testing, and various treatment approaches on a rather trial-and-error principle with no groundbreaking success, as the mechanism of action is still not clear [4]. The common denominator of most of these studies is their production of controversy: on the method, on the existence of the condition, on the diagnosis and on treatment. This controversy on the phenomenon whiplash is not new; see the British term "railway spine" that was established already in 19th century for patients who had developed posttraumatic symptoms after railway accidents [5, 6].

The whiplash hypotheses

What is whiplash injury, what is the acute and chronic whiplash syndrome, and is whiplash a real disease or only a fiction?

In terms of the hypotheses for the mechanisms of whiplash there are, up to date, mainly three: a) The "whiplash is not existent" hypothesis considering the chronic whiplash as a myth or rather a pseudosyndrome that is not related to the injury and cannot be determined using medical investigations [7]; b) The nociceptive-vascular hypothesis [8]. Widespread effects were revealed on local vasoactive peptides and the cranial vascular system by experimental stimulation of pain-sensitive afferents in the trigeminal system [9]; and c) The midbrain hypothesis yielding a mismatch between aberrant information from the cervical spinal cord and the input from the vestibular and visual systems, which are integrated in the mesencephalic periaqueductal gray and adjacent regions [4, 10, 11].

Functional imaging studies

On the central nervous system (CNS) level, functional imaging (i.e., nuclear medicine) data were gathered from patients with whiplash syndrome-many small-scale studies documented that subjective complaints result in deactivation and activation of certain brain regions, though the regions described by different research groups vary to a huge amount, yielding hypo- and hyperperfusion, also in adjacent brain regions, yet something that makes the understanding of the mechanism even more inscrutable.

To demonstrate this, these research results were summarized using 3D visualization software (MRIcroGL) based on an implemented magnetic resonance imaging template and assembled in an image comprising regions of cerebral activation (hyperperfusion/hypermetabolism) and deactivation (hypoperfusion/hypometabolism) [18]. For this, study results from Otte et al., (1995-1998) shown in blue color [9, 12-14], Bicik et al., (1998) shown in red [15], Lorberboym et al., (2002) shown in yellow [16], Linnman et al., (2009) shown in green [17], and Vállez García et al., (2016) shown in violet [4] were included [18].

The obtained Figure 1 - as we think - speaks a thousand words. Whereas there seems to be a "consensus" on deactivation of the posterior parietal occipital region at both brain sides by a number of studies, heterogeneity can be seen in the other regions. Interestingly, the focus of the older studies was only on deactivation, whereas only the more recent studies also looked after regions that are activated. It is, however, not excluded that the data of the older studies could be revisited to tease out the latter situation and thus may help to better understand the condition of whiplash.

Many patients with whiplash syndrome had to give-up their normal social lives, partnerships and jobs due to persistent

peripheral and cerebral symptoms. Not always are only insurance claims an issue, but often their problems are subject to litigation or pushed on to the psychological level.

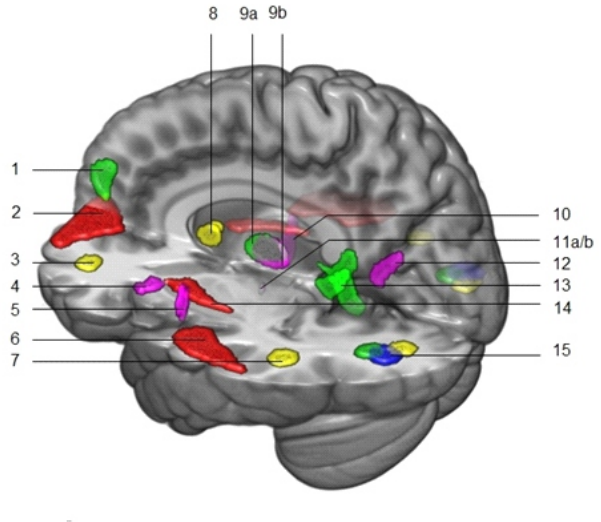

Figure 1. Summary of functional imaging study results over the past 20 years comprising hypometabolism and hypermetabolism in whiplash patients. MRIcroGL 3D visualization adapted from Biendara, 2017, with kind permission. The numbers indicate different conditions of cerebral activation in various regions: 1 - increased tracer uptake in the right medial prefrontal cortex; 2 -decreased tracer uptake in the fronto-polar region; 3 - decreased tracer uptake in the frontal lobe; 4 - decreased tracer uptake in the left frontal inferior gyrus; 5 - decreased tracer uptake in the insula at both sides; 6 - decreased tracer uptake in the anterior temporo-lateral cortex at both sides; 7 - decreased tracer uptake in the temporal lobe at both sides; 8 - asymmetric perfusion in the basal ganglia; 9a - increased tracer uptake in the right thalamus (Linnman et al., 2009); 9b - decreased tracer uptake in the right dorsomedial thalamus (Vállez García et al., 2016); 10 - decreased tracer uptake in the right superior temporal gyrus; 11a - increased tracer uptake in the gyrus parahippocampalis posterior at both sides (Linnman et al., 2009); 11b - increased tracer uptake in the right gyrus parahippocampalis (Vállez García et al., 2016); 12 - increased tracer uptake in the right superior gyrus cinguli and the right precuneus; 13 - increased tracer uptake in the posterior gyrus cinguli at both sides; 14 - decreased tracer uptake in the putamen at both sides; 15 - decreased tracer uptake in the posterior parietal occipital (also: parieto-occipital) region at both sides. Visualized study results are from Otte et al., 1995-1998 (shown in blue color), Bicik et al., 1998 (shown in red), Lorberboym et al., 2002 (shown in yellow), Linnman et al., 2009 (shown in green), and Vállez García et al., 2016 (shown in violet).

The most frequent symptoms in patients with whiplash syndrome are neck pain and headache, followed by visual and vestibular problems, cognitive limitations or emotional disturbances [11, 18]. These symptoms were mostly caused by rear-end vehicle collisions while only 50 % of the injured recover from initial symptoms within one year after the accident [19]. Interestingly, the most frequent clinical symptoms of whiplash syndrome (i.e., visual problems such as impaired spatial ability or blurred vision and cognitive limitations such as the difficulty in forming thoughts and difficulties not in attending but in disengaging attention once the subject has focused on an object of attention [20]) fit to hypoperfusion in the posterior parietal occipital region [4].

Basic research, translational and clinical studies focusing on various aspects or viewpoints, and meta-analyses on what is known, what can be learnt and what has to be discussed, especially from the functional neuroimaging studies, are still urgently needed.

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