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Cervicogenic somatosensory tinnitus: An indication for manual therapy? Part 1: Theoretical concept

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ABSTRACT

Tinnitus can be evoked or modulated by input from the somatosensory and somatomotor systems. This means that the loudness or intensity of tinnitus can be changed by sensory or motor stimuli such as muscle contractions, mechanical pressure on myofascial trigger points, transcutaneous electrical stimulation or joint movements. The neural connections and integration of the auditory and somatosensory systems of the upper cervical region and head have been confirmed by many studies. These connections can give rise to a form of tinnitus known as somatosensory tinnitus.

To date only a handful of publications have focussed on (cervicogenic) somatosensory tinnitus and manual therapy. Broadening the current understanding of somatosensory tinnitus would represent a first step towards providing therapeutic approaches relevant to manual therapists. Treatment modalities involving the somatosensory systems, and particularly manual therapy, should now be re-assessed in the subgroup of patients with cervicogenic somatosensory tinnitus.

The conceptual phase of this study aims to uncover underlying mechanisms linking the auditory and somatosensory systems in relation to subjective tinnitus through (i) review of the literature (part 1) and (ii) through design of a pilot study that will explore characteristics of the study population and identify relevant components and outcomes of manual therapy in patients with cervicogenic somatosensory tinnitus (part 2). This manuscript focusses the theoretical concept of (cervicogenic) somatosensory tinnitus, either with or without secondary central tinnitus or tinnitus sensitization.

1. Introduction

In clinical practice, tinnitus is often considered untreatable and many patients are familiar with the phrase “you have to learn to live with it”. The leading manual therapy journals have published very little on tinnitus, particularly (cervicogenic) somatosensory tinnitus (CeT) (Levine et al., 2007; Sanchez and Bezerra Rocha, 2011). Broadening the understanding of CeT would be a first step towards providing new therapeutic opportunities for manual therapists.

The subject ‘CeT’ is relatively new to the field of manual therapy. Therefore, the present study (in two parts) is presented as the first phase within a Medical Research Council (MRC) study framework (Campbell et al., 2007; Craig et al., 2008). This (conceptual) phase

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aims to uncover underlying mechanisms between the auditory and somatosensory systems of the cervical region in relation to tinnitus by reviewing the literature (part 1) and to explore patient characteristics and relevant outcomes of manual therapy in patients with CeT by designing of a pilot study (part 2).

2. Definition and prevalence of tinnitus

Tinnitus is defined as the perception of sound in the absence of external auditory stimulation (Hoekstra, 2013). The overall prevalence of tinnitus in adult populations ranges from 7% to 19%. The prevalence of tinnitus increases with age and seems to attain a plateau or even decrease at around 60–80 years (Henry et al., 2005). Within the group of treatment-seeking patients, the male-female ratio is 2:1. In up to 5% of the adult population, tinnitus interferes negatively with the ability to lead a normal daily life, and in 2%, it has a severe effect on daily life (Nondahl et al., 2002). The most common additional complaints are sleep problems, depression and anxiety (Zoger et al., 2006). Patients report limitations in activity and restrictions to participation in work and employment, and in social and civic life (Tyler and Baker, 1983). The distress can become so intense as to drive patients to suicide (Pridmore et al., 2012).

3. Classification of tinnitus

The first step in the classification of tinnitus is the definition of tinnitus as objective (can be heard by both patient and examiner) or subjective (only heard by the patient) (Zenner, 1998). Subjective tinnitus is very common and cannot be assessed objectively. A large number of underlying mechanisms have been described (Zenner, 1998; Shore et al., 2007, 2008; Baguley et al., 2013; Levine and Oron, 2015).

Subjective tinnitus can be subdivided into peripheral and central tinnitus (Zenner, 1998). See Table 1 for classification of tinnitus.

Peripheral tinnitus correlates with the anatomical region of the ear affected (outer, middle, or inner ear) and can be divided into conductive tinnitus and sensorineural tinnitus. Damage to the outer and middle ear causes conductive hearing loss and tinnitus, whereas inner ear damage results in sensorineural hearing loss and tinnitus. Tinnitus associated with the cochlear amplification mechanism of the outer hair cells is termed 'motor tinnitus' (sensorineural tinnitus type I). Tinnitus associated with the electromechanical transduction of the inner hair cells is termed 'transduction tinnitus' (sensorineural tinnitus type II). 'Transformation tinnitus' (sensorineural tinnitus type III) is associated with signal transfer from the inner hair cells and along the auditory nerve fibres. These sensory elements of sensorineural tinnitus (including amplification, transduction and transformation) can be supported by extrasensory disorders (e.g., circulatory disorders of the cochlea or endolymph hydrops). The remaining mechanisms of extrasensory, sensorineural tinnitus can be classified as sensorineural tinnitus type IV. It is plausible to classify the (cervicogenic) somatosensory type of tinnitus as extrasensory, sensorineural tinnitus type IV.

Central tinnitus can be divided into primary and secondary central tinnitus (Zenner, 1998). The pathogenesis of primary central tinnitus originates in the brain. The perception of tinnitus (first triggered peripherally but then manifesting itself in the brain independently of the original source in the ear) can be classified as either secondary central tinnitus or tinnitus sensitization (TS).

Table 1
Tinnitus classification.

1. Objective tinnitus
2. Subjective tinnitus
2.1. Peripheral tinnitus
2.1.1. Conductive tinnitus
2.1.2. Sensorineural tinnitus
2.1.2.1. Motor tinnitus (Type I)
2.1.2.2. Transduction tinnitus (Type II)
2.1.2.3. Transformation tinnitus (Type III)
2.1.2.4. Extrasensory tinnitus (Type IV) (e.g., cervicogenic somatosensory tinnitus [CeT])
2.2. Central tinnitus
2.2.1. Primary central tinnitus
2.2.2. Secondary central tinnitus (centralized tinnitus or tinnitus sensitization [TS])

Adapted with permission from Zenner (1998).

4. Somatosensory tinnitus

Somatosensory tinnitus is a type of tinnitus that can be evoked or modulated by inputs from the somatosensory and somatomotor systems (Levine, 1999; Levine et al., 2007; Shore et al., 2007, 2008; Sanchez and Bezerra Rocha, 2011; Dehmel et al., 2012; Levine and Oron, 2015). Loudness or intensity of tinnitus can be changed by sensory or motor stimuli such as muscle contractions, mechanical pressure on myofascial trigger points, cutaneous stimulation or joint movements. This type is called 'somatosensory tinnitus' and is based on the neural connections and integration of the auditory and somatosensory systems in the central nervous system (CNS) (Levine, 1999; Shore et al., 2007; Levine et al., 2007; Shore et al., 2008; Levine and Oron, 2015).

5. Cervicogenic somatosensory tinnitus

Within the group of patients with (chronic) somatosensory tinnitus, a subgroup of patients can be recognized in which tinnitus is related to changes in anatomical structures and physiological functions of the cervical region (Levine, 1999; Levine et al., 2007; Biesinger et al., 2008; Sanchez and Bezerra Rocha, 2011; Levine and Oron, 2015).

The existence of neural connections between the auditory system and the cervical region can be assumed based on a number of (animal) studies (Young et al., 1995; Kanold and Young, 2001; Zhan et al., 2006; Shore et al., 2007, 2008; Shore, 2011). The underlying principle is based on the convergence of auditory signals originating from the cochlea and somatosensory input originating from the face and the segmentally innervated structures of the cervical region (C1–C4) at the cochlear nuclei (somatosensory subpopulation of dorsal cochlear nucleus neurons), the caudal part of the spinal tract nucleus of the trigeminal nerve (V) and the external and middle ears via the common spinal tract of the facial (VII), glossopharyngeal (IX) and vagus (X) cranial nerves. Many ascending pathways from the cochlear nuclei are finally projected to the auditory cortex (Nieuwenhuys et al., 1988). A schematic model of the dorsal cochlear nucleus hypothesis in relation to (cervicogenic) somatosensory tinnitus is presented in Fig. 1 (Levine et al., 2007). Ongoing noxious activity in the somatosensory nucleus of the medulla to the dorsal cochlear nucleus pathways (e.g., after a whiplash injury or chronic neck pain) results in disinhibition of the dorsal cochlear nucleus and in increased tinnitus.

6. Chronic somatosensory tinnitus

The proposed mechanisms underlying the development of chronic somatosensory tinnitus are quite similar to events