Chapter 43
Tinnitus Caused and Influenced by the Somatosensory System

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Keypoints

1. It is now recognized that many forms of tinnitus-related neural activity are much more complex and multimodal than ever thought.
2. It has become evident that contribution of non-auditory pathways is involved in eliciting or modulating many forms of tinnitus.
3. Many forms of tinnitus can be modulated by different actions such as forceful muscle contractions of the head and neck as well as eye movements.
4. Somatosensory stimulation such as that from pressure of myofascial trigger points, cutaneous stimulation at specific locations, electrical stimulation of the median nerve and hand, finger movements, and orofacial movements can also modulate or cause tinnitus, as can pressure applied to the temporomandibular joint or lateral pterygoid muscle.
5. This chapter discusses the causes of somatosensory tinnitus and in particular the influence from both head and neck regions on the auditory pathways in individuals with tinnitus.

Keywords Tinnitus • Somatic • Somatosensory • Central nervous system • Muscle • Cervical spine • Temporomandibular joint.

Abbreviations

MTP	Myofascial trigger point
AMTP	Active myofascial trigger point
LMTP	Latent myofascial trigger point

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Introduction

For many years, tinnitus was thought to arise almost exclusively from abnormal neuronal activity within the auditory pathway. However, accumulated evidence suggested that tinnitus-related neural activity is much more complex and multimodal than previously anticipated.

More often than ever thought, tinnitus can be evoked or modulated by inputs from somatosensory, somatomotor, and visual-motor systems in some individuals. This means that the psychoacoustic attributes of tinnitus might be changed temporarily by different stimuli, such as:

- Forceful muscle contractions of head, neck, and limbs [1–3];
- Eye movements in horizontal or vertical axis [4–7];
- Pressure of myofascial trigger points [8];
- Cutaneous stimulation of the hand/fingertip region [4] and the face [9];
- Electrical stimulation of the median nerve and hand [10];
- Finger movements [11];
- Orofacial movements [12];
- Pressure applied to the temporomandibular joint or lateral pterygoid muscle [13, 14].

Such temporary changes are known as modulation of tinnitus. So, the contribution of non-auditory pathways has become more and more evident in eliciting or modulating existent tinnitus.

Although this phenomenon is yet to be fully understood, it seems to be clinical evidence of the existing neural connections between the somatosensory and auditory systems, whose "activation" may play a role in tinnitus. Anatomic and physiological findings in animal studies have shown that the trigeminal and

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dorsal root ganglia relay some afferent somatosensory information from the periphery to secondary sensory neurons in the brainstem, specifically, the spinal trigeminal nucleus and dorsal column nuclei, respectively [15]. Each of these structures sends excitatory projections to the cochlear nucleus. Mossy fibers from the spinal trigeminal and dorsal column nuclei terminate in the granule cell domain while en passant boutons from the ganglia terminate in the granule cell domain and core region of the cochlear nucleus. Single unit and evoked potential recordings in the dorsal cochlear nucleus indicate that these pathways are physiologically active.

So, these clinical findings strongly suggest that those who are able to modulate their tinnitus should be considered as a specific subgroup of patients. Among all types of modulating factors that have been described, we are particularly interested in the influence of both head and neck regions on the auditory pathways.

Now, there is yet no consensus on the definition of "somatosensory tinnitus," and this term has been used with different meanings. A group of researchers in the Tinnitus Research Initiative is presently working to define and differentiate "somatosensory tinnitus" (primary origin in head and neck trauma, dental or cervical manipulation, or even in unknown chronic pain) from "somatosensory modulation" (auditory origin with temporary somatosensory influence in loudness, pitch, or localization). Although many aspects still need clarification, we have already progressed in establishing some specific causes, methods of diagnosis, and treatment options to this subgroup, which will be described in this chapter.

Theories About Tinnitus Modulation

It is widely known that reorganization or re-mapping of specific central nervous areas occurs as a normal response of brain tissue to injury [16, 17]. However, as any double-edged sword, it is not possible to predict whether injury-induced plasticity will end up in limited or cross-modal effects, which in turn may result in compensatory or pathologic effects. Neuroplasticity is often implicated in tinnitus, and aberrant cross-modal plasticity seems to play a role in recently described cases of tinnitus evoked by somatosensory activation. This suggests that abnormal interaction between different sensory modalities, sensorimotor systems, neurocognitive, and neuroemotional networks may contribute to certain aspects of tinnitus [17].

Tinnitus modulation indicates that the psychoacoustic attributes of tinnitus change temporarily during some sort of stimuli [18]. Some of these modulation patterns (gaze-evoked, finger-evoked, and cutaneous-evoked tinnitus) were first described after acute unilateral total deafferentation of the auditory afferents, usually caused by the removal of skull base and posterior cranial fossa tumors. Some authors have hypothesized that in this form of modulation, important plastic changes occurred in the central nervous system after such deafferentation.

However, our own clinical experience showed that other types of modulation occur regardless of any surgical manipulation or degree of hearing loss [2, 3, 19]. An altered afferent input to the auditory pathway may be the initiator of a complex sequence of events finally resulting in the generation of tinnitus at the central level of the auditory nervous system. The effects of neural plasticity can generally be divided into early and later modifications, depending on the time of onset. Unmasking of dormant synapses, diminishing of (surround) inhibition, and generation of new connections through axonal sprouting are early manifestations of neural plasticity, resulting in lateral spread of neural activity and development of hyperexcitability regions in the central nervous system. The remodeling of tonotopic receptive fields within auditory structures (dorsal cochlear nucleus, inferior colliculus, and auditory cortex) seems to be a late manifestation of neural plasticity. The modulation of tinnitus by stimulating the somatosensory system might be explained by activating auditory regions through the non-classical pathway.

Tinnitus Modulation by Muscle Contractions

Sometimes tinnitus patients spontaneously report that contractions of head and neck muscle may change the loudness or pitch of their tinnitus. However, recent studies showed that a surprisingly large number of patients modulate tinnitus when they are specifically tested for it. Levine initially found that 68% of patients with tinnitus experienced some kind of modulation

when performing muscular contractions [1, 20]. Regardless of etiology or audiometric pattern, 71% could modify their tinnitus with a variety of cephalocervical isometric maneuvers or extremity contractions [21]. The head/neck isometric maneuvers were much more effective in modulating tinnitus than contractions of the limbs. Using a control group, Sanchez et al. pointed out that 65.3% of patients modulated loudness or pitch of their tinnitus during muscle contractions, while 14% of asymptomatic subjects could evoke tinnitus perception during the same maneuvers [2]. Later, other studies confirmed that the majority of tinnitus patients can modulate the phantom sound by stimulation of the somatosensory system [3, 19, 21, 22].

Considering the structure of the auditory pathway, it consists of several well-defined centers, although precise information about their interaction is still lacking. The cochlear nucleus is the first central nucleus of the auditory pathway, receiving information from the cochlear hair cells. In higher portions of the auditory pathway, the lemniscal system sends the received information to the primary cortical auditory areas, whereas the extralemniscal portion of the ascending pathways transmits auditory information to associated areas [10]. Many neurons of the extralemniscal system receive information from other sensorial tracts, such as the somatosensory system [23, 24].

The cuneate and gracile nuclei collectively form the dorsal medullary nucleus, whose position in the somatosensory system is analogous to that of the cochlear nucleus in the auditory system. It receives information directly from the dorsal roots, which in turn get information from the proprioceptive, tactile, and vibratory receptors of the body surface. The lateral cuneate nucleus is the end point of afferent fibers from the neck, ear, and suboccipital muscles, and carries information on head and ear position needed to process the acoustic information [25]. Because of reciprocal connections between the auditory and somatosensory systems, these authors postulated that projections from the cuneate to the cochlear nucleus may lead to excitation of the cochlear nucleus. Nevertheless, some electrophysiological studies in cats showed that the final effect of cuneate nucleus activation is the inhibition of the dorsal cochlear nucleus [26]. The exact mechanisms responsible for somatic modulation of tinnitus are currently unclear. If one considers that tinnitus results from aberrant neuronal activity within the auditory pathway, this could mean that somatosensory stimuli coming from head and neck muscle contractions might, through a multisynaptic pathway, disinhibit the ipsilateral cochlear nucleus, producing an excitatory neuronal activity within the auditory pathway that results in tinnitus.

As muscular contraction represents an activation of the somatosensory system, these anatomical connections between both systems might explain the influence of voluntary muscle contractions upon some types of tinnitus, thereby stimulating or inhibiting this symptom and presenting clinically as a modulation factor. In fact, we have seen patients with a typical history of acoustic trauma that could also clearly evoke tinnitus by several different stimuli, including during abdominal contraction.

Tinnitus Modulation Through Myofascial Trigger Points

Myofascial trigger points (MTP) are small hypersensitive spots located within the palpable taut bands of skeletal muscle fibers. Either spontaneously or under mechanical stimulation, they may cause local and referred pain [27].

MTP may be active (AMTP) when their stimulation causes a pattern of referred pain that is similar to the patient's pre-existent pain complaint or may aggravate such pain [28]. They are frequently found on the neck, shoulders, pelvic girdle, and masticatory muscles [29], where they provoke spontaneous pain or movement-related pain.

MTP can also be latent (LMTP), which are located in symptom-free areas and provoke local and referred pain only when stimulated [28].

Although MTP may be detected in pain-free subjects, they are typical of patients with myofascial pain syndrome, who often complaint of an associated tinnitus [30].

Travell and Simons first reported that MTP palpation of the sternal division of the sternocleidomastoid evoked a sound perception in a tinnitus-free patient [27]. Later, Eriksson et al. described a patient who noticed differences in tinnitus when palpating a MTP in the sternocleidomastoid. Such association has also been verified in studies where tinnitus patients had their conditions improved through anesthesia-based MTP deactivation [31].

Recently, Rocha et al. (2007) [8] investigated whether myofascial trigger points could modulate tinnitus and examined the association between tinnitus and MTP. They evaluated 94 subjects with tinnitus and 94 without the disorder, who underwent bilateral digital pressure of nine muscles of the head, neck, and shoulder girdle usually tested in myofascial pain syndromes (infraspinatus, levator scapulae, superior trapezius, splenius capitis, scalenus medius, sternal portion of sternocleidomastoid, posterior digastric, superficial masseter, and anterior temporalis). Temporary tinnitus modulation was observed in 56% of the subjects during digital pressure, mainly in the masseter, splenius capitis, sternocleidomastoid, and temporalis muscles. The rate of tinnitus modulation was significantly higher on the same side of MTP tinnitus subjects to examination in six out of the nine muscles. A strong association between tinnitus and the presence of MTP was observed, as well as a laterality association between the ear with tinnitus and the side of the body with MTP [19].

We initially assumed that only AMTP (related to pain) would be able to modulate tinnitus. However, the compression of LMTP may also end up with modulation of tinnitus. One possible explanation is that both active and latent MTP evoke referred pain when stimulated. Another interesting discovery of this study was the fact that MTP located in head and neck muscles produced more tinnitus modulation than those located in the shoulder girdle, which supports previous study [2, 20] findings, in which head and neck muscular contraction maneuvers produced more modulation than those of the members. These results can be possibly explained by neuroanatomy, since connections between somatic and auditory pathways at the cephalic level would be richer.

One of the mechanisms that explains referred pain is transmission by autonomic pathways [32]. The autonomic phenomena referred to other areas besides the MTP region can be explained by increased sensitivity of sensory nerve endings (thin terminal axons) at the MTP region and consequent neural mechanisms to spread referred pain [25]. Whenever those LMTP remain in a given subject for lengthy periods, they give rise to sensitization of nervous fibers associated to vasoconstriction due to increased sympathetic neurovegetative activity [33]. According to Hubbard and Berkoff, sympathetic activity explains the autonomic symptoms associated with MTP and provides a mechanism through which local injury and nociception cause local tension. It is now accepted that there is direct sympathetic innervation to the intrafusal fibers of muscle spindles. In some tinnitus patients, the sympathetic nervous system apparently plays an important role. Studies have found that blocking the sympathetic input to the ear or a sympathectomy can alleviate tinnitus in some patients. Thus, the autonomic nervous system (sympathetic) may explain some of the findings regarding the effects of MTP stimulation on tinnitus.

Thus, the possible explanation for the relationship between tinnitus and MTP would be not only somatosensory–auditory system interactions but also the influence of the sympathetic system.

Tinnitus Modulation During Tender Point Compression

Tender points are discrete areas of pain in response to palpation on body surfaces and can be identified in many people, but those suffering from chronic pain disorders tend to be more affected. The difference between MTP and tender points is the location of pain and the point of maximum tenderness that causes the symptoms. MTP refers pain to a distant spot upon pressure; tender points do not [34]. Researchers have been debating whether trigger points are a subset of tender points.

Even with such similarities, there has been no report of tender points being able to modulate tinnitus. However, during the examination of 11 patients with tinnitus and frequent regional pain for at least 3 months in the head, neck, and shoulder girdle (ten with myofascial pain syndrome and one with only tender points), we surprisingly found that 5 of them modulated tinnitus upon digital pressure on some tender points, besides the modulation by trigger points. Moreover, two other patients only modulated tinnitus by tender points, including the subject who did not have myofascial pain syndrome.

As this finding appeared by chance during the development of a study focused to trigger points, new clinical studies with bigger samples are necessary in order to demonstrate a possible relationship between tender points and tinnitus, with or without an associated myofascial trigger point.

Tinnitus Associated with Cervical Whiplash

As a consequence of cervical whiplash, extensive injuries to the cervical joints, ligaments, and discs may occur [35]. These bony and soft tissue injuries may lead to a variety of clinical manifestations [36]. Neck pain is the most common symptom, reported in 88–100% of cases [37]. Surprisingly, tinnitus and other otological symptoms are found in approximately 10–15% of the patients [38–40]. However, among 109 patients evaluated, none reported otological symptoms in the acute phase following the whiplash injury [41]. In our opinion, a possible explanation might involve the secondary vicious muscular postures that patients adopt in order to avoid neck pain. Considering the relation previously described between tinnitus modulation and muscular tension, myofascial trigger and tender points [8, 19, 31, 42], it is possible that secondary findings in patients with whiplash injury may justify the later onset of tinnitus. As the relationship between whiplash itself and tinnitus is yet controversial, caution is recommended whenever attributing these symptoms to such an injury.

On the other hand, some studies have suggested a possible link between whiplash and temporomandibular joint dysfunction [43–45]. Whiplash might induce joint lesions and posttraumatic malocclusions, which would lead to dysfunction of the masticatory muscle, resulting in tinnitus [46]. However, other researchers claim that temporomandibular joint dysfunction is not associated with whiplash injuries [47–49].

In short, although whiplash is considered a cervical spinal disorder, its relation with tinnitus is controversial. Furthermore, evidence of somatosensory modulation of tinnitus in such patients is not yet supported by the literature.

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