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The plasticity of whiplash theories

In a small, retrospective study of non-consecutive, highly select, so-called whiplash and head injury patients, without an examination of pre-accident medical charts or use of adequate control population, and without psychometric effort tests to validate symptom reporting, Radanov et al. [1] seem to have once again modified their theories without being able to validate them according to currently available data.

Radanov, the lead author, believes that lack of sufficient initial analgesia is the potential basis for central desensitization and chronic pain. That is an interesting shift and represents the latest in a dramatic alteration in philosophies. We can all agree that there is likely an acute lesion (the nature of which may well vary) in most whiplash patients, even those who do not have neurological injury or fracture. Dr. Radanov, however, used to also believe in the necessity of a phenomenon wherein that acute lesion renders ongoing

pathology that in itself is the primary basis for symptom reporting. In trying to defend that myth, he used to write that "...findings appear to suggest that in some whiplash patients there may be an undetected disc (or other?) lesion" [2]. As an explanation for chronic pain, he also referred to autopsy studies and wrote that "...in a proportion of whiplash patients a soft tissue injury may be overlooked" [2]. He once also viewed whiplash as a "syndrome that may leave some treatable organic damage" and that "psychosocial factors are not primarily relevant for outcome" [3].

Dr. Radanov has eventually realized that sociocultural factors are chiefly, primarily, and quintessentially relevant for outcome, after he saw prospective studies from other countries break down his past theories [4]. These sociocultural factors do indeed determine the outcome, irrespective of the nature of the initial injury that is commonly labeled "soft-tissue injury."

Thus Dr. Radanov has come a long way, but now he believes the acute injury resolves, but that a failure of initial analgesia is the problem! This is contrary to scientific knowledge. Though no one has published any criticisms of the prospective (not retrospective as in the current study [1]) designs of studies that include headache evaluation in Lithuania [5], Greece [6], and Ger-

many [7, 8], Radanov et al. have not included these data in their considerations. In these better-designed studies (the Greek and German studies include detailed patient examination and analysis), we find that post-traumatic headache is very short-lived despite no special or unusually high usage of analgesia and otherwise minimal therapy. Radanov et al. must explain this fact without merely dismissing these data and failing to have insight into their own misconception, or they risk being labeled by the readership as grossly biased and unwilling to learn from others.

Considering that Germans, Lithuanians, and Greeks also have seven cervical vertebrae and accompanying tissues at risk for "central desensitisation" following the acute injury, Radanov et al. need to explain what sociocultural factors separate their patients from those in other countries. They need to explain what sociocultural factors encourage or incite central desensitization, a phenomenon that is perhaps better known as symptom amplification, a result of the environment accident victims find themselves in [9].

In light of the classical conditioning theory of chronic pain syndrome, the postulation of central desensitization by Radanov and coauthors seems unnecessary.

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