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Reference:
Full text (Publishers DOI): http://dx.doi.org/doi:10.1016/j.math.2015.11.008

Rob A.B. Oostendorp, Iem Bakker, Hans Elvers, Emilia Mikolajewska, Sarah Michiels, Willem De Hertogh, Han Samwel

PII: S1356-689X(15)00233-7
DOI: 10.1016/j.math.2015.11.008
Reference: YMATH 1798

To appear in: Manual Therapy

Received Date: 15 September 2015
Revised Date: 23 November 2015
Accepted Date: 25 November 2015


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Cervicogenic somatosensory tinnitus: an indication for manual therapy?

Part 1: Theoretical concept

Rob A.B. Oostendorp$^{1,2,3}$, Iem Bakker$^4$, Hans Elvers$^{5,6}$, Emilia Mikolajewska$^{7,8,9}$, Sarah Michiels$^{10,11}$, Willem De Hertogh$^{10}$, Han Samwel$^{12}$

1. Department of Manual Therapy, Faculty of Medicine and Pharmacy, Vrije Universiteit Brussel, Brussels, Belgium
2. Scientific Institute for Quality of Healthcare, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands
3. Pain in Motion International Research Group (www.paininmotion.be), Vrije Universiteit Brussel, Brussels, Belgium
4. Practice for Manual Therapy, Arnhem, The Netherlands
5. Department of Public Health and Research, Radboud University Nijmegen Medical, Nijmegen, The Netherlands
6. Institute for Methodology and Statistics Beuningen, Beuningen, The Netherlands
7. Department of Physiotherapy, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland
8. Rehabilitation Clinic, Military Clinical Hospital No. 10 with Polyclinic in Bydgoszcz, Poland
9. Neurocognitive Laboratory, Center for Modern Interdisciplinary Technologies, Nicolaus Copernicus University in Toruń, Poland
10. Department of Rehabilitation Sciences and Physiotherapy, Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium
11. Department of Otorhinolaryngology, Antwerp University Hospital, Antwerp, Belgium.
12. Department Medical Psychology, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands

Corresponding author:

R.A.B. Oostendorp (e-mail address: rob.oostendorp@planet.nl)

Postal address:


Tel.: +31 246423419.
Keywords

Auditory-somatosensory interactions, neuroplasticity, somatosensory tinnitus, tinnitus sensitization, cervical spine, somatosensory stimulation, manual therapy

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 reassessed 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 practice test 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.
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Part 1: Theoretical concept

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 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 a practice test (part 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).

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; Shore et al., 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.

Insert Table 1.

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).

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; Shore et al., 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).

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; Shore et al., 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 Figure 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.
Insert Figure 1.

**Chronic somatosensory tinnitus**

The proposed mechanisms underlying the development of chronic somatosensory tinnitus are quite similar to events occurring in connection with chronic pain (Isaacson et al., 2003; Møller, 2007), particularly in patients with musculoskeletal disorders (Nijs et al., 2013). Chronic somatosensory tinnitus often originates in the CNS as secondary central tinnitus or TS (Zenner and Zalaman, 2004; Zenner et al., 2006; Zenner, 2006). Tinnitus will be perceived as a sound and is thus often referred to the ear. The common denominator for chronic pain and chronic somatosensory tinnitus is sensitization of the peripheral and CNS, including involvement of the sympathetic nervous system (Møller, 2000). The outcome of this process is an increased responsiveness to a variety of peripheral stimuli including mechanical pressure, light, sound, heat and cold. Once in this state, physiological sensory stimuli are potentially noxious. It is conceivable that under conditions of central sensitization, the sensitivity of sympathetic innervated cochlear hair cells is increased and that even in absence of a peripheral auditory signal, sound will be perceived and identified as noxious (Isaacson et al., 2003). These sounds may induce helplessness cognitions and subsequent anxiety and/or depression in patients due to lack of control and a sense of being unable to cope.

Chronic somatosensory tinnitus can be categorized within a group of overlapping conditions, such as chronic back pain, chronic neck pain, chronic whiplash associated disorders and chronic fatigue syndrome, united by a common pathophysiological mechanism of central sensitization (Nijs et al., 2013). Chronic somatosensory tinnitus can be associated with, but not uniformly characterized by, central sensitization. Clinicians should be aware of the potential presence of central sensitization in the form of TS in a subgroup of patients with chronic somatosensory tinnitus.

**Clinical criteria of cervicogenic somatosensory tinnitus**

Clinical criteria of cervicogenic somatosensory tinnitus are based on clinical expertise, clinical reasoning processes and guidelines describing diagnostic criteria for cervicogenic headache (CeH) (Sjaastad et al., 1990; Sjaastad et al., 1998), dizziness (CeD) (Wrisley et al., 2000) and tinnitus (CeT) (Sanchez and Bezerra Rocha, 2011) and consensus between the authors. The following clinical criteria were considered for cervicogenic somatosensory tinnitus for the practice test in part 2: (1) neck pain; (2) impairment of cervical range of motion, preferably rotation; (3) modulation of tinnitus by head and neck movements and/or posture; (4) tenderness of cervical-occipital muscles. The variables were dichotomized into yes / no. A case was considered positive (yes) for cervicogenic somatosensory tinnitus if 'yes' was given for all four criteria.

**Clinical criteria of tinnitus sensitization**

Based on clinical expertise and using guidelines for clinical recognition of central sensitization and TS (Zenner et al., 2006; Nijs et al., 2010) and consensus between the authors, the following clinical criteria were considered for tinnitus sensitization for the practice test in part 2: (1) widespread hyperalgesia and pain remote from the symptomatic region, such as shoulder pain and back pain; (2) impairment in quality of vision; (3) burning eyes; (4) modulation of tinnitus by psychological stress, such as sound phobia (fear of sound); (5) modulation of tinnitus by sensory stimulation; (6) presence of headache; (7) presence of dizziness; (8) tingling in arms or legs. The variables were dichotomized
into yes / no. Tinnitus sensitization was considered positive (yes) if ‘yes’ was given for five of the eight variables by analogy with the clinical recognition of central sensitization (Nijs et al., 2010).

**Treatment evidence**

Excluding medication, two main treatment approaches for tinnitus are available: i) sound-based therapies, such as tinnitus retraining therapy (Philips and McFerran, 2010), amplification with hearing aids (Hoare et al., 2014) and sound masking therapy (Hobson et al., 2012), and ii) cognitive behavioural therapies (Martinez-Devesa et al., 2010), including relaxation, exposure techniques and mindfulness-based training. Based on systematic reviews of randomized controlled trials, the efficacy of most interventions remains to be demonstrated conclusively (Hoare et al., 2011). Cognitive behavioural therapy has the strongest evidence-based support to date (Cima et al., 2014).

Management of tinnitus by manual therapy is currently supported by limited evidence (a few controlled and non-controlled studies [Levine et al, 2007] and some narrative reviews and case reports [Sanchez and Bezerra Rocha, 2011]). History taking and physical examination of the cervical spine and muscle functions are considered essential in the assessment of the utility of potential components of somatosensory tinnitus (Langguth et al., 2007).

**Manual therapy**

The first step towards providing the treatment option of manual therapy is an understanding of the underlying somatosensory mechanisms of the proprioceptive and nociceptive innervated structures in the head and neck region (joints, muscles, ligaments, fasciae, and spinal and peripheral nerves), and of dysregulated nociceptive processing in the CNS in relation to the hypersensitivity of the dorsal cochlear nucleus. These convergent anatomical and physiological connections between somatosensory systems and the dorsal cochlear nucleus system, in addition to peripheral and central sensitization, might explain the ability to inhibit the intensity of tinnitus and to decrease the sensitivity of the auditory system by non-noxious somatosensory treatment of the cervical region, particularly myofascial trigger points (Biesinger et al., 2008; Bezerra Rocha and Sanchez, 2007; 2012; Latifpour et al., 2009; Sanchez and Bezerra Rocha, 2011). See Figure 1. Non-noxious (preferably proprioceptive) activity in the somatosensory nucleus of the medulla to the somatosensory subset of the dorsal cochlear nucleus pathways results in inhibition of the dorsal cochlear nucleus and in decreased tinnitus. To achieve selective somatosensory mechanical stimulation of proprioceptive joint and muscle receptors, the therapist should repeatedly perform very gentle, low velocity passive articular movements in the spinal joints (including pelvic), and lengthening contraction exercises for the cranio cervical muscles.

The focus of manual therapy in patients with CeT is on musculoskeletal impairments (static posture [forward head position], restricted range of motion, dysfunctions of motion segments of the joints of the [cervical] spine, palpably painful upper cervical joints, impaired muscle function [tone, endurance, tenderness and trigger points], asymmetry in skin-fold tenderness, and head and cervical dyskinesthetic position sense) (Zito et al., 2006; Latifpour et al., 2009; Michiels et al., 2014; 2015; Levine and Oron, 2015).

It is common practice to provide information-based advice on pain, including pain education, to patients with chronic neck pain and to recommend and encourage active mobilizing and stabilizing exercises at home. Practice guidelines are also available on how to explain central sensitization to
patients with chronic musculoskeletal pain (Nijs et al. 2011; McKenna et al., 2014). The challenge now is to translate these existing guidelines, with the inclusion of the new science regarding TS, into forms that will help the manual therapist apply them to patients with CeT.

Conclusion

A number of studies have been pointing towards a neural connection between the dorsal cochlear nucleus and the somatosensory systems of the head and the high-cervical region, as the cause of tinnitus in some patients with (cervicogenic) somatosensory tinnitus. Broadening the knowledge and understanding of (chronic) somatosensory tinnitus might enable the use manual therapy in the assessment and treatment of patients with cervicogenic somatosensory tinnitus. Hence, it is necessary to design a practice test (part 2) to explore patient characteristics and outcome relevant to manual therapy.
References


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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)
Fig. 1. Schematic depiction of the anatomic and physiological basis for modulation of somatosensory tinnitus. 
+ = disinhibition of the somatosensory subset of the dorsal cochlear nucleus (increased tinnitus)
- = inhibition of the somatosensory subset of the dorsal cochlear nucleus (decreased tinnitus)
V = trigeminal nerve; VII = facial nerve; VIII = cochlear nerve; IX = glossopharyngeal nerve; X = vagus nerve
C1 – C4 = cervical spinal nerves
Adapted with permission from Levine et al., 2007; Levine and Oron, 2015.
**Highlights**

- Understanding of neural mechanisms underlying somatosensory tinnitus is the basics for application of manual therapy.
- Chronic subjective tinnitus combined with secondary central tinnitus is comparable with chronic pain with central sensitization.
- Manual therapy is a potential treatment in patients with cervicogenic somatosensory tinnitus.