# **Characteristics of Tinnitus and Etiology of Associated Hearing Loss: A Study of 123 Patients**

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**Abstract:** The aim of this study was to highlight the clinical characteristics of tinnitus and to attempt a quantitative assessment in relation to any underlying etiologies. We undertook to study a population of 123 patients attending a tinnitus clinic between 1998 and 2000. Their answers on a questionnaire allowed detailed evaluation of the characteristics of tinnitus, including such variables as the circumstances in which the tinnitus was first noticed and evaluation of its intensity and frequency. The patients each underwent a full neurootological examination with the aim of diagnosing an etiology. The great majority of tinnitus patients had an endocochlear deafness and, among these patients, acoustic trauma, endolymphatic hydrops, and presbyacusis were the commonest diagnoses (32%, 32%, and 23%, respectively). Of these patients, 93.7% with noise trauma and 86.9% with presbyacusis described their tinnitus as a stable, high-pitched whistling. Those patients with active Ménière's disease or Ménière's-like syndrome described a low-pitched buzzing tinnitus. Analysis of those patients with a stable high-pitched tinnitus associated with a high-frequency hearing loss shows a statistically significant correlation between the elevation of the audiometric thresholds and the loudness of the tinnitus. For a large majority of patients with tinnitus, therefore, audiometry provides an indirect test for evaluating the tinnitus.

*Key Words:* endocochlear hearing loss; frequency characteristics; loudness perception; tinnitus

Tinnitus constitutes an important public health problem; it is estimated that in France alone, there are 5 million tinnitus sufferers. Although previous studies have been descriptive, few have attempted a quantitative assessment of tinnitus qualities, such as its intensity, and have correlated it with the etiology of any associated hearing loss. We studied 123 patients attending a tinnitus clinic between 1998 and 2000. The assessment of patients with tinnitus was similar to that for patients with vertigo and hearing loss,

within the setting of a neurootology clinic. We submitted to them a questionnaire that enabled characterization of symptoms (e.g., circumstances of initial presentation, frequency, intensity, and perception of the tinnitus over time). The aim of the clinical examination was to diagnose the etiology of any underlying hearing loss.

Two main types of tinnitus were defined: (1) tinnitus of a stable intensity, almost always described as a high-pitched "whistling," found mainly in patients suffering from acoustic trauma, presbyacusis, and other lesion-type deafness, and (2) tinnitus of a fluctuating nature, found in patients with active, variable deafness (e.g., Ménière's disease or Ménière's-like syndrome), which was usually described as a lowpitched "buzzing."

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# **METHODS**

One hundred twenty-three patients were treated in a specialist tinnitus clinic at Gui de Chauliac<sup>1</sup> Hospital, Montpellier, France, between 1998 and 2000 [1]. Of these, 50 were female and 73 were male. The average age of the patients was 56 years (range, 17–80). All consultations had been motivated by patients' irritation with their tinnitus at best to their inability to bear it at worst.

## Assessment

The assessment was based on a questionnaire that was used to evaluate the clinical aspects of tinnitus (see the appendix at end of article). To allow its easy use during normal otological clinic visits, the questionnaire was based on a vertigo and hearing loss questionnaire already in use, and it was limited to certain aspects.

The duration of tinnitus was defined by the patient. Circumstances of appearance and mode of presentation were directed toward determining the events that might have caused the tinnitus (e.g., surgery or trauma, acoustic or otherwise). If the assessment did not uncover any obvious precipitating events, the tinnitus was considered to have arisen spontaneously. We were also interested in the manner of origin of the symptoms. Those types of tinnitus that appeared within 24 hours were classified as *sudden-onset tinnitus* (e.g., after acute acoustic trauma or temporal bone fracture), and those types of tinnitus with a less acute onset were classified as *progressive-onset tinnitus* (i.e., those that became established over several days or weeks).

When asked, patients usually liken the sound of their tinnitus to environmental noise, such as the sound of the wind or the sound of an insect. The frequency composition of tinnitus is extremely difficult both to describe and to understand, especially for those patients with low-pitched or complex tinnitus, even when using an audiometer or a synthesizer to define tinnitus pitch [2]. Again, so that this assessment could be carried out easily in a nonspecialist clinic, we specifically limited ourselves here to the two characteristics most frequently voiced by the patients when describing their tinnitus: high-pitched whistling or low-pitched buzzing. Therefore, we asked patients to designate their tinnitus as either of these categories or as "other" if neither category fit. To establish the relative intensity of tinnitus (i.e., loud or soft), we used a visual analog scale similar to that used for measuring pain intensity. This scale was graded from 0 to 100. The instructions were as follows: "Mark a point on the line between 0 and 100. Zero corresponds to an absence of tinnitus; 100 corresponds to the loudest tinnitus you can imagine. For example, a mark of 60 would correspond to a tinnitus which is as loud as the sound of your own voice when talking, 30 would be as loud as your own voice when whispering, and 90 would be the sound of your own voice when shouting."

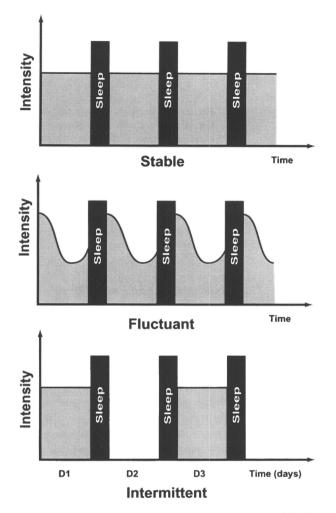
To investigate the temporal variation of tinnitus, patients were asked whether the intensity of their tinnitus was stable or whether it fluctuated during the course of the day. If the latter was the case, we asked them to evaluate this fluctuation on a visual analog scale, with the maximum value corresponding to the loudest intensity during the course of the day and the minimum value corresponding to the weakest intensity. Tinnitus that was present on some days but entirely absent on others was designated as intermittent (Fig. 1). Positional tinnitus (e.g., louder when lying down) was also documented. Other questions, such as spatial perception of tinnitus, are shown in the appendix.

# **Clinical Examination**

The aim of the clinical examination was to determine any underlying etiology that would explain the presence of tinnitus. All patients underwent a full otological, audiological, and vestibular examination, which included audiometry, evoked otoacoustic emissions (OAEs), evoked auditory brainstem potentials, and videonystagmoscopy (two CCD cameras mounted on lightproof goggles). If it was necessary for diagnosing the hearing loss, we also performed supplementary investigations: videonystagmography when hearing loss was associated with vertigo, magnetic resonance imaging when retrocochlear hearing loss was suspected, or magnetic resonance angiography, Doppler imaging of the local vasculature, and tomodensitometry of the temporal bone to evidence vascular problems.

Audiometry was performed at 0.125, 0.25, 0.5, 1.0, 2.0, 4.0, 6.0, and 8.0 kHz. Audiometric results were grouped into four main categories: normal hearing (thresholds <20 dB at each of the frequencies tested), conductive hearing loss, sensorineural hearing loss, and mixed loss. Sensorineural hearing loss was further subdivided into four groups: high-frequency hearing loss (in the frequency of 4–8 kHz), low-frequency hearing loss, and "dead" ear. The diagnosis of presbyacusis was reserved for those patients who were older than 60 years and demonstrated a bilateral, symmetrical, high-frequency

<sup>&</sup>lt;sup>1</sup>Gui de Chauliac (1300–1368) was a physician associated with the Montpellier School of Medicine; he wrote what became the standard book on surgery for the next two centuries. He recommended fumigation of the ear with ox urine, vinegar, and myrrh for certain cases of tinnitus.



**Figure 1.** Schematic representation of the perception of tinnitus intensity over time. Three categories of tinnitus emerged from our survey: tinnitus with a stable intensity over time, tinnitus perceived to fluctuate in relation to periods of sleep, and tinnitus perceived as intermittent (present on certain days but not on others).

hearing loss, with no other cause of hearing loss evident in the patient's history or on clinical examination.

# RESULTS

# Tinnitus and the Etiology and Anatomical Location of Hearing Loss

No objective tinnitus was present in the 123 patients in our study. All but one of the patients with tinnitus seen in this specialist clinic had an associated hearing loss.

*Tinnitus Associated with Deafness of Known Etiology* The great majority of patients (almost 90%) presented with tinnitus associated with deafness of known etiology (Table 1). Two-thirds of cases were made up of four etiologies: chronic noise exposure (23.6%), presbyacusis (18.7%), Ménière's disease (15.4%), or Ménière's-like syndrome (10.6%). The remaining patients had deafness associated with ototoxicity (4%) or genetic hearing loss (3.25%) or noticed it immediately after one of the following events: traumatic event (acute noise trauma, 2.44%; temporal bone fracture, 1.62%), middle ear surgery (2.44%), or acute otitis media (0.8%). Sudden deafness accounted for 1.62% of cases. One patient attending the tinnitus clinic was given a diagnosis of an acoustic neuroma, two patients of vascular loops in the internal auditory meatus, and two of cholesteatoma.

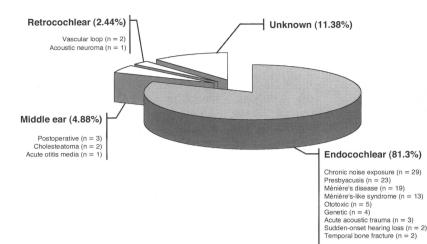
# Tinnitus of Unknown Origin

In a small proportion of patients (n = 14; 11.38%), it was not possible definitively to correlate the tinnitus with a known etiology or anatomical site. In this group, four types of tinnitus were identified. Sudden-onset tinnitus was diagnosed in five patients. In each case, the patients were able to state the exact time and date of onset of their tinnitus. The noise was heard in one ear only and was described as whistling and stable over time. These cases of sudden onset of tinnitus were not linked to a sudden deafness. However, four patients did have a presbyacusis, and one had a mild hearing loss in the higher frequencies.

Pulsatile tinnitus was identified in four patients. These patients described unilateral tinnitus with no precipitating factors. The audiometry was symmetrical and correlated with the age of the patient. In none of these

Table 1. Tinnitus and Diagnosis of Hearing Loss

|   | No. of<br>Patients | Percentage |
|---|--------------------|------------|
| Tinnitus and hearing loss of known etiology |                    |            |
| Chronic noise exposure                      | 29                 | 23.57      |
| Presbyacusis                                | 23                 | 18.69      |
| Ménière's disease                           | 19                 | 15.44      |
| Ménière's-like syndrome                     | 13                 | 10.57      |
| Ototoxic origin                             | 5                  | 4.06       |
| Genetic origin                              | 4                  | 3.25       |
| Acute acoustic trauma                       | 3                  | 2.44       |
| Postoperative effects (middle-ear surgery)  | 3                  | 2.44       |
| Sudden-onset hearing loss                   | 2                  | 1.62       |
| Temporal bone fracture                      | 2                  | 1.62       |
| Cholesteatoma                               | 2                  | 1.62       |
| Vascular loop                               | 2                  | 1.62       |
| Acute otitis media                          | 1                  | 0.81       |
| Acoustic neuroma                            | 1                  | 0.81       |
| Tinnitus of unknown etiology                |                    |            |
| Sudden-onset tinnitus                       | 5                  | 4.06       |
| Pulsatile tinnitus                          | 4                  | 3.25       |
| Head trauma                                 | 4                  | 3.25       |
| Unclassified origin                         | 1                  | 0.81       |



**Figure 2.** Tinnitus and anatomical location of hearing loss. Each portion of the chart represents tinnitus associated with an endocochlear hearing loss (81.3%), with middle-ear pathology (4.88%), with a retrocochlear hearing loss (2.44%), or with an unknown etiology (11.38%). Note the large incidence of tinnitus associated with an endocochlear hearing loss. The number of patients is represented by *n* (total, 123).

four patients did clinical or vascular examination identify an underlying cause.

In four patients, tinnitus was precipitated by head trauma (unilateral tinnitus in all). All had initial loss of consciousness. Even though deafness had not been described by any of the patients, audiometry unmasked in each patient a high-frequency loss that was more marked (10 dB) in the ear affected with tinnitus. It is impossible to say whether the deafness was linked to the head trauma (although this is likely) or whether it preceded it.

Tinnitus and normal hearing were observed in one patient. We were unable to classify this patient. At age 23, he described his tinnitus as "creaking"; it was precipitated by movement of his head. No abnormality was found on examination. Even though it was thought that his tinnitus had an origin in some head or neck joint and thus was a somatic noise, it was not possible to define it further.

Slightly more than 80% of the patients had an endocochlear hearing loss, 2.4% had retrocochlear loss, and 4.9% had hearing loss associated with middle-ear pathology (Fig. 2). Because such a significant number of patients with tinnitus had an endocochlear hearing loss (81.3%), we looked in more detail at this subgroup.

## **Endocochlear Hearing Loss and Tinnitus**

#### Endocochlear Hearing Loss Thresholds

Hearing losses were divided into four groups: high-frequency (60%), low-frequency (25%), flat losses (11%), and dead ear (2%). Evoked OAEs were performed in the first 50 patients only, because the high incidence of hearing thresholds greater than 30 dB in our patients meant that OAE assessment contributed little to the evaluation of the etiology.

# Endocochlear Hearing Loss and Variability of Intensity of Tinnitus Over Time

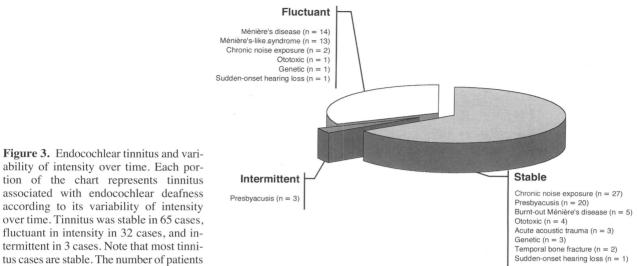
Of the patients from the endocochlear group, 65% had stable tinnitus, and 32% had fluctuating tinnitus. Only 3% of this group of patients had intermittent tinnitus (Fig. 3). In the case of certain pathological processes, patients described stable or fluctuating tinnitus equally (e.g., ototoxic and genetic hearing loss, sudden-onset deafness). Interestingly, 93% of the patients with chronic noise exposure and 87% of patients with presbyacusis described stable tinnitus. Both patients with a temporal bone fracture and the three patients with acute acoustic trauma described stable tinnitus (see Fig. 3).

Of patients with Ménière's disease or Ménière's-like syndrome, 84% described fluctuating tinnitus. Patients with active endolymphatic hydrops described fluctuation of their tinnitus during vertiginous attacks but, interestingly, also every day between crises, even when they thought that their hearing was stable. It is interesting to note that the remaining 16% of patients with Ménière's disease or syndrome had had it for more than 5 years; they had a stable hearing loss and no vertiginous attacks for 2 years, and they described their tinnitus as stable (see Fig. 3). All patients who described their tinnitus as fluctuating in intensity described a very clear increase in intensity of their tinnitus after periods of sleep (whether nighttime or siesta).

# Endocochlear Hearing Loss and Frequency Characteristics of Tinnitus

Of patients with endocochlear deafness, 77% described their tinnitus as a high-pitched whistling, and 21% described it as a low-pitched buzzing (Fig. 4). Only 2% of patients were unable to categorize their tinnitus into either of these categories.

Comparison between the characteristics of tinnitus (i.e., high- or low-pitched) and the etiology reveals that



over time. Tinnitus was stable in 65 cases, fluctuant in intensity in 32 cases, and intermittent in 3 cases. Note that most tinnitus cases are stable. The number of patients is represented by *n* (total, 100).
100% of the patients with acoustic trauma and 91.3% of the patients with presbyacusis categorized their tin-

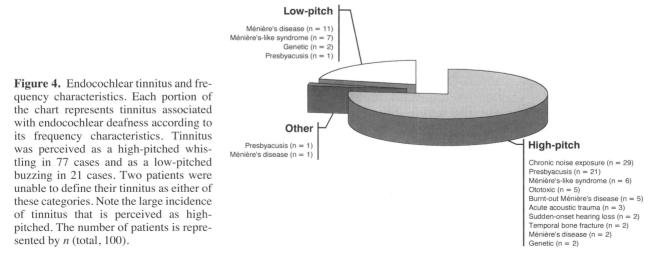
of the patients with presbyacusis categorized their tinnitus as high-pitched. In the same way, all the patients presenting with ototoxic or sudden-onset hearing loss, temporal bone fracture, or "burnt-out" Ménière's disease also described a high-pitched tinnitus. Conversely, the patients with active endocochlear hydrops described a low-pitched tinnitus in two-thirds of the cases. The four patients with genetic deafness described both categories equally (see Fig. 4).

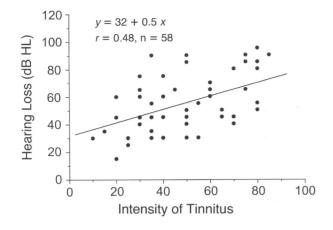
In our study, 58 of the 77 patients with a stable high-pitched tinnitus (77%) had a high-tone hearing loss. Therefore, we looked more closely at those patients with both a stable, high-frequency hearing loss and high-frequency tinnitus. The pure-tone thresh-

olds at 4, 6, and 8 kHz were averaged and correlated with the perceived intensity of each patient's tinnitus. Statistical analysis demonstrated that tinnitus is perceived as louder as the high-tone hearing thresholds are elevated and that this correlation is statistically significant (regression coefficient, r = 0.48; p < .001; Fig. 5).

# DISCUSSION

This study attests to the rare incidence of objective tinnitus: There was no case of objective tinnitus in this series. Obviously, our hospital population was selfselected and does not necessarily reflect the true epide-





**Figure 5.** Correlation between the intensity of tinnitus and hearing loss. This correlation was calculated for those patients with stable, high-pitched tinnitus and a high-pitched hearing loss. The intensity of the tinnitus (*x* axis) was measured on a visual analog scale graduated from 0 to 100. The high-frequency hearing loss (*y* axis) was calculated as a mean of the thresholds over three frequencies (4, 6, and 8 kHz). Each point represents the result from each patient. A line of the form y = a + bx was fitted to the measurements. The quality of the best-fitting linear regression curve is indicated by *r*, and the number of patients by *n*. Note that the intensity of high-pitched tinnitus is significantly correlated with high-frequency hearing loss (p < .001).

miology of tinnitus. However, large-scale epidemiological studies show that objective tinnitus is universally infrequent [3].

Except for one case, all the subjective tinnitus cases were accompanied by a hearing loss. This type of subjective tinnitus reflected a cochlear problem in the majority of cases: More than 80% were associated with an endocochlear hearing loss. Among the latter, acute and chronic noise trauma (32%), Ménière's disease and Ménière's-like syndrome (32%), and presbyacusis (23%) were the principal etiologies associated with tinnitus. By contrast, it was rare to find a tinnitus resulting from middle-ear disease (4.88%) or from retrocochlear pathology (2.44%). A series by Shulman [4] had similar findings, with a high incidence (25%) of Ménière's disease or Ménière's-like syndrome and a 10-15% incidence of tinnitus associated with middle-ear disease. Probably, in most of the 14 patients with tinnitus of unknown etiology, the tinnitus is associated with their underlying hearing loss, but it is not possible to be definite about this because whether their hearing loss preceded the onset of their tinnitus is unknown. Thus, we have not been able to correlate characteristics of tinnitus in this group of patients with tinnitus of unknown etiology, even though most have high-pitched stable tinnitus and a high-frequency hearing loss. For the four patients with pulsatile tinnitus, no etiological conclusions can be drawn.

Our results confirm that chronic noise exposure is the main cause of tinnitus [5, 6]. A more detailed analysis of this noise exposure group revealed that 83% of them were male. One reason for this could be that men are generally exposed more to industrial noise. Consistent with this finding, an imbalance was also found in the group with presbyacusis, of whom 74% were male. This overrepresentation could testify to the importance of noise exposure in the development of presbyacusis: That is, it is possible that hidden among those patients with presbyacusis are patients who experienced previous noise trauma.

The study of the etiology of the underlying deafness offers interesting results for understanding tinnitus, especially in patients with high-frequency hearing loss. A strong correlation was seen between clinical characteristics of tinnitus and the nature of the cochlear pathology. Tinnitus linked to acoustic trauma, presbyacusis, and other lesion-type deafness (e.g., ototoxic hearing loss with high-frequency hearing loss) was almost always described as a stable high-pitched noise. In cases of active Ménière's disease or Ménière's-like syndrome with low-frequency hearing loss, the tinnitus was almost always described as a fluctuating low-pitched noise, even between vertiginous crises. These results suggest that patients suffering from an irreversible lesion-type deafness (e.g., presbyacusis, acoustic trauma, temporal bone fracture, and "burnt-out" Ménière's disease) have tinnitus of a stable intensity, whereas those with an active, variable disease, such as endocochlear hydrops, have tinnitus of a fluctuating nature.

We have confirmed in this study that the general frequency of tinnitus pitch perceived by the patient is correlated in broad terms with the frequency of the hearing loss, in agreement with some other studies [7]. By simply having three categories of tinnitus pitch in the questionnaire (high pitch, low pitch, or other), we have avoided some of the problems associated with pitch matching using an audiometer. Studies that use an audiometer or synthesizer for pitch matching have found the process to be very difficult, both because of the difficulty that patients have in matching complex tinnitus and because of the possible modulation of the tinnitus by the external sound [8].

Importantly, we have also shown that in the majority of people with tinnitus (i.e., those with a high-frequency stable hearing loss), the loudness perception of their tinnitus measured on a visual analog scale is strongly and statistically correlated with the degree of their hearing loss. This is not the case in studies that have used audiometers to attempt to match the loudness of tinnitus, wherein often a soft sound (<10 dB) appears to

match their tinnitus, despite the intense irritation that tinnitus engenders [9]. Loudness matching using audiometry is a time-consuming, complex investigation [10]. If an audiometer is used, it can be difficult for patients to agree on an equivalent loudness; this may be owing in part to recruitment associated with their hearing loss and in part to the fact that patients can be confused between loudness and irritation that is induced by their tinnitus. There is a feeling that loudness matching per se is not necessarily relevant to patients and what is more pertinent is a personal loudness level [11]. The visual analog scale that we use works in just this way. Its advantages are that it is simple to explain and understand, it is easy to use for both the clinician and the patient and, as discussed, there is a strong correlation between this scale and the audiometric thresholds. This scale is also very useful in evaluating the efficacy during and after treatment.

This study has raised interesting findings about the fluctuation of tinnitus after periods of sleep, especially in patients with low-frequency tinnitus. Many studies report tinnitus associated with sleep disturbance, but a literature review does not reveal any other studies reporting such marked increase in tinnitus on waking. We feel that this aspect of tinnitus requires further investigation.

# CONCLUSION

We have shown in this study that audiometry can be used as a simple indirect test to evaluate tinnitus, as it correlates well with a visual analog scale of tinnitus intensity in the majority of our patients. We believe that it is important to develop this clinically based approach, and we will continue to investigate further correlations in our clinic.

# ACKNOWLEDGMENTS

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# **APPENDIX.** Tinnitus Clinical Evaluation Sheet

| Surname / First name:                               | Date:  |  |  |  |  |
|---|--|--|--|--|--|
| Date of birth:                                      | Age:   |  |  |  |  |
| Profession:   | Identification number:   |  |  |  |  |
|   |  |  |  |  |  |
| History   |  |  |  |  |  |
| Duration: Right ear (R) : .                         | ; Left ear (L) : ; Head : ;  |  |  |  |  |
| Circumstances of presenta                           | tion : Spontaneous<br>: Acute acoustic trauma<br>: Other   |  |  |  |  |
| Mode of presentation                                | : Progressive<br>: Sudden (< 24 hours)   |  |  |  |  |
| : Head  | ; ear:; Left ear :; Right and left ear :;<br>: lateralised to R – central – lateralised to L<br>ide the head: lateralised to R – central – lateralised L |  |  |  |  |
| Pitch : Continuous no<br>: Discontinuous<br>: Other | : Low-pitched, buzzing   |  |  |  |  |
| : Fluctuant: to .                                   | Intensity         : Stable:  |  |  |  |  |
| Any factors modifying the i                         | intensity of the tinnitus : Additional acoustic trauma<br>: Sleep<br>: Lying down<br>: Previous treatment  |  |  |  |  |
| Associated symptoms                                 | : Hyperacusis: R ear / L ear<br>: Pressure around temporal: R side / L side<br>: Diplacusis: R side / L side<br>: Vertigo:                               |  |  |  |  |

| Examination                        |  |  |              |   |  |  |  |
|------------------------------------|--|--|--------------|---|--|--|--|
| Otoscopy: Right ear :; Left ear :; |  |  |              |   |  |  |  |
| Clinico                            | ıl vestibular exam   | : Romberg test<br>: Unterberger's<br>: Videonystagr  | test : D / G | : Spontaneous nystagmus<br>: Evoked nystagmus |  |  |  |
| Audiog                             | : Condu<br>: Sensor  | : Normal: R / L<br>: Conductive hearing loss: R / L<br>: Sensorineural hearing loss : High-frequency D / G<br>: Low-frequency D / G<br>: Flat loss D / G<br>: Mixed hearing loss : D / G |              |   |  |  |  |
|                                    |  | 0  | ., 0         |   |  |  |  |
| Brainsi                            | Brainstem evoked response audiometry :           : R : latency I-III :         :           : R : latency I-III :         :           : R : latency I-V :         : |  |              |   |  |  |  |
| Videon                             | ystagmography  | : D :  | ;;           | G :;  |  |  |  |
| CT Sca                             | n  | : D :  | ;            | G :;  |  |  |  |
| Magne                              | tic Resonance Imag   | ging: : D :  | ;            | G :;  |  |  |  |
| Other :                            |  |  |              |   |  |  |  |
|                                    |  |  |              |   |  |  |  |
| Conclusions                        |  |  |              |   |  |  |  |

## REFERENCES

- 1. Stephens SDG. The treatment of tinnitus—a historical perspective. *J Laryngol Otol* 98:963–972, 1984.
- 2. Feldmann H. Homolateral and contralateral masking of tinnitus by noise band and by pure tones. *Audiology* 10:138–144, 1971.
- Shulman A. Epidemiology of Tinnitus. In A Shulman, J-M Aran, J Tonndorf, et al (eds), *Tinnitus: Diagnosis/ Treatment*. Philadelphia: Lea & Febiger, 1991:237–247.
- Shulman A. Diagnosis of Tinnitus. In M Kitahara (ed), *Tinnitus—Pathophysiology and Management*. Tokyo: Igaku-Shoin, 1988:53–63.
- Chung DY, Gannon RP, Mason K. Factors affecting the prevalence of tinnitus. *Audiology* 23:441–452, 1984.
- 6. Dieroff HG, Meissner W. Prevalence of tinnitus in noiseinduced hearing loss. In *Proceedings of the Third Inter-*

national Tinnitus Seminar. Karlsruhe: Harsch Verlag, 1987:159-161.

- 7. Douek E, Reid J. The diagnostic value of tinnitus pitch. *J Laryngol Otol* 82:1039–1042, 1968.
- Tyler RS. The Psychophysical Measurement of Tinnitus. In J-M Aran, R Dauman (eds), *Tinnitus 91: Proceedings* of the Fourth International Tinnitus Seminar. Amsterdam: Kugler Publications, 1992:17–26.
- 9. Goodwin PE, Johnson RM. The loudness of tinnitus. *Acta Otolaryngol (Stockh)* 90:353–359, 1980.
- Penner MJ. Variability in matches to subjective tinnitus. J Speech Hear Res 27:247–249, 1983.
- Hallam RS, Jakes SC, Chambers C, Hinchcliffe R. A comparison of different methods for assessing the "intensity" of tinnitus. *Acta Otolaryngol (Stockh)* 99:501–508, 1985.