

ACÚFENOS

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CLASIFICACIÓN

ACUFENOS VERDADEROS

Percepción de un sonido como resultado de una actividad anómala en uno o varios lugares de las vías auditivas. Habitualmente se describe como un “pito” o un ruido de “vapor o corriente de agua”.



CLASIFICACIÓN

ACÚFENO FALSO O PSEUDOACÚFENO.

Percepción de un sonido que no es debido ni a una actividad anómala de las vías auditivas ni a una fuente sonora externa del cuerpo.



CLASIFICACIÓN

ACÚFENO FALSO O PSEUDOACÚFENO.

El pseudoacúfeno es un sonido producido por alguna parte del cuerpo distinta a los órganos auditivos, vías auditivas y centros auditivos, pero que es audible en el oído por la persona cuyo cuerpo lo produce. Se trata de estructuras que por si mismas producen ruido.



CLASIFICACIÓN

ACÚFENO FALSO O PSEUDOACÚFENO.

Habitualmente se describe como un ruido pulsátil o de tambor (vascular).



ACÚFENO FALSO O PSEUDOACUFENO.

Aquí se incluyen:

- los sonidos objetivos como los originados al deglutir, al movilizar la ATM, al bostezar, los cardiovasculares, etc.
- Sonidos producidos por prótesis, como válvulas cardíacas, etc.
- Sonidos pulsátiles por malformación arterial o venosa (fistulas arteriovenosas, neoplasias, etc).
- Sonidos producidos por mioclonias, por contracturas o espasmos en masetero, tensor del martillo, tensor del tímpano,, etc..
- Sonidos secundarios a traumatismos como el whiplash.



ACÚFENO FALSO O PSEUDOACUFENO.

A nivel de exploración osteopática estos sonidos se pueden explorar haciendo cambios posturales cefálicos en sedestación, hiperextensión en decúbito, presiones sobre la musculatura del cuello y cara, presiones sobre la yugular interna, en maniobras de Valsalva.

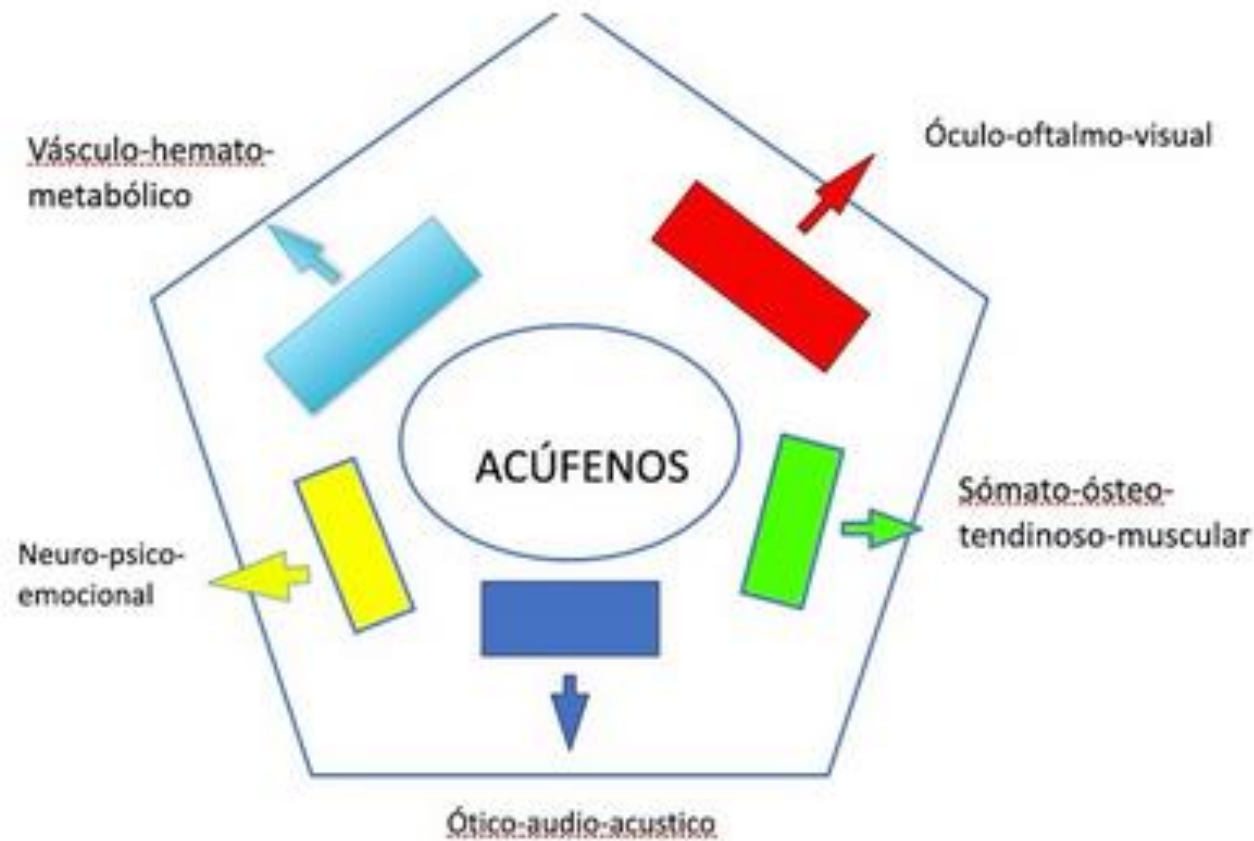
Otros medios sería la ECO Doppler, ECG, angio RM.



CLASIFICACIÓN

Otros ruidos son aquellas alucinaciones auditivas presentes en esquizofrénicos o personas que se han dedicado a la música





Todos los acúfenos se hallan dentro de alguno o algunos de los grupos citados . Generalmente habrán varias causas que actúan de forma simultanea.

1-ACÚFENO ÓCULO-OFTAÁLMICO-VISUAL (AOOV).

Este acúfeno está habitualmente vinculado a los movimientos oculares extremos en las cuatro direcciones del espacio.

Hay áreas auditivas con un 64 % de respuestas a estímulos visuales, y un 33 % a movimientos oculares.

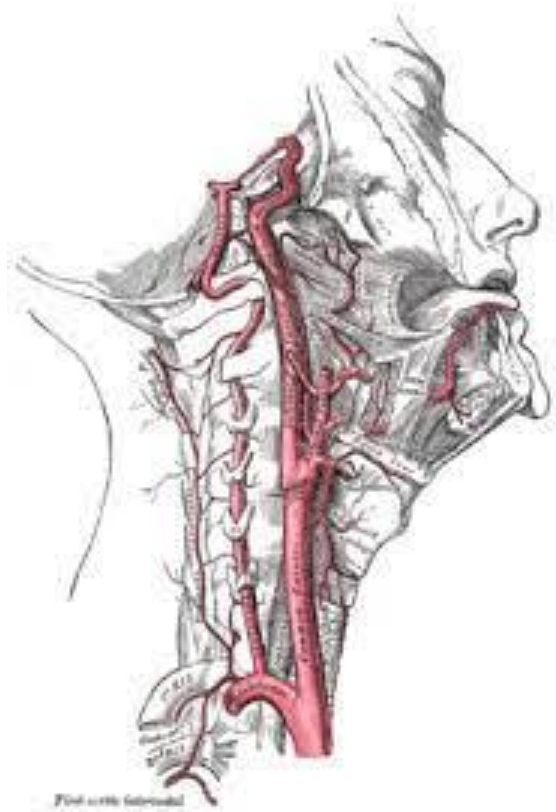


2- ACÚFENO SOMATOSENSORIAL-ÓSTEO- TENDINOSO (ASOTM).

- Habitualmente de etiología cervical y ATM.
- Las fibras somatosensoriales están involucradas en el procesamiento de la información del sistema auditivo.
- Con frecuencia este acúfeno se origina en el territorio cervical y trigeminal cutáneo.



3-ACÚFENO VÁSCULO-HEMÁTICO-METABÓLICO (AVHM).



Se debe, habitualmente, a alteraciones de los ejes carotídeos y de las ramas vasculares cerebrales; sin descartar alteraciones cardíacas o neurovasculares.

La etiología, en este caso, corresponde a alteraciones vasculares, hemática y bioquímicas que pueda sufrir el paciente.

Lo más frecuente son los procesos vasculares de los ejes carotídeos.



4-ACÚFENO NEURO-PSICO-EMOCIONAL (ANPE).

Habitualmente está vinculado a situaciones de ansiedad, estrés y gestión de la depresión.

Las vías neurológicas actúan tanto en sentido de la cóclea hacia el cerebro como a la inversa; sucede, por ejemplo, que el sistema nervioso central mantiene determinados acúfenos, que son de origen coclear, pero también los procesos neuro psicológicos son una fuente importante de Acufenos, que el propio cerebro puede activar en sí mismo o en la cóclea.



5-ACÚFENO ÓTICO-AUDIO-ACÚSTICO (AOAA).

Habitualmente está vinculado a lesiones del circuito auditivo (oído externo, medio, interno).

Se trata de acúfenos producidos en el recorrido que va de la cóclea al córtex auditivo.

En un 90 % es coclear debido a alteraciones de células ciliadas, internas o externas, alteraciones de los neurotransmisores, alteraciones de los estímulos, aferentes, cerebrales, y alteraciones de la estría vascular.



EN RESUMEN

El tinnitus es multifactorial, pero la principal causa de su aparición es la pérdida de audición, que está presente en hasta el 90% de las personas.



EN RESUMEN

Como consecuencia de la plasticidad neuronal, la pérdida de información al sistema nervioso auditivo puede promover el tinnitus:

la privación de información auditiva puede provocar una reorganización del mapa topográfico en las cortezas cerebrales

Kaas, 1991; Kral et al., 2000.



Medicamentos que pueden causar lesiones auditivas temporales:



- Ácido Acetil-Salicílico: Se encuentra en forma de Aspirina y son peligrosos en dosis muy elevadas o frecuentes. Se usa habitualmente como antiagregante plaquetario.
- Antimaláricos: Presente en la quinina, la cloroquina, y la pirimetamina.



Medicamentos que pueden causar lesiones auditivas permanentes:



- **Antibióticos aminoglucósidos:** Como la gentamicina, la estreptomicina, neomicina y netilmicina.
- **Diuréticos de asa:** Entre ellos la piretanida, la bumetadina, y la furosemida.
- **Citostáticos:** Como el cisplatino, la ciclofosfamida, la bleomicina, la vincristina y el carboplatino.
- **Antibióticos macrólifos y sus derivados:** Aquí encontramos la clindamicina, la claritomicina, la eritromicina y la azitromicina.
- **Antibióticos glucopeptídicos:** Entre ellos la vancomicina y la teicoplanina.
- **Betabloqueantes:** Propanolol y practolol.



Association Between Subjective Tinnitus and Cervical Spine or Temporomandibular Disorders: A Systematic Review

E. J. Brouwer^{1,2}, E. A. Koops^{1,3}, P. van Dijk^{1,3} and P. U. Dijkstra^{4,5}

Abstract

Movements of the neck and jaw may modulate the loudness and pitch of tinnitus. The aim of the present study was to systematically analyse the strength of associations between subjective tinnitus, cervical spine disorders (CSD), and temporomandibular disorders (TMD). A systematic literature search of the Medline, Embase, and Pedro databases was carried out on articles published up to September 2017. This covered studies in which tinnitus and CSD or TMD were studied as a primary or a secondary outcome and in which outcomes were compared with a control group. Included articles were evaluated on nine methodological quality criteria. Associations between tinnitus and CSD or TMD were expressed as odds ratios. In total, 2,139 articles were identified, of which 24 studies met the inclusion criteria. Twice, two studies were based on the same data set; consequently, 22 studies were included in the meta-analysis. Methodological quality was generally limited by a lack of blinding, comparability of groups, and nonvalidated instruments for assessing CSD. Results indicated that patients with tinnitus more frequently reported CSD than subjects without tinnitus. The odds ratio was 2.6 (95% CI [1.1, 6.4]). For TMD, a bidirectional association with tinnitus was found; odds ratios ranged from 2.3 (95% CI [1.5, 3.6]) for arthrogenous TMD to 6.7 (95% CI [2.4, 18.8]) for unspecified TMD. Funnel plots suggested a publication bias. After adjusting for this, the odds ratios decreased, but associations persisted. There is weak evidence for an association between subjective tinnitus and CSD and a bidirectional association between tinnitus and TMD.

Keywords

subjective tinnitus, systematic review, neck pain, temporomandibular disorder

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Introduction

Tinnitus is a sound that is perceived in the absence of an acoustic event occurring external to the listener's body. It is commonly described as the sound of, for example, crickets, winds, falling tap water, grinding steel, escaping vacuum sound, or as a combination of sounds (Han, Lee, Kim, Lim, & Shin, 2009). Tinnitus can be perceived in one ear, both ears, or more centrally located "in the head" (Heller, 2003). In adults of the general population, the prevalence of tinnitus ranges between 10% and 15% (D. Baguley, McFerran, & Hall, 2013). In people older than 60 years of age, the prevalence of tinnitus increases to about 18% (Davis & El Rafea, 2000).

In a minority of people with tinnitus, the sound is audible to an observer and is therefore called *objective tinnitus*. In most of these patients, it is possible to determine the underlying etiology. However, in the vast

majority of tinnitus sufferers, the sound is audible only to the patient and is called *subjective tinnitus* (Ward, Vella, Hume, & Hall, 2015).

Numerous studies suggest that subjective tinnitus arises in the central auditory system due to neuroplastic adaptations that occur in response to changes in the

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Central nervous system neurodegeneration and tinnitus: a clinical experience. Part I: Diagnosis

Abraham Shulman¹, Barbara Goldstein, Arnold M Strashun

Affiliations + expand

PMID: 18229791

Abstract

In an evolving clinical experience since 1979, the medical significance of the symptom of tinnitus has been identified as a "soft" sign of neurodegeneration (ND) in the central nervous system (CNS) in a particular subset of tinnitus patients diagnosed with a predominantly central-type, severe, disabling, subjective idiopathic tinnitus. To highlight this experience, a retrospective review and analysis of consecutive tinnitus patients (N = 96) was conducted. Ninety-six tinnitus patients (ages 22-90 years) were seen in neurotological consultation from November 1, 2005, to June 30, 2007, all of whom had subjective idiopathic tinnitus of the severe disabling type (SIT). Of these 96 patients, 54 had SIT of the predominantly central type and of these, 18 (ages 39-75 years) were recommended for nuclear medicine imaging (single-photon emission computed tomography [SPECT] and fluorodeoxyglucose-positron emission tomography/computed tomography [FDG-PET/CT]). Patient selection for nuclear medicine imaging fulfilled the criteria of a medical-audiological ND tinnitus profile: completion of a patient protocol that diagnosed a predominantly central-type, severe, disabling, subjective, idiopathic tinnitus lasting in excess of 1 year, and failure of existing modalities of treatment attempting tinnitus relief. In 16 of the 18 patients, objective evidence of ND was reported in multiple neural substrates of brain obtained with SPECT or FDG-PET/CT of brain. Classification of CNS ND and tinnitus differentiated between (1) ND of nonspecific or unknown etiology; (2) ND manifested by perfusion asymmetries in brain associated with ischemia (n = 11/18); and (3) neurodegenerative CNS disease consistent with nuclear medicine criteria for senile dementia of the Alzheimer's type (n = 5/18). The diagnosis has been associated with cerebrovascular disease (n = 16/18). The identification of neurodegenerative CNS disease in a selected cohort of patients with subjective idiopathic tinnitus as a soft sign of such CNS disease has implications for diagnosis and treatment.

Review > Prog Brain Res. 2007;166:37-45. doi: 10.1016/S0079-6123(07)66003-8.

The role of neural plasticity in tinnitus

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Affiliations + expand

PMID: 17956769 DOI: 10.1016/S0079-6123(07)66003-8

Abstract

There is considerable evidence that expression of neural plasticity plays a central role in the development of the abnormalities that cause many forms of tinnitus. Expression of neural plasticity can change the balance between excitation and inhibition, promote hyperactivity, and cause re-organization of specific parts of the nervous system or redirection of information to parts of the nervous system not normally involved in processing of sounds (such as the non-classical, or extralemniscal pathways). The strongest promoter of expression of neural plasticity is deprivation of input, which explains why tinnitus often occurs together with hearing loss or injury to the auditory nerve.

Existe evidencia considerable de que la expresión de la plasticidad neuronal puede causar tinnitus, hiperacusia y trastornos afectivos como la depresión y la fonofobia.

La plasticidad neuronal es una propiedad del sistema nervioso que permite que partes específicas del sistema nervioso cambien su función y su organización

Review > J Craniomandib Disord. 1990 Summer;4(3):186-92.

Prevalence of signs and symptoms of craniomandibular disorders in tinnitus patients

B Rubinstein ¹, A Axelsson, G E Carlsson

1990

Affiliations + expand

PMID: 2098394

Abstract

One hundred two tinnitus patients were examined to determine the prevalence of signs and symptoms of CMD. The examination comprised determination of the anamnestic and clinical dysfunction indices according to Helkimo; assessment of the dentition, occlusal factors, and signs of parafunctions; and accomplishment of a tension test. Patients also answered a questionnaire concerning the presence and frequency of CMD symptoms and headaches, as well as influence on tinnitus by mandibular movements, by pressure applied to the TMJ, or by dental therapy. Frequent headaches and fatigue/tenderness in jaw muscles were more prevalent in tinnitus patients than in epidemiologic samples, as was the prevalence of clinical findings of pain on palpation of masticatory muscles, impaired mandibular mobility, and signs of parafunctions. About one-third of the patients reported influence on tinnitus by mandibular movements and/or pressure applied to the TMJs. A theoretic model of causal connections between tinnitus and signs and symptoms of CMD in some tinnitus patients is suggested.

PubMed Disclaimer

Las cefaleas frecuentes, la fatiga y/o sensibilidad por presión en los músculos masticatorios así como la alteración de la movilidad fisiológica de la mandíbula, fueron más prevalentes en los pacientes con tinnitus.

Aproximadamente un tercio de los pacientes informaron que los movimientos mandibulares y/o la presión aplicada a las ATM tenía repercusión sobre el tinnitus.

Signs of temporomandibular disorders in tinnitus patients and in a population-based group of volunteers: results of the Study of Health in Pomerania

O Bernhardt¹, D Gesch, C Schwahn, K Bitter, T Mundt, F Mack, T Kocher, G Meyer, E Hensel, U John

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PMID: 15089935 DOI: 10.1046/j.1365-2842.2003.01249.x

2004

Abstract

The literature has documented a controversial discussion on the possible relationship of otogenous symptoms and craniomandibular dysfunction since the 1920s. Therefore, an investigation was conducted which consisted of two parts: a case study with population-based controls and a cross-sectional study. The aim of the first study was to screen a group of patients suffering from acute or chronic tinnitus for temporomandibular disorders (TMD) in comparison with a population-based group of volunteers without tinnitus. To this end, 30 patients (13 females and 17 males, age 18-71 years) suffering from acute hearing loss associated with tinnitus, isolated acute tinnitus, and chronically transient tinnitus were examined for symptoms of craniomandibular dysfunction. The results were compared with those of clinical functional analysis from 1907 subjects selected representatively and according to age distribution from the epidemiological 'Study of Health in Pomerania' (SHIP); the occurrence of tinnitus was ruled out in these control subjects. Statistical analysis was performed with Chi-square and Mann-Whitney U-tests. Sixty per cent of the tinnitus patients and 36.5% of the control subjects exhibited more than two symptoms of TMD ($P = 0.004$). Tinnitus patients had significantly more muscle palpation pain ($P < 0.001$), temporomandibular joint (TMJ) palpation pain ($P < 0.001$), and pain upon mouth opening ($P < 0.001$) than the general population group. No statistical differences were found in TMJ sounds, limitation of mandibular movement, or hypermobility of the TMJ. Furthermore, 4228 subjects of the population group examined in the epidemiological study were screened for co-factors of tinnitus with the help of a multivariate logistic regression model which was adjusted for gender, age, and a variety of anamnestic and examined data. Increased odds ratios (OR) were found for tenderness of the masticatory muscles (OR = 1.6 for one to three painful muscles and OR = 2.53 for four or more painful muscles), TMJ tenderness to dorsal cranial compression (OR = 2.99), listlessness (OR = 2.0) and frequent headache (OR = 1.84). A relationship between tinnitus and TMD was established in both examinations. Tinnitus patients seem to suffer especially from myofascial and TMJ pain. A screening for TMD should be included in the diagnostic survey for tinnitus patients.

El objetivo del estudio fue examinar a un grupo de pacientes que padecían tinnitus agudo o crónico para detectar trastornos temporomandibulares (TMD) en comparación con un grupo poblacional de voluntarios sin tinnitus.

Se encontraron mayores odds ratios (OR) para la sensibilidad a la presión de los músculos masticatorios, sensibilidad a la presión y compresión de la ATM y cefaleas frecuente.

Como conclusión se estableció relación entre tinnitus y DTM.

Los pacientes con tinnitus parecen sufrir especialmente dolor miofascial y de ATM.

FREE

The Somatic Connection | October 2016

Manual Therapy and OMT May Be of Benefit in the Management of Somatosensory Tinnitus

Hollis H. King, DO, PhD

+ Author Notes

The Journal of the American Osteopathic Association, October 2016, Vol. 116, 684-685. doi:<https://doi.org/10.7556/jaoa.2016.134>

> [Pain Med.](#) 2020 Mar 1;21(3):613-624. doi: 10.1093/pm/pnz278.

Effects of Cervico-Mandibular Manual Therapy in Patients with Temporomandibular Pain Disorders and Associated Somatic Tinnitus: A Randomized Clinical Trial

[Pablo Delgado de la Serna](#) ¹, [Gustavo Plaza-Manzano](#) ² ³, [Joshua Cleland](#) ⁴ ⁵ ⁶, [César Fernández-de-Las-Peñas](#) ⁷ ⁸, [Patricia Martín-Casas](#) ², [María José Díaz-Arribas](#) ²

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PMID: 31665507 DOI: [10.1093/pm/pnz278](#)

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

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PMID: 26749460 DOI: [10.1016/j.math.2015.11.008](#)

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.

Comparative Study

> Man Ther. 2016 Jun;23:106-13. doi: 10.1016/j.math.2016.02.006.

Epub 2016 Feb 21.

Cervicogenic somatosensory tinnitus: An indication for manual therapy plus education? Part 2: A pilot study

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PMID: 26971019 DOI: 10.1016/j.math.2016.02.006

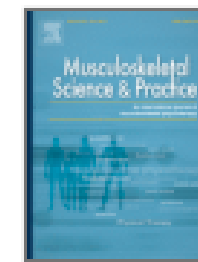
> [Pain Pract.](#) 2020 May 18. doi: 10.1111/papr.12919. Online ahead of print.

Influence of Clinical, Physical, Psychological, and Psychophysical Variables on Treatment Outcomes in Somatic Tinnitus Associated With Temporomandibular Pain: Evidence From a Randomized Clinical Trial

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PMID: 32419303 DOI: [10.1111/papr.12919](#)



Original article

Prognostic indicators for decrease in tinnitus severity after cervical physical therapy in patients with cervicogenic somatic tinnitus

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<https://doi.org/10.1016/j.msksp.2017.02.008>

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Randomized Controlled Trial

> Man Ther. 2016 Dec;26:125-131.

doi: 10.1016/j.math.2016.08.005. Epub 2016 Aug 26.

Does multi-modal cervical physical therapy improve tinnitus in patients with cervicogenic somatic tinnitus?

S Michiels¹, P Van de Heyning², S Truijen³, A Hallemans⁴, W De Hertogh⁵

Affiliations + expand

PMID: 27592038 DOI: 10.1016/j.math.2016.08.005

Case Reports

> J Am Acad Audiol. Jul-Aug 2013;24(7):544-55. doi: 10.3766/jaaa.24.7.3.

Improving tinnitus with mechanical treatment of the cervical spine and jaw

Kay Cherian¹, Neil Cherian, Chad Cook, James A Kaltenbach

Affiliations + expand

PMID: 24047942 DOI: [10.3766/jaaa.24.7.3](https://doi.org/10.3766/jaaa.24.7.3)

> [Trials](#). 2018 Jun 27;19(1):342. doi: 10.1186/s13063-018-2738-9.

Comparison of the effectiveness between transcutaneous electrical nerve stimulation, manual acupuncture, and electroacupuncture on tinnitus: study protocol for a randomized controlled trial

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Affiliations + expand

PMID: 29945669 PMCID: [PMC6020312](#) DOI: [10.1186/s13063-018-2738-9](#)

Free PMC article

Randomized Controlled Trial

> Braz J Otorhinolaryngol. 2012 Dec;78(6):21-6.

doi: 10.5935/1808-8694.20120028.

Efficacy of myofascial trigger point deactivation for tinnitus control

Carina Bezerra Rocha ¹, Tanit Ganz Sanchez

Affiliations + expand

PMID: 23306563 DOI: 10.5935/1808-8694.20120028

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