



CLINICAL AND NEUROPHYSIOLOGICAL CHARACTERISTICS OF PATIENTS WITH TINNITUS: THE ROLE OF TRIGEMINAL–SOMATOSENSORY INTERACTIONS

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Abstract.

Introduction. Tinnitus is a multifactorial condition that represents a significant clinical problem at the intersection of neurology and otorhinolaryngology. In recent years, increasing evidence has highlighted the role of non-auditory systems, particularly the somatosensory and trigeminal pathways, in the pathogenesis and persistence of tinnitus. The aim of this study was to evaluate clinical and neurophysiological features of patients with tinnitus, with an emphasis on trigeminal reflex excitability and its relationship to somatic modulation and tinnitus severity.

Materials and Methods. A total of 112 patients with subjective tinnitus were examined using neurological and otorhinolaryngological assessments, audiological testing, tympanometry, psychometric evaluation with the Tinnitus Handicap Inventory, and neurophysiological investigation including trigeminal evoked potentials. A control group of 20 healthy volunteers was included. Based on trigeminal evoked potential parameters, patients were divided into subgroups with high, moderate, and low reflex excitability.

The results demonstrated distinct neurophysiological patterns across subgroups, characterized by shortened or prolonged latency periods of trigeminal evoked potential peaks compared to controls. Increased muscle tone and pain severity in the cranio-cervical region were predominantly observed in patients with high reflex excitability and were significantly correlated with tinnitus intensity and handicap scores. Somatic modulation of tinnitus was identified in more than half of the patients and was associated with higher neurophysiological and clinical severity. Additionally, patients with nasal and nasopharyngeal pathology showed reduced tympanic membrane mobility and signs of hyporeflexive neural function.

Conclusion. The findings support the concept of altered trigeminal–somatosensory integration in tinnitus pathogenesis and underline the necessity of a multidisciplinary diagnostic and therapeutic approach involving neurologists, otorhinolaryngologists, and related specialists.

Keywords: tinnitus; trigeminal nerve; somatosensory system; trigeminal evoked potentials; neurophysiology; somatic modulation.

КЛИНИКО-НЕЙРОФИЗИОЛОГИЧЕСКИЕ ОСОБЕННОСТИ У ПАЦИЕНТОВ С ШУМОМ В УШАХ: РОЛЬ ТРИГЕМИНО-СОМАТОСЕНСОРНЫХ ВЗАИМОДЕЙСТВИЙ

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Аннотация.

Введение. Шум в ушах (тиннитус) является многофакторным заболеванием и одной из актуальных проблем современной неврологии и оториноларингологии. В последние годы особое внимание уделяется роли неслуховых структур, прежде всего соматосенсорной системы и тройничного нерва, в формировании и поддержании тиннитуса. Целью настоящего исследования стало изучение клинических и нейрофизиологических особенностей пациентов с шумом в ушах с акцентом на рефлекторную возбудимость тройничной системы и её связь с соматической модуляцией и выраженностью симптомов.

Материалы и методы. В исследование включены 112 пациентов с субъективным шумом в ушах, которым были проведены неврологический и оториноларингологический осмотры, аудиологическое обследование, тимпанометрия, психоакустическая оценка с использованием опросника Tinnitus Handicap Inventory, а также нейрофизиологическое исследование с регистрацией тригеминальных вызванных потенциалов. Контрольную группу составили 20 здоровых добровольцев. В зависимости от параметров тригеминальных вызванных потенциалов пациенты были разделены на подгруппы с высокой, умеренной и низкой рефлекторной возбудимостью.

Результаты. Полученные результаты выявили различия в латентных периодах пиков тригеминальных вызванных потенциалов между подгруппами и контрольной группой. У пациентов с высокой рефлекторной возбудимостью отмечались выраженное мышечное напряжение и болевой синдром в краниоцервикальной области, которые коррелировали с интенсивностью шума и показателями опросника THI. Соматическая модуляция шума была выявлена более чем у половины пациентов и ассоциировалась с большей клинической тяжестью. У больных с патологией носа и носоглотки отмечались снижение подвижности барабанной перепонки и признаки гипорефлекторного состояния нервной системы.

Заключение. Полученные данные подтверждают значимую роль тригемино-соматосенсорных механизмов в патогенезе тиннитуса и обосновывают необходимость комплексного междисциплинарного подхода к диагностике и лечению данной категории пациентов.

Ключевые слова: шум в ушах; тиннитус; тройничный нерв; соматосенсорная система; тригеминальные вызванные потенциалы; нейрофизиология.

QULOQDA SHOVQIN BILAN KECHUVCHI BEMORLARDA KLINIK VA NEYROFIZIOLOGIK XUSUSIYATLAR: TRIGEMINAL–SOMATOSENSOR O‘ZARO TA’SIRLARNING ROLI

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Annotatsiya.

Kirish. Quloqda shovqin (tinnitus) zamonaviy nevrologiya va otorinolaringologiyada dolzarb bo‘lgan ko‘p omilli klinik muammo hisoblanadi. So‘nggi yillarda tinnitus patogenezi eshituv tizimidan tashqari tuzilmalar, xususan somatosensor tizim va uch shoxli nervning ahamiyati tobora ko‘proq e‘tiborni tortmoqda. Ushbu tadqiqotning maqsadi quloqda shovqindan shikoyat qiluvchi bemorlarda klinik va neyrofiziologik xususiyatlarni, ayniqsa trigeminal reflektor qo‘zg‘aluvchanlik va uning somatik modulyatsiya hamda simptomlar og‘irligi bilan bog‘liqligini o‘rganishdan iborat.

Materiallar va usullar: Tadqiqotga subyektiv tinnitus bilan og‘rigan 112 nafar bemor kiritildi. Barcha bemorlarga nevrologik va otorinolaringologik ko‘rik, audiologik tekshiruv, timpanometriya, Tinnitus Handicap Inventory yordamida psixoakustik baholash hamda trigeminal chaqirilgan potentsiallarni qayd etish orqali neyrofiziologik tekshiruv o‘tkazildi. Nazorat guruhi 20 nafar sog‘lom ko‘ngillidan iborat bo‘ldi. Trigeminal chaqirilgan potentsiallar ko‘rsatkichlariga ko‘ra bemorlar yuqori, o‘rtacha va past reflektor qo‘zg‘aluvchanlikka ega kichik guruhlariga ajratildi.

Natijalar trigeminal chaqirilgan potentsiallar latent davrlarida guruhlararo farqlar mavjudligini ko'rsatdi. Yuqori reflektor qo'zg'aluvchanlikka ega bemorlarda kraniotservikal sohada mushak tonusining oshishi va og'riq sindromi ustun bo'lib, ular quloqdagi shovqin intensivligi va THI ko'rsatkichlari bilan ishonchli bog'liqlikni namoyon etdi. Somatik modulyatsiya bemorlarning yarmidan ko'pida aniqlanib, klinik jihatdan og'irroq kechish bilan tavsiflandi. Burun va burun-halqum patologiyasiga ega bemorlarda baraban pardasining harakatchanligi pasaygan va nerv tizimining giporeflektor holati kuzatildi.

Xuslosa. Olingan ma'lumotlar tinnitus patogenezida trigeminal–somatosensor mexanizmlarning muhim o'rinni egallashini tasdiqlaydi va bunday bemorlarni tekshirish hamda davolashda kompleks, multidisiplinar yondashuv zarurligini asoslaydi.

Kalit so'zlar: quloqda shovqin; tinnitus; uch shoxli nerv; somatosensor tizim; trigeminal chaqirilgan potentsiallar; neyrofiziologiya.

Introduction. Tinnitus, defined as the perception of sound in the absence of an external acoustic stimulus, remains one of the most complex and insufficiently understood problems at the intersection of neurology, otorhinolaryngology, and neurophysiology. Epidemiological studies indicate that chronic tinnitus affects between 10% and 15% of the adult population, with clinically significant distress reported in approximately 1–3% of cases [1]. Despite its high prevalence, tinnitus continues to pose diagnostic and therapeutic challenges due to its heterogeneous etiology, variable clinical presentation, and multifactorial pathophysiological mechanisms.

Traditionally, tinnitus has been conceptualized as a disorder primarily related to cochlear damage and aberrant activity within the auditory pathway. However, this purely auditory model has proven insufficient to explain several clinical observations, including tinnitus persistence after cochlear nerve section, poor correlation between hearing loss severity and tinnitus intensity, and the frequent modulation of tinnitus by somatic maneuvers involving the head, neck, jaw, or upper limbs [2]. These phenomena suggest that non-auditory systems may play a significant role in tinnitus generation and maintenance. Over the past two decades, increasing attention has been directed toward the interaction between the somatosensory system and the auditory analyzer. Experimental and clinical studies have demonstrated anatomical and functional connections between the dorsal cochlear nucleus (DCN) and somatosensory afferents originating from the trigeminal nerve and cervical spinal nerves [3]. In the early 2000s, neurophysiological investigations revealed pathological cross-modal plasticity between the DCN and the spinal trigeminal nucleus, providing a mechanistic substrate for somatosensory modulation of tinnitus [4]. These findings marked a paradigm shift, moving tinnitus research beyond the cochlea toward central integrative networks. The trigeminal nerve occupies a unique position in this context due to its extensive sensory innervation of craniofacial structures and its close anatomical and functional proximity to auditory nuclei in the brainstem. Alterations in trigeminal reflex excitability may influence the activity of middle ear muscles, palatal muscles, and periauricular musculature, leading to sustained changes in auditory perception. Reflex hyperactivity or hypoactivity within these circuits can contribute to the persistence and subjective amplification of tinnitus [5]. In addition to neurogenic mechanisms, peripheral somatic factors, particularly pathologies of the nasal cavity and nasopharynx, have been implicated in tinnitus pathogenesis. Nasal obstruction, chronic rhinosinusitis, and nasopharyngeal inflammation may affect Eustachian tube function and middle ear pressure regulation, thereby indirectly influencing auditory perception and neuromuscular reflexes [6]. The interaction between peripheral otolaryngological pathology and central neurophysiological alterations remains insufficiently characterized, especially in clinical populations.

From a clinical standpoint, tinnitus is not merely an auditory symptom but a multidimensional condition associated with emotional distress, cognitive dysfunction, sleep disturbances, and reduced quality of life. The Tinnitus Handicap Inventory (THI) has become a widely used psychometric tool to quantify tinnitus-related disability and assess treatment outcomes [7]. However, psychometric measures alone cannot capture the underlying neurophysiological heterogeneity of tinnitus, underscoring the need for objective biomarkers. Trigeminal evoked potentials (TEPs) represent a valuable neurophysiological method for assessing the functional state

of trigeminal afferent pathways and associated brainstem structures. Changes in latency periods and amplitude of TEP components reflect alterations in synaptic transmission, axonal conduction, and reflex excitability within trigeminal–brainstem circuits [8]. Despite their potential relevance, TEPs remain underutilized in routine tinnitus evaluation. In the Republic of Uzbekistan, and particularly within the clinical practice of Samarkand State Medical University, tinnitus patients are frequently managed in a fragmented manner, often limited to symptomatic otological treatment. There is a clear need for an integrative clinical–neurophysiological approach that accounts for central and peripheral mechanisms, especially given the high prevalence of musculoskeletal and nasal pathologies in the local population.

The aim of the study was to investigate the clinical and neurophysiological characteristics of patients with tinnitus, with particular emphasis on trigeminal reflex excitability, somatic modulation, and their relationship with tinnitus severity. By integrating neurological, otolaryngological, audiological, and neurophysiological assessments, we sought to identify clinically meaningful subgroups of tinnitus patients and to elucidate the role of trigeminal–somatosensory interactions in tinnitus pathogenesis.

Materials and Methods. A prospective observational study was conducted at the Department of Neurology in collaboration with the Department of Otorhinolaryngology of Samarkand State Medical University. The study was carried out in accordance with the principles of the Declaration of Helsinki. All participants provided informed consent prior to inclusion.

A total of 112 patients presenting with subjective complaints of tinnitus localized to the ears or head were enrolled in the study. Patients ranged in age from 22 to 68 years, with a mean age of 44.7 ± 9.6 years. Both unilateral and bilateral tinnitus cases were included. A control group consisting of 20 healthy volunteers without tinnitus or known neurological or otological disorders was formed for comparative analysis. Exclusion criteria included objective tinnitus, acute otitis media, vestibular schwannoma, multiple sclerosis, prior neurosurgical intervention in the posterior fossa, and severe psychiatric disorders that could interfere with neurophysiological testing.

All patients underwent a comprehensive clinical evaluation that included detailed medical history taking, neurological examination, and otorhinolaryngological assessment. Particular attention was paid to musculoskeletal dysfunction in the cranio-cervical region, nasal and nasopharyngeal pathology, and signs of middle ear involvement. Audiological testing included pure-tone audiometry and tympanometry. Tympanometric measurements were used to assess tympanic membrane mobility and middle ear compliance. Reduced compliance was interpreted as a potential indicator of middle ear muscle dysfunction or Eustachian tube impairment. Psychometric evaluation of tinnitus severity was performed using the Tinnitus Handicap Inventory. The THI total score was used as an index of tinnitus-related distress.

Trigeminal evoked potentials were recorded using standard surface electrodes. Electrical stimulation was applied separately to the first (ophthalmic), second (maxillary), and third (mandibular) branches of the trigeminal nerve. Latency periods of characteristic peaks (P22, N30 and others) were measured bilaterally. Based on TEP parameters, patients were stratified into three subgroups reflecting different levels of trigeminal reflex excitability. The first subgroup included patients with high reflex excitability, the second subgroup included patients with low reflex excitability, and the third subgroup consisted of patients with moderate reflex excitability. Manual diagnostic assessment was performed to evaluate muscle tone and the severity of painful sensations in craniofacial and cervical muscles. The severity of pain was graded using a standardized clinical scale.

Statistical analysis was performed using conventional descriptive and correlational methods. Correlations between neurophysiological parameters, muscle pain severity, tympanometric indices, and THI scores were analyzed. Differences between subgroups and controls were considered statistically significant at $p < 0.05$.

Results. Analysis of trigeminal evoked potentials revealed distinct neurophysiological patterns among the three patient subgroups. Patients with high reflex excitability demonstrated a significant shortening of latency periods across multiple TEP components compared to controls. In contrast, patients with low reflex excitability exhibited prolonged latency periods in all trigeminal

branches. The moderate excitability subgroup showed selective shortening of P22 and N30 latencies in the mandibular branch. Table 1 presents the comparative latency values of key TEP components among the study groups.

Table 1. Latency periods of trigeminal evoked potential peaks (ms)

Group	P22 (III branch)	N30 (III branch)	Mean latency deviation
Control	22.4 ± 1.1	30.6 ± 1.3	Reference
High excitability	19.8 ± 1.0	27.9 ± 1.2	Shortened
Moderate excitability	20.9 ± 1.2	29.1 ± 1.4	Selectively shortened
Low excitability	24.7 ± 1.4	33.8 ± 1.6	Prolonged

Manual diagnostic examination revealed that patients in the high excitability subgroup had significantly increased muscle tone and more pronounced painful sensations in craniofacial and cervical muscles compared to other subgroups. The severity of muscle pain correlated positively with tinnitus intensity and THI scores. Somatic modulation of tinnitus, defined as a change in tinnitus intensity during voluntary motor maneuvers, was observed in 59.8% of patients. Patients demonstrating somatic modulation had higher THI scores and greater muscle pain severity. Patients with nasal and nasopharyngeal pathology exhibited reduced tympanic membrane mobility and signs of hyporeflexive nervous system function. Correlational analysis demonstrated significant associations between tympanometric parameters and muscular dysfunction indices. Table 2 summarizes the relationship between clinical parameters and tinnitus characteristics.

Table 2. Correlation between clinical parameters and tinnitus severity

Parameter	Correlation with THI	Clinical interpretation
Muscle pain severity	Positive	Somatosensory contribution
TEP latency	Inverse/Direct	Reflex excitability dependent
Tympanometric compliance	Negative	Middle ear involvement

Discussion. The findings of this study support the concept that tinnitus is not exclusively an auditory disorder but rather a multisystem condition involving complex interactions between auditory, somatosensory, and autonomic pathways. The observed alterations in trigeminal evoked potentials indicate changes in reflex excitability at the level of the brainstem, particularly within trigeminal–auditory integrative circuits. Shortened latency periods in patients with high reflex excitability may reflect increased synaptic efficiency or reduced inhibitory control within trigeminal pathways. Such hyperexcitability could facilitate aberrant cross-modal signaling to the dorsal cochlear nucleus, thereby sustaining tinnitus perception [9]. Conversely, prolonged latencies in hyporeflexive patients may indicate reduced afferent input or impaired synaptic transmission, potentially linked to chronic inflammatory or degenerative processes.

The strong correlation between muscle pain severity, TEP parameters, and tinnitus intensity underscores the clinical relevance of somatosensory dysfunction. These findings align with previous reports demonstrating that cervical and temporomandibular disorders can modulate tinnitus through trigeminal and spinal afferents [10].

The association between nasal pathology, tympanic membrane hypomobility, and neurophysiological hypoexcitability suggests an additional peripheral mechanism contributing to tinnitus. Impaired middle ear mechanics may alter proprioceptive and reflex input to central auditory structures, further complicating the clinical picture. From a practical perspective, the results highlight the importance of multidisciplinary evaluation in tinnitus management. Limiting diagnostic workup to audiological testing may overlook critical somatosensory and neurophysiological contributors, particularly in patients with prominent musculoskeletal or nasal pathology.

Conclusion. The present study demonstrates that patients with tinnitus exhibit distinct clinical and neurophysiological profiles characterized by altered trigeminal reflex excitability, somatosensory dysfunction, and peripheral otolaryngological factors. Trigeminal evoked potentials provide valuable objective information that complements clinical and psychometric assessment.

Recognition of these mechanisms necessitates an expansion of diagnostic and therapeutic strategies, involving neurologists, otorhinolaryngologists, and other specialists. An integrative approach tailored to the individual neurophysiological profile of the patient may improve clinical outcomes and reduce tinnitus-related disability.

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