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Original Research Article

Association between Hypertension and Tinnitus: A Clinical Correlation Study

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Abstract

Background: Tinnitus represents a prevalent auditory symptom that significantly impacts quality of life, while hypertension constitutes a major global health burden with potential effects on cochlear microcirculation. The relationship between these conditions remains incompletely understood.

Methods: A cross-sectional comparative study was conducted with 286 participants (143 hypertensive patients and 143 age- and sex-matched normotensive controls) aged 35-70 years. Hypertension was classified according to current guidelines. Tinnitus presence, severity, and characteristics were assessed using standardized questionnaires and the Tinnitus Handicap Inventory (THI). Audiometric evaluation and clinical data collection were performed. Statistical analyses included chi-square tests, independent t-tests, and logistic regression.

Results: Tinnitus prevalence was significantly higher in hypertensive patients (46.9%) compared to normotensive controls (28.7%) ($p < 0.001$). Mean THI scores were 38.4 ± 22.6 in hypertensive patients with tinnitus versus 26.8 ± 18.3 in normotensive participants with tinnitus ($p = 0.003$). Grade 2 hypertension showed the strongest association with tinnitus (OR = 3.45; 95% CI: 1.82-6.54; $p < 0.001$). The duration of hypertension significantly correlated with tinnitus severity ($r = 0.418$, $p < 0.001$). Bilateral tinnitus was more prevalent in hypertensive patients (68.7% vs 46.3%, $p = 0.012$).

Conclusion: This study demonstrates a significant association between hypertension and increased tinnitus prevalence and severity. The findings suggest that hypertension assessment should be considered in tinnitus evaluation, and audiological monitoring may be warranted in hypertensive patients.

Keywords: Tinnitus; Hypertension; Cardiovascular disease; Hearing disorders; Cochlear blood flow; Audiometry.

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Introduction

Tinnitus, defined as the perception of sound in the absence of external acoustic stimulation, represents a prevalent auditory symptom affecting approximately 10-25% of the global adult population [1]. This condition substantially diminishes quality of life, frequently associating with psychological distress, sleep disturbances, and cognitive impairment [2]. The pathophysiological mechanisms underlying tinnitus remain multifactorial and incompletely elucidated, involving peripheral auditory dysfunction, central neural reorganization, and potential vascular components [3].

Hypertension constitutes a major cardiovascular risk factor with an estimated global prevalence exceeding 1.3 billion individuals, representing a significant public health challenge [4]. Beyond its well-established systemic complications, emerging evidence suggests that hypertension may influence auditory function through multiple pathophysiological pathways [5]. The cochlear microcirculation demonstrates exquisite sensitivity to hemodynamic alterations, as cochlear blood flow directly determines oxygen and nutrient delivery to metabolically active hair cells and neural structures [6]. Hypertensive vascular changes, including increased blood viscosity, endothelial dysfunction, and microvascular remodeling, may compromise cochlear perfusion and contribute to auditory symptoms [7].

Several epidemiological investigations have explored the relationship between hypertension and

tinnitus, yielding inconsistent findings. A systematic review and meta-analysis demonstrated a pooled odds ratio of 1.37 (95% CI: 1.16-1.62) for the association between hypertension and tinnitus [8]. However, the relationship appears complex and potentially age-dependent, with one large cross-sectional analysis reporting significant associations in younger adults (20-39 years) but not in older age groups [9]. Another investigation found hypertension prevalence of 44.4% among tinnitus patients compared to 31.4% in controls without tinnitus [10]. Furthermore, a recent South African study reported higher tinnitus prevalence (41.5%) in hypertensive adults compared to normotensive controls (22.8%), with grade 3 hypertension showing the strongest association [11].

The mechanisms potentially linking hypertension to tinnitus include compromised cochlear microcirculation, structural damage to the stria vascularis, alterations in endocochlear potential, and potential ototoxicity of antihypertensive medications [12]. Animal studies have demonstrated that systemic blood pressure changes directly influence cochlear blood flow, with autoregulatory mechanisms attempting to maintain stable perfusion [13]. Additionally, chronic hypertension may induce inner ear hemorrhage or ischemic changes that precipitate auditory dysfunction [14].

Despite accumulating evidence, significant research gaps persist regarding the precise nature and magnitude of the hypertension-tinnitus relationship, the influence of hypertension severity and duration on tinnitus characteristics, and the clinical implications for integrated cardiovascular-audiological care. Furthermore, most available data derive from population-based surveys with limited clinical characterization of both conditions.

Aim of the study: This investigation aimed to examine the association between hypertension and tinnitus in a clinically well-characterized sample, to assess whether hypertension severity correlates with tinnitus prevalence and characteristics, and to identify clinical factors that may mediate this relationship.

Materials and Methods

Study Design and Setting: This cross-sectional comparative study was conducted at the Department of Otolaryngology and Cardiology.

Sample Size Calculation: Sample size was calculated based on previous literature reporting tinnitus prevalence of approximately 45% in hypertensive patients and 30% in normotensive controls. Using a two-sided significance level of 0.05 and power of 80%, with an anticipated effect size (Cohen's h) of 0.31, the minimum required sample size was calculated as 130 participants per group. Accounting for potential 10% dropout, we aimed to recruit 143 participants in each group.

Participants

Inclusion criteria for the hypertensive group comprised: (1) age 35-70 years; (2) documented hypertension diagnosis according to current guidelines (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg on multiple occasions, or current antihypertensive medication use); (3) hypertension duration ≥6 months; (4) willingness to participate. The control group consisted of age-matched (±3 years) and sex-matched normotensive individuals recruited from general health screening programs.

Exclusion criteria for both groups included: (1) history of chronic ear disease (chronic otitis media, cholesteatoma, otosclerosis); (2) previous ear surgery; (3) occupational noise exposure exceeding 85 dB for >2 hours daily; (4) current use of known ototoxic medications (aminoglycosides, platinum-based chemotherapy, loop diuretics at high doses); (5) neurological disorders affecting auditory pathways; (6) acoustic neuroma or other cerebellopontine angle pathology; (7) diabetes mellitus; (8) severe renal impairment (eGFR <30 mL/min/1.73m²); (9) active psychiatric disorders; (10) pregnancy.

Hypertension Classification: Hypertension severity was classified according to current European Society of Cardiology guidelines: Grade 1 (systolic 140-159 mmHg or diastolic 90-99 mmHg), Grade 2 (systolic 160-179 mmHg or diastolic 100-109 mmHg), and Grade 3 (systolic ≥180 mmHg or diastolic ≥110 mmHg). Blood pressure measurements were obtained using standardized protocols with validated automated devices after 5 minutes of rest in a seated position, with the average of three readings recorded.

Tinnitus Assessment: Tinnitus presence was determined through direct questioning: "Have you experienced ringing, buzzing, or other sounds in your ears or head not caused by external sounds during the past 12 months?" Participants endorsing tinnitus underwent comprehensive evaluation including:

1. **Tinnitus characteristics:** laterality (unilateral right, unilateral left, bilateral), quality (tonal, noise-like, pulsatile), frequency (constant, intermittent), and duration.
2. **Tinnitus Handicap Inventory (THI):** A validated 25-item questionnaire assessing tinnitus-related disability across functional, emotional, and catastrophic subscales. Total scores range 0-100, with higher scores indicating greater handicap. Severity classification: slight (0-16), mild (18-36), moderate (38-56), severe (58-76), catastrophic (78-100).

3. **Visual Analog Scale (VAS):** Participants rated tinnitus loudness and annoyance on 0-10 scales.

Audiological Evaluation: Pure-tone audiometry was performed in a sound-treated booth using a calibrated clinical audiometer (Interacoustics AC40, Denmark). Air conduction thresholds were obtained at frequencies 250, 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz, with bone conduction assessed at 500-4000 Hz when indicated. Pure-tone average (PTA) was calculated as the mean threshold at 500, 1000, 2000, and 4000 Hz. Hearing loss was classified according to World Health Organization criteria: normal (<25 dB HL), mild (26-40 dB HL), moderate (41-60 dB HL), moderately severe (61-80 dB HL), severe (>80 dB HL).

Tympanometry and acoustic reflex testing were conducted using immittance audiometry to evaluate middle ear function.

Clinical Data Collection: A structured interview and medical record review captured demographic information (age, sex, education, occupation), cardiovascular parameters (hypertension duration, current blood pressure, antihypertensive medication use and classes), lifestyle factors (smoking status, alcohol consumption, physical activity), and medical history.

Statistical Analysis: Data were analyzed using SPSS version 26.0 (IBM Corporation, Armonk, NY, USA). Descriptive statistics included means with standard deviations for continuous variables and frequencies with percentages for categorical variables. Normality was assessed using Kolmogorov-Smirnov tests. Independent t-tests or Mann-Whitney U tests compared continuous variables between groups, while chi-square tests or Fisher's exact tests analyzed categorical variables. Pearson or Spearman correlation coefficients examined associations between continuous variables. Multivariate logistic regression assessed independent predictors of tinnitus presence, with odds ratios (OR) and 95% confidence intervals (CI) reported. Statistical significance was set at $p < 0.05$ (two-tailed).

Results

Demographic and Clinical Characteristics: A total of 286 participants were enrolled, comprising 143 hypertensive patients and 143 normotensive controls. The groups were well-matched for age (hypertensive: 54.8 ± 9.2 years; normotensive: 53.6 ± 9.7 years; p = 0.289) and sex distribution (hypertensive: 58.0% male; normotensive: 55.2% male; p = 0.629). Table 1 presents comprehensive baseline characteristics.

In the hypertensive group, mean systolic blood pressure was 152.4 ± 16.8 mmHg and diastolic blood pressure was 94.6 ± 11.3 mmHg. Mean duration of hypertension was 7.8 ± 4.6 years. Hypertension grade distribution comprised: Grade 1 (n=62, 43.4%), Grade 2 (n=58, 40.6%), and Grade 3 (n=23, 16.1%). Antihypertensive medication use was documented in 89.5% of hypertensive patients, with common classes including angiotensin-converting enzyme inhibitors (38.5%), angiotensin receptor blockers (32.2%), calcium channel blockers (28.7%), beta-blockers (24.5%), and diuretics (19.6%).

Tinnitus Prevalence and Characteristics: Tinnitus prevalence was significantly higher in hypertensive patients (67 of 143, 46.9%) compared to normotensive controls (41 of 143, 28.7%) ($\chi^2 = 10.44$, $p < 0.001$). Among participants with tinnitus, bilateral presentation was more common in hypertensive patients (46 of 67, 68.7%) than controls (19 of 41, 46.3%) ($\chi^2 = 6.34$, $p = 0.012$). The hypertensive group demonstrated higher proportions of constant tinnitus (59.7% vs 41.5%, p = 0.048) and tonal quality (52.2% vs 36.6%, p = 0.097).

Mean tinnitus duration was 4.2 ± 3.8 years in hypertensive patients and 3.1 ± 2.9 years in normotensive controls (p = 0.128). Pulsatile tinnitus, though relatively uncommon overall, showed higher prevalence in the hypertensive group (13.4% vs 4.9%, p = 0.127).

Tinnitus Severity: Among participants with tinnitus, mean THI scores were significantly higher in hypertensive patients (38.4 ± 22.6) compared to normotensive controls (26.8 ± 18.3) (t = 2.98, p = 0.003). THI severity classification revealed that moderate-to-catastrophic tinnitus affected 52.2% of hypertensive patients with tinnitus versus 31.7% of normotensive participants with tinnitus ($\chi^2 = 4.68$, p = 0.031). VAS ratings for tinnitus loudness (hypertensive: 5.8 ± 2.4; normotensive: 4.6 ± 2.1; p = 0.011) and annoyance (hypertensive: 6.2 ± 2.6; normotensive: 4.9 ± 2.3; p = 0.008) were significantly elevated in the hypertensive group.

Association between Hypertension Severity and Tinnitus: Analysis of hypertension grade demonstrated differential tinnitus prevalence: Grade 1 (24 of 62, 38.7%), Grade 2 (33 of 58, 56.9%), and Grade 3 (10 of 23, 43.5%) ($\chi^2 = 4.92$, p = 0.085). Compared to normotensive controls, Grade 2 hypertension showed the strongest association with tinnitus presence (OR = 3.45; 95% CI: 1.82-6.54; $p < 0.001$), followed by Grade 3 (OR = 2.08; 95% CI: 0.84-5.16; p = 0.112) and Grade 1 (OR = 1.58; 95% CI: 0.84-2.97; p = 0.156). Duration of hypertension demonstrated significant positive correlation with THI scores in hypertensive patients with tinnitus ($r = 0.418$, $p < 0.001$) and with tinnitus loudness VAS ($r = 0.362$, p = 0.003). Participants with hypertension duration ≥10 years exhibited higher tinnitus prevalence

(58.8%) compared to those with duration <10 years (42.1%) (p = 0.048).

Audiometric Findings: Mean PTA values were higher in hypertensive patients (right ear: 28.6 ± 12.4 dB HL; left ear: 29.2 ± 13.1 dB HL) compared to normotensive controls (right ear: 22.8 ± 11.6 dB HL; left ear: 23.4 ± 12.3 dB HL) (p = 0.001 for both ears). The prevalence of hearing loss (PTA >25 dB HL) was 58.7% in hypertensive patients versus 39.9% in controls (χ^2 = 10.21, p = 0.001). Among participants with tinnitus, hearing loss coexisted in 76.1% of hypertensive patients compared to 63.4% of normotensive controls (p = 0.141). However, 23.9% of hypertensive patients with tinnitus demonstrated normal hearing, suggesting hearing-independent mechanisms.

Multivariate Analysis: Logistic regression analysis, adjusting for age, sex, smoking status, body mass index, and hearing loss, confirmed hypertension as an independent predictor of tinnitus presence (adjusted OR = 2.24; 95% CI: 1.32-3.81; p = 0.003). Other significant predictors included hearing loss (adjusted OR = 3.87; 95% CI: 2.26-6.63; p < 0.001) and current smoking (adjusted OR = 1.92; 95% CI: 1.08-3.41; p = 0.026).

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Characteristic	Hypertensive Group (n=143)	Normotensive Group (n=143)	p-value
Age (years), mean ± SD	54.8 ± 9.2	53.6 ± 9.7	0.289
Male sex, n (%)	83 (58.0)	79 (55.2)	0.629
BMI (kg/m²), mean ± SD	28.4 ± 4.3	25.8 ± 3.6	<0.001
Current smoker, n (%)	38 (26.6)	32 (22.4)	0.412
Alcohol consumption, n (%)	42 (29.4)	45 (31.5)	0.704
Systolic BP (mmHg), mean ± SD	152.4 ± 16.8	118.6 ± 9.4	<0.001
Diastolic BP (mmHg), mean ± SD	94.6 ± 11.3	76.2 ± 6.8	<0.001
Duration of hypertension (years), mean ± SD	7.8 ± 4.6	N/A	-
Antihypertensive medication use, n (%)	128 (89.5)	N/A	-
PTA right ear (dB HL), mean ± SD	28.6 ± 12.4	22.8 ± 11.6	0.001
PTA left ear (dB HL), mean ± SD	29.2 ± 13.1	23.4 ± 12.3	0.001
Hearing loss present, n (%)	84 (58.7)	57 (39.9)	0.001

BMI = body mass index; BP = blood pressure; PTA = pure-tone average; N/A = not applicable; SD = standard deviation

Table 2: Tinnitus Prevalence and Characteristics by Study Group

Characteristic	Hypertensive Group (n=143)	Normotensive Group (n=143)	p-value
Tinnitus Prevalence			
Any tinnitus, n (%)	67 (46.9)	41 (28.7)	<0.001
Laterality (among those with tinnitus)			
Bilateral, n (%)	46 (68.7)	19 (46.3)	0.012
Unilateral right, n (%)	11 (16.4)	12 (29.3)	0.109
Unilateral left, n (%)	10 (14.9)	10 (24.4)	0.214
Quality			
Tonal, n (%)	35 (52.2)	15 (36.6)	0.097
Noise-like, n (%)	23 (34.3)	21 (51.2)	0.072
Pulsatile, n (%)	9 (13.4)	2 (4.9)	0.127
Frequency			
Constant, n (%)	40 (59.7)	17 (41.5)	0.048
Intermittent, n (%)	27 (40.3)	24 (58.5)	0.048
Tinnitus Severity			
THI score, mean ± SD	38.4 ± 22.6	26.8 ± 18.3	0.003
VAS loudness (0-10), mean ± SD	5.8 ± 2.4	4.6 ± 2.1	0.011
VAS annoyance (0-10), mean ± SD	6.2 ± 2.6	4.9 ± 2.3	0.008
THI Severity Classification			
Slight (0-16), n (%)	12 (17.9)	12 (29.3)	0.159
Mild (18-36), n (%)	20 (29.9)	16 (39.0)	0.315
Moderate (38-56), n (%)	21 (31.3)	10 (24.4)	0.411
Severe (58-76), n (%)	10 (14.9)	3 (7.3)	0.236
Catastrophic (78-100), n (%)	4 (6.0)	0 (0)	0.093

THI = Tinnitus Handicap Inventory; VAS = Visual Analog Scale; SD = standard deviation

Table 3: Association between Hypertension Grade and Tinnitus Characteristics

Parameter	Grade 1 HTN (n=62)	Grade 2 HTN (n=58)	Grade 3 HTN (n=23)	p-value
Tinnitus prevalence, n (%)	24 (38.7)	33 (56.9)	10 (43.5)	0.085
OR vs. controls (95% CI)	1.58 (0.84-2.97)	3.45 (1.82-6.54)*	2.08 (0.84-5.16)	-
Among those with tinnitus:				
THI score, mean ± SD	32.6 ± 20.4	42.8 ± 23.1	39.4 ± 24.8	0.194
Bilateral tinnitus, n (%)	15 (62.5)	24 (72.7)	7 (70.0)	0.687
Constant tinnitus, n (%)	13 (54.2)	21 (63.6)	6 (60.0)	0.722
PTA (dB HL), mean ± SD	26.4 ± 11.8	30.2 ± 12.6	29.8 ± 13.9	0.441
Hearing loss present, n (%)	17 (70.8)	26 (78.8)	8 (80.0)	0.712

HTN = hypertension; OR = odds ratio; CI = confidence interval; THI = Tinnitus Handicap Inventory; PTA = pure-tone average; SD = standard deviation; *p < 0.001

Discussion

This cross-sectional comparative study demonstrates a robust association between hypertension and increased tinnitus prevalence and severity in a well-characterized clinical sample. Our findings reveal that hypertensive patients exhibit 63% higher tinnitus prevalence (46.9% vs 28.7%) compared to age- and sex-matched normotensive controls, with significantly greater symptom severity as reflected by THI scores and subjective rating scales. These results align with accumulating epidemiological evidence while providing enhanced clinical characterization of the hypertension-tinnitus relationship [15].

The observed tinnitus prevalence in our hypertensive cohort (46.9%) corresponds closely to the 44.4% reported in a Spanish cross-sectional study [16], while exceeding the 41.5% documented in a South African investigation [17]. This consistency across diverse populations strengthens the evidence base for a meaningful association. Notably, our study's multivariate analysis confirmed hypertension as an independent tinnitus predictor (adjusted OR = 2.24) after controlling for established risk factors including age, hearing loss, and smoking, suggesting pathophysiological mechanisms beyond shared confounders.

The differential association observed across hypertension grades merits particular attention. Grade 2 hypertension demonstrated the strongest relationship with tinnitus (OR = 3.45), potentially reflecting a threshold effect wherein moderate elevations in blood pressure exert maximal impact on cochlear microcirculation before compensatory vascular adaptations occur. This finding partially aligns with the South African study demonstrating strongest associations with Grade 3 hypertension [18], although methodological differences in population characteristics and hypertension control may explain this discrepancy. The relatively weaker association with Grade 3 hypertension in our study might reflect more aggressive treatment in this subset or survivorship bias.

Several pathophysiological mechanisms may explain the hypertension-tinnitus association. First, chronic hypertension compromises cochlear microcirculation through structural vascular changes [19]. The stria vascularis, which maintains the endocochlear potential essential for auditory transduction, demonstrates particular vulnerability to hypertensive damage [20].

Experimental evidence indicates that hypertension-induced alterations in cochlear blood flow, reduced endocochlear potential, and increased perilymph sodium concentration collectively contribute to auditory dysfunction [21]. Second, hypertension-associated atherosclerosis may reduce arterial compliance and impair the autoregulatory capacity that normally maintains stable cochlear perfusion despite systemic pressure fluctuations [22]. Third, increased blood viscosity and endothelial dysfunction in hypertensive states may further compromise oxygen delivery to metabolically active cochlear structures [23].

The significantly higher THI scores in hypertensive patients with tinnitus (38.4 vs 26.8) represent a clinically meaningful difference, suggesting not merely increased prevalence but also enhanced symptom burden. This severity differential may reflect underlying disease chronicity, as evidenced by our finding that hypertension duration correlated positively with tinnitus handicap (r = 0.418). Alternatively, shared pathophysiological mechanisms involving sympathetic nervous system hyperactivity and psychological stress may amplify both cardiovascular and tinnitus-related distress [24].

Our audiometric findings revealed higher hearing thresholds in hypertensive patients, consistent with previous investigations demonstrating hypertension as a risk factor for hearing loss [25]. However, the observation that 23.9% of hypertensive patients with tinnitus maintained normal hearing thresholds suggests hearing-independent pathways, potentially involving altered neural processing or vascular tinnitus mechanisms. The higher prevalence of pulsatile tinnitus in hypertensive patients (13.4% vs

4.9%), though not reaching statistical significance in our sample, warrants clinical attention as pulsatile tinnitus may indicate vascular pathology requiring further investigation [26].

The bilateral tinnitus predominance in hypertensive patients (68.7% vs 46.3%) provides additional mechanistic insight, as bilateral presentation suggests systemic rather than focal pathology. This pattern supports the hypothesis that generalized microvascular dysfunction, rather than localized vascular events, mediates the hypertension-tinnitus relationship [27].

The potential role of antihypertensive medications requires consideration. Our study documented high medication use (89.5%) with various drug classes. While certain antihypertensives, particularly diuretics and calcium channel blockers, have been implicated in ototoxicity [28], distinguishing medication effects from underlying disease effects remains challenging in cross-sectional designs. Previous research suggesting associations between specific antihypertensive classes and tinnitus [29] highlights the need for longitudinal investigations examining tinnitus incidence relative to treatment initiation and modification.

Clinical implications of our findings include the potential value of routine blood pressure assessment in patients presenting with tinnitus, particularly when bilateral, constant, or pulsatile characteristics are present. Conversely, audiological screening and tinnitus inquiry may enhance comprehensive cardiovascular risk assessment. Integration of cardiovascular and audiological care could optimize outcomes for patients with coexisting conditions [30].

Study limitations include the cross-sectional design precluding causal inference and temporal sequence determination. Although our matching strategy and multivariate adjustment addressed confounding, residual confounding from unmeasured variables remains possible. The single-center recruitment may limit generalizability to populations with different ethnic compositions and healthcare access patterns. Tinnitus assessment relied partly on subjective reports, although validated instruments were employed. The relatively modest sample size, while adequately powered for primary comparisons, limited subgroup analyses by specific antihypertensive medication classes. Finally, our exclusion of diabetic patients, while reducing confounding, limits applicability to this common comorbid population.

Future research directions should include prospective cohort studies examining tinnitus incidence in relation to hypertension onset and treatment, investigations of specific antihypertensive medication effects through controlled trials or propensity-matched analyses, mechanistic studies employing advanced imaging techniques to assess cochlear blood flow and strial function in hypertensive patients, and intervention studies evaluating whether optimal blood pressure control reduces tinnitus burden.

Conclusion

This study demonstrates significant associations between hypertension and both increased tinnitus prevalence and greater symptom severity in a clinically characterized adult population. Hypertensive patients exhibited nearly twice the tinnitus prevalence of normotensive controls, with more severe handicap and predominantly bilateral presentation. Grade 2 hypertension showed the strongest association, and hypertension duration correlated with tinnitus severity. These findings support the hypothesis that hypertension-related microvascular dysfunction contributes to tinnitus pathogenesis through compromised cochlear perfusion and structural changes. The results underscore the importance of integrated cardiovascular-audiological assessment and suggest that blood pressure optimization may represent a potentially modifiable risk factor for tinnitus. Further prospective investigations are warranted to elucidate causal pathways and evaluate therapeutic interventions targeting the hypertension-tinnitus relationship.

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