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CERVICAL SPINE DYSFUNCTIONS IN PATIENTS WITH CHRONIC SUBJECTIVE TINNITUS

To the Editor: We read with interest the paper by Michiels et al., who performed a study to characterize cervical spine issues in “Cervical Spine Tinnitus” (CST) (1). Our primary concern is the assumption that CST is a clinical entity. The theory behind CST is based on the supposition that aberrant cervical somatosensory information conveyed to the cochlear nucleus can cause tinnitus independent of cochlear hair cell loss or other auditory pathway pathology. Unfortunately, there is little scientific basis to this assumption in the literature. Additionally, causality or correlation between the tinnitus precept and cervical spine symptoms was not proven. This lack of causality is pervasive in the literature, as descriptions of neuronal connections between

the cervical spine and auditory pathways have only been shown in cadaveric studies in a single animal species (2). Moreover, there is an absence of literature that demonstrates tinnitus directly caused by cervical spine abnormalities in animals or humans. Accordingly, the concept of cervical spine tinnitus is purely theoretical and currently an unproven and unestablished diagnosis, which should be acknowledged by the authors.

Furthermore, the authors use the Neck Bournemouth Questionnaire (NBQ) as an indicator of cervical neck pathology. The NBQ was originally designed to measure biopsychosocial aspects of neck pain as a symptom inventory tool for clinical trials and outcomes research (3). Consequently, there is a potential for confounding when using the NBQ to rate neck symptoms in the setting of tinnitus, as many of the risk factors for neck pain, including psychological health conditions (4), are also risk factors for tinnitus (5–7). In particular, perceived stress and anxiety have consistently shown to be related to neck symptoms (8) and tinnitus (5). Thus, it is conceivable that anxiety could be a common causative factor for neck pain and tinnitus exacerbation, leading to higher NBQ scores and the labeling of the patient with a CST diagnosis. However, the authors did not control for these confounding factors, which may have contributed to the differences found between the two groups.

Another limitation of the study is the creation of a single subjective criterion for the diagnosis of CST, which relies exclusively on patient recall to associate the onset or exacerbation of tinnitus and neck symptoms. Given that the annual prevalence of neck pain can be as high as 50% (4); this diagnostic criterion requires further validation before its use in clinical studies.

The finding that a statistically equal number of CST and non-CST patients reported modulation of tinnitus during one of their physiotherapeutic tests is counterintuitive. Indeed, if we were to assume CST to be an independent and distinct clinical entity with an established cervical somatosensory pathophysiology, we would expect to see a higher incidence of manipulation-related tinnitus modulation in the CST group when compared to the non-CST group.

Cervical spine tinnitus is a hypothetical clinical entity in need of validation in studies to establish the relationship between spinal pathology and tinnitus. Caution should be exercised when suggesting that “[the] presence of cervical spine complaints can be a first indicator for the CST diagnosis (1).”

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**RESPONSE TO LETTER TO THE EDITOR:
“CERVICAL SPINE DYSFUNCTIONS
IN PATIENTS WITH CHRONIC
SUBJECTIVE TINNITUS”**

In Reply: We appreciate the interest of the authors in our study. Cervicogenic somatic tinnitus (CST) is indeed a frequently encountered challenge.

As the authors mentioned, the scientific background for CST is indeed based on animal studies. We add moreover to the concept of CST that several studies (1–3) have found positive effects of cervical spine treatment on tinnitus complaints. It was up to then unclear which cervical dysfunctions were present in these patients. Therefore, we aimed to investigate cervical spine dysfunction in patients with tinnitus (including CST and non-CST). Both were included because neck pain is indeed very common and can easily exist as a separate entity from tinnitus complaints. This is reflected in the equal percentages of reported neck pain in the CST and non-CST groups. The results of our clinical tests, carried out by a blinded rater, were however significantly different between both groups. This can be indicative of an underlying cervical dysfunction.

As stated in our article (4), the diagnosis of CST was made after a thorough ENT examination (including microotoscopy, audiologic assessment, and brain magnetic resonance imaging) to exclude other causes of tinnitus. In case no other cause was found, the CST diagnosis was made

based on all diagnostic criteria for CST (5). The association between onset or exacerbation of tinnitus and neck complaints was an important but not exclusive criterion for the diagnosis. Indeed, diagnostic criteria should be based on more than medical history. Therefore, our article (4) also suggests further research to provide more detailed and more objective diagnostic criteria. Within this framework, we can mention a recently accepted paper (6), where we elaborate on the potential diagnostic support of clinical cervical spine tests in CST.

The Neck Bournemouth Questionnaire consists of seven questions, of which two address perceived stress and depression. These can consequently influence the Neck Bournemouth Questionnaire scores. To take this into account, we used a 14-point cutoff. This rationale was applied previously in a sample of neck pain patients, where it has shown high sensitivity (83.3%) and specificity (90.9%) in identifying patients with neck disorders (7).

Possible explanations for the unexpected equal numbers of CST and non-CST patients to report modulation of the tinnitus were provided in the Discussion of our article (4).

Finally, as clearly stated in our article (4), we agree that caution is needed when using cervical spine problems as a first indicator for CST diagnosis because cervical spine dysfunction can also be present in non-CST patients.

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