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Review

Transcranial magnetic stimulation and subjective tinnitus. A review of the literature, 2014–2016

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ABSTRACT

Subjective tinnitus is a symptom in many ENT pathologies, for which there is no curative treatment. It may be poorly tolerated by some patients, who develop attention or sleep disorder or even major anxiety and depression, severely impairing quality of life. Pathophysiological models of the genesis and maintenance of tinnitus symptomatology highlight maladaptive cerebral plasticity induced by peripheral hearing loss. Although not fully elucidated, these changes in neuronal activity are the target of various attempts at neuromodulation, particularly using repetitive transcranial magnetic stimulation (rTMS), which has been the focus of various clinical studies and meta-analyses. A recent consensus statement (Lefaucheur, 2014) reported level-C evidence (possible efficacy) for rTMS using low frequency (1 Hz) tonic stimulation targeting the left cerebral cortex. However, many questions remain concerning the use of this technique in everyday practice. The present article reports a recent literature review using the search-terms "tinnitus" and "rTMS" in the PubMed and Cochrane databases for April 2014 to December 2016.

1. Introduction

Disabling subjective tinnitus is a frequent symptom, affecting 5–15% of the general population [1], with considerable socio-economic impact [2]. Although generally well tolerated [3], it can also severely impair quality of life [4] in some subjects, who show attention disorder [5,6], sleep disorder [7], hypersensitivity to noise [8], or even sometimes major anxiety and depression [9] caused by this parasitic auditory perception. Origin is usually multifactorial, and various pathophysiological models have been suggested to explain both onset and potentially harmful long-term consequences [10]. Most of these models are based either on abnormal neuronal activation [11] or on faulty reorganization of cerebral networks [12] involving the central auditory pathways (Heschl's gyrus), other non-auditory cortices such as the frontal cortex [13] or subcortical structures such as the limbic system [14]. These maladaptive phenomena are implicitly underpinned by brain plasticity mechanisms presumed to be triggered by a peripheral auditory lesion [15]. Consensually, subjective tinnitus usually results from a peripheral otologic lesion, either acute (acoustic or pressure trauma, drug toxicity, etc.) or chronic (chronic acoustic trauma, presbycusis, chronic otitis, vestibular schwannoma, etc.) [16]. Total or partial deafferentation caused by the hearing loss is supposed to underlie these self-sustaining cerebral dysfunctions. In the last analysis, these abnormal central reorganizations, of whatever type, constitute the neural correlate of tinnitus and of the resulting discomfort [17]. These pathophysiological models have much in common with those suggested to account for chronic pain syndromes, and especially the "phantom limb" pain [18]. Furthermore, it is unfortunately clear that there exists at present no curative treatment, and notably no drug therapy, providing reproducible and lasting resolution of tinnitus [19]. Although it seems reasonable to act first on the associated hearing impairment and treat any concomitant psychological or somatosensory disorders, management is basically palliative, seeking to alleviate the intrusiveness of the symptom and restore better quality of life. This, for example, is the aim of cognitive and behavioral therapies [20] and of the various techniques using hearing aids and acoustic enrichment to optimize auditory input [21] or mask the tinnitus [22] so as to facilitate natural adaptation and habituation. However, this situation is only partially satisfactory, both for patients and for the medical community in search of innovative treatment modalities [23]. In this context, various neuromodulation techniques have been proposed, to interact with cerebral dysfunction to achieve a more

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Table 1
Studies published between April 2014 and December 2016. Search-terms "tinnitus" and "rTMS" on PubMed and Cochrane databases.

Author	Year	Number of subjects	rTMS protocol (target, frequency of stimulation, number of stimuli)	Number and frequency of sessions	No data	Sham stimulation	Control group	Follow-up	Effect	Remarks
Wang et al. [32]	2016	289	Left temporal cortex, 1 Hz, 1000 stimuli per session	10 sessions, 5 days per week for 2 weeks	No	No	No	2 weeks	Significant (visual analog scale)	
Kreuzer et al. [33]	2016	37	Left temporoparietal junction, 1 Hz et left prefrontal cortex, 20 Hz, 1,000 stimuli per session associated to relaxation (including music)	10 sessions, 5 days per week for 2 weeks	No	Yes, but from a previous study	Yes, but from a previous study	10 weeks	Borderline versus placebo (questionnaires)	
Lehner et al. [34]	2016	49	Right and left temporoparietal junction, 1 Hz; left dorsolateral prefrontal cortex, 20 Hz; 1,000 stimuli per session per site, or left temporoparietal junction, 3,000 stimuli per session	10 sessions, 5 days per week for 2 weeks	Yes	No	No data	6 months	Significant (questionnaires)	No superiority for triple stimulation
Schecklmann et al. [35]	2016	23 (12 active, 11 sham)	Left temporoparietal junction, continuous theta-burst stimulation	10 sessions, 5 days per week for 2 weeks	Yes	Yes	No data	2 months	Borderline (questionnaires)	
Labar et al. [36]	2016	8	Left temporoparietal junction, 1,800 stimuli per session	5 sessions, 1 per week for 5 weeks, then 1 session per month for 5 months	No	No	No	6 months	Significantly reduced scores (questionnaires)	
Kreuzer et al. [37]	2015	40	Anterior cingulate cortex by double cone coil, 10 Hz, 2,000 stimuli; then left temporoparietal junction,1 Hz, 2,000 stimuli per session or left prefrontal cortex 10 Hz then left temporoparietal junction, 1 Hz, 2,000 stimuli per session	10 sessions, 5 days per week for 2 weeks	Yes	No	No data	3 months	Significantly reduced scores (questionnaires) but not superior to cingulate cortex stimulation	

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