ORIGINAL ARTICLE

Sound Therapy in Sudden Deafness

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KEYWORDS
Sudden hearing loss; Sudden deafness; Stress; Sound treatment

Abstract
Introduction and goals: Idiopathic sudden sensorineural hearing loss is a hearing disorder of unknown cause. The spontaneous recovery rate ranges from 50% to 75% of the patients. Scientific experiments on animals support the present study in patients with sudden deafness treated with sounds.

Patients and methods: During the period 2003–2009, patients with idiopathic sudden sensorineural hearing loss were administered steroids, piracetam and antioxidants, together with the addition of sounds by means of music and words.

Results: Comparing the results of patients treated with medication (n=65) and those treated with medication and sounds (n=67), it was observed that patients treated with medication and sounds had higher recovery. Within the group of patients treated with medication and sounds, 25 (37%) experienced complete recovery, 28 (42%) good recovery, 11 (16%) slight recovery and 3 (5%) poor or no recovery.

Conclusion: The patients who recovered more than half of their audition accounted for 54% in the group treated with medication and for 79% in the group of patients receiving medication and sounds. Auditory recuperation showed no alterations, at least up to 12 months after therapy. © 2011 Elsevier España, S.L. All rights reserved.

PALABRAS CLAVE
Sordera súbita; Hipoacusia brusca; Estrés; Tratamiento sonoro

Terapia sonora en sordera súbita

Resumen
Introducción y objetivos: La hipoacusia neurosensorial súbita idiopática es un trastorno auditivo de causa desconocida. El índice de recuperación espontánea puede variar, según la literatura, en un rango del 50–75% de los pacientes. Experimentos científicos mediante terapia sonora en animales hipoacusícos avalan el presente estudio en pacientes con sordera súbita tratados con sonidos.

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Introduction

Sudden idiopathic sensorineural hypoacusis or hearing loss develops over a period of hours. It may be accompanied by tinnitus and dizziness. Its incidence ranges from 5 to 160/100,000.1 Various possible aetiologies have been suggested, such as rupture of the cochlear membrane, microangiopathic processes, viral infections, autoimmune disorders, Meniere’s syndrome, schwannoma and meningioma. Stress is also present in numerous cases of sudden deafness. It has even been suggested that pathological activation of the cellular stress pathway could affect nuclear factor kappa-B in the cochlea and act as a possible mechanism of sudden deafness.2,3

According to the literature,4 sudden deafness has a spontaneous recovery in 50%–75% of cases. Consequently, the results of the different treatments proposed in many studies are difficult to ensure.

There is no agreement regarding the treatment of choice for sudden deafness. Different drug therapies are commonly used: corticosteroids, vaso- dilators, antiviral and antioxidant agents. Corticosteroids are the most commonly used drugs, although a review by Cochrane5 questions this statement. According to this review, the value of corticosteroids in the treatment of sudden deafness is not clear; they are administered in different formulations, doses and durations, and the evidence from randomised controlled trials is contradictory, partly due to studies based on too few patients. Other authors even consider not giving any treatment.6

The application of hyperbaric oxygen appears to improve hearing, but the level of significance is not clear.7 Intratympanic steroids are recommended in cases where systemic corticosteroid therapy has proven ineffective.1

This study is based on animal experiments. The first consisted in acoustic deprivation.8 A group of chinchillas underwent bilateral acoustic trauma. Immediately after, ossiculectomy was performed on one ear. The removal of the ossicles of one ear prevented environmental noise from reaching the cochlea. In the non-operated ear, the acoustic trauma caused damage to ciliated cells and hearing loss, while the operated ear, which received less environmental sound, suffered more damage to ciliated cells and greater hearing loss. The second study consisted in acoustic conditioning.9 A number of guinea pigs underwent bilateral acoustic trauma. They were then divided into 2 groups, with one of them receiving sound therapy. The group without sound therapy displayed hearing loss, while the sound therapy group recovered hearing. The third study was an animal model of presbycusis.10 As young adults, C57BL/6J mice developed genetically determined progressive sensorineural hearing loss at high frequencies. The animals were divided into 2 groups, with one of them receiving sound therapy. The group without sound therapy developed presbycusis and damage to the external and internal ciliated cells, spiral ganglion and anterior ventral cochlear nucleus. The sound therapy group developed less presbycusis and less cell damage to the mentioned structures. The fourth study was based on the cortical tonotopic map.11 Various cats were subjected to acoustic trauma and the representation of cochlear frequencies at the cortical level was determined. The acoustic trauma caused a reorganisation of the cortical tonotopic map, causing deafness, tinnitus and hyperacusis. Those cats which received environmental noise did not display reorganisation of the cortical tonotopic map. They recovered from hearing loss, and tinnitus and hyperacusis disappeared.

In the present study, patients with sudden deafness were treated with medication (corticosteroids, piracetam and antioxidants) or with the same medication plus sound therapy. We analysed recovery of audiovestibular symptoms in both groups.

Materials and Methods

Type of Study

This was a non-randomised study of a retrospective case series.

Patients

Inclusion criteria were: unilateral sensorineural hearing loss with an evolution of hours, attended within 30 days of onset, with a loss of over 30 dB HL in at least 3 consecutive frequencies of the audiogram and with an unknown cause. A total of 65 patients received medication (deflazacort, piracetam and alpha-tocopherol) and a further 67 were treated with the same medication plus sound therapy. Participants were treated between the years 2003 and 2009. Patients between 2003 and 2006 received only medication and those treated between 2007 and 2009 were given the same medication plus sound therapy.

Conclusión: El 54% de los pacientes del grupo con medicación ha recuperado más de la mitad de la audición perdida y el 79% del grupo que recibió medicación y terapia sonora. La recuperación auditiva no sufró alteraciones, al menos, en los siguientes 12 meses del tratamiento.

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Medication

All patients (n=132) received outpatient drug treatment with deflazacort (30 mg every 8 h for 1 week, every 12 h for 3 days and every 24 h for another 3 days), piracetam (1200 mg every 8 h for 1 week), alpha-tocopherol (400 IU every 24 h for 30 days) and omeprazole (20 mg daily for 28 days).

Sound Therapy

We used a combination of music and speech, either through the use of those songs most pleasing to each patient or through listening to radio. The intention was to ensure stimulation of the whole cochlear spectrum and its auditory cortical representation, thus reaching various emotional brain regions (prefrontal, temporal or parietal) through neural networks, in order to stimulate recovery.

The practical manner to obtain the correct intensity for each patient was the following: patients were instructed to place the headset on their healthy ear and then change it to their pathological ear as soon as they began to hear the music slightly (range of 60±10 dB). In some cases, patients could not hear the sounds through their pathological ear at that time due to their sudden deafness, but later, as hearing recovered, they were able to hear them. The same methodology was applied even in cases of cophosis. The duration of this sound therapy was 12 h per day for 30 days. This sound therapy was applied to the second group of patients (n=67) in conjunction with medication.

Complementary Tests

The clinical history collected information on symptoms of vertigo and stress (vertigo was identified as rotary dizziness and stress was identified by the presence of stressful situations related to work, family, studies, disease, economy or anxiety).

In addition to the clinical history and the stress factor survey, we conducted pure tone audiometry, speech audiometry, tinnitus test (determination of the frequency and intensity of tinnitus), impedance audiometry and magnetic resonance imaging with gadolinium.

The speech test was conducted through the emission of a series of phonetically balanced words, recorded on a compact disc. Their frequencies were 500, 1000 and 2000 Hz, corresponding to the central frequencies in the audiogram. This test reported the understanding or intelligibility threshold, which measured the intensity (in decibels) at which the patient responded to 100% of the words emitted.

The degree of hearing loss was classified as: mild (mean of 26–34 dB HL at 250, 500, 1000, 2000, 4000 and 8000 Hz), moderate (mean of 35–54 dB HL), severe (mean of 55–74 dB HL) or profound (mean >74 dB HL).

The classification criteria for the configuration of audiometric curves were as follows: a plane curve was defined when the difference was less than 20 dB intensity between the low (<1000 Hz), medium (1000–3000 Hz) and high frequencies (>3000 Hz). An ascending curve was defined when the hearing loss at low frequencies was greater than 20 dB of the intensity at high frequencies. A descending curve was defined when the hearing loss at high frequencies was greater than 20 dB of the intensity at low frequencies. A curve in middle frequencies (U type) was defined when the hearing loss at intermediate frequencies was greater than 20 dB of the intensities at low and high frequencies.

Evaluation

The clinical history and pure tone audiometry were performed on admission of patients with sudden deafness, and repeated at 1, 3, 6 and 12 months. Hearing recovery was based on the recovery rate and the speech test.

The recovery rate (%) was calculated in order to determine overall hearing recovery. Hearing thresholds were calculated using the mean of the thresholds at 250, 500, 1000, 2000, 4000 and 8000 Hz. Recovery rate (%)=[[initial thresholds−final thresholds]/[initial thresholds−final thresholds in the contralateral ear]]×100. This recovery rate was categorized as: complete recovery (>90%), good recovery (51%–90%), slight recovery (21%–50%) and poor recovery or no recovery (0%–20%).

Statistical Procedures

The theoretical results for the calculation of sample size suggested a minimum sample size of 31 patients in order to obtain a 95% confidence when α=0.05, indicating a statistically significant difference of P<.05.

We conducted the following nonparametric tests: z test with Yates correction to study gender, age, vertigo, tinnitus, hearing capacity before start of therapy, results of medication plus sound therapy and season of the year; the Spearman test was used to compare the audiometric configuration and level of hearing loss; the Chi-square test was used to study the improvement in speech audiometry due to sound therapy (Chi-square compares categorical responses between 2 groups).

Ethical Considerations

The study was conducted according to the principles of the Declaration of Helsinki (1975, 1983). The research protocol, patient information sheet and written informed consent form were approved by the Ethics Committee of the hospital.

Results

Patients

The control group comprised 65 patients who only received medication (32 females and 33 males) with a mean age of 44.1±18.3 years (range 17–80 years). In the control group, sudden deafness affected the right ear in 31 cases and the left ear in 34 cases. The other group included 67 patients (31 females and 36 males) who were treated with the same medication plus sound therapy and had a mean age of 44.6±17.9 years (range 13–76 years). Sudden deafness affected the right ear in 33 cases and the left ear in 34 cases.
Recovery Rate of Sudden Deafness

Recovery Rate and Gender

The mean recovery rate in the medication group was 38.6% among females and 44.5% among males. This difference was not statistically significant (P=.549). The mean recovery rate in the group treated with medication plus sound therapy was 60.4% among females and 79.3% among males. This difference was not statistically significant (P=.097).

Recovery Rate and Age

The recovery rate showed a dependence on age. In the medication group, patients aged up to 25 years (10/65) presented a mean recovery rate of 63.4% and those aged over 25 years (55/65) presented a mean recovery rate of 38.5%. This difference was statistically significant (P<.005). In the group of patients treated with medication plus sound therapy, patients aged up to 25 years (14/67) presented a mean recovery rate of 89.7% and those aged over 25 years (51/65) presented a mean recovery rate of 65.8%. This difference was statistically significant (P=.03). The cut-off age of 25 years was selected because the recovery rate reached a minimum of 50% in the control group.

Recovery Rate and Vertigo

In the medication group, patients with vertigo (30/65) presented a mean recovery rate of 32.3% and patients without vertigo (35/65) presented a mean recovery rate of 51.1%. This difference was statistically significant (P=.050). In the group treated with medication plus sound therapy, patients with vertigo (28/67) presented a mean recovery rate of 65.7% and patients without vertigo (39/65) presented a mean recovery rate of 73.2%. This difference was not statistically significant (P=.597).

Recovery Rate and Tinnitus

In the group treated with medication plus sound therapy, 62 (93%) patients suffered sudden deafness with tinnitus. Tinnitus persisted in 28 (45%) patients after completing the treatment protocol, reaching a mean recovery rate of 59.8%, while in the 34 (55%) patients in whom tinnitus remitted after treatment, the recovery rate reached 85.6% (P=.030).

Recovery Rate and Prior Auditive Capacity

In the group treated with medication, 43 (66%) patients with normal hearing before suffering sudden deafness presented a mean recovery rate of 45.4%, whilst the 22 (34%) patients who suffered prior sensorineural hearing loss presented a mean recovery rate of 38.5%. There was no statistical significance (P=.532). In the group treated with medication plus sound therapy, the 41 (63%) patients with normal prior hearing presented a mean recovery rate of 72.4%, whilst the 24 (37%) patients with prior sensorineural hearing loss presented a mean recovery rate of 68.7%. The difference was not statistically significant (P=.892).

Recovery Rate and Audiometric Configuration

Group With Medication. A flat curve was observed in 29 (45%) patients with a mean recovery rate of 43.7%; an upward curve in 9 (14%) patients with a mean recovery rate of 47.5%; a downward curve in 24 (37%) patients with a mean recovery rate of 43.2%; and a medium frequency curve in 3 (4%) patients with a mean recovery rate of 40.4%. These results were not statistically significant (P=.050).

Group With Medication Plus Sound Therapy. A flat curve was observed in 34 (51%) patients with a mean recovery rate of 70.1%; an upward curve in 10 (14%) patients with a mean recovery rate of 87.6%; a downward curve in 18 (27%) patients with a mean recovery rate of 73.2%; and a medium frequency curve in 5 (7%) patients with a mean recovery rate of 68.7%. These results were not statistically significant (P=.050).

Recovery Rate and Level of Hypoacusis in Sudden Deafness

Group With Medication. Slight hearing loss was present in 34 (52%) patients with a mean recovery rate of 43.7%; moderate hearing loss in 15 (23%) patients with a mean recovery rate of 42.2%; severe hearing loss in 13 (20%) patients with a mean recovery rate of 36.6%; and profound hearing loss in 3 (5%) patients with a mean recovery rate of 39.1%. These results were not statistically significant (P=.050).

Group With Medication Plus Sound Therapy. Slight hearing loss was present in 31 (46%) patients with a mean recovery rate of 70.1%; moderate hearing loss in 17 (25%) patients with a mean recovery rate of 72.5%; severe hearing loss in 13 (20%) patients with a mean recovery rate of 77.6%; and profound hearing loss in 6 (9%) patients with a mean recovery rate of 69.1%. These results were not statistically significant (P=.050).

Recovery Rate and Start of Therapy

Group With Medication. The mean start of therapy took place after 7.1 days. Patients treated within the first 15 days (n=58; 89%) presented a mean recovery rate of 43.2% and those treated after 15 days (n=7; 11%) presented a mean recovery rate of 30.2%. There was no statistical significance (P=.161).

Group With Medication Plus Sound Therapy. The mean start of therapy took place after 7.2 days. Patients treated within the first 15 days (n=59; 91%) presented a mean recovery rate of 76.5% and those treated after 15 days (n=6; 9%) presented a mean recovery rate of 30.8% (P<.001).

Comparison of the Recovery Rate Among Patients With Sudden Deafness Treated With Medication and Patients Treated With Medication Plus Sound Therapy

The recovery rate in the group with medication and the group with medication plus sound therapy is shown in Fig. 1. Only 54.6% of the recovery rate values in the medication group were greater than 50%, compared with 79.4% in the group treated with medication plus sound therapy (P<.020). In patients treated with medication plus sound therapy, the best recovery took place during the first month of treatment (Fig. 2).
Pure Tone Audiometry and Sudden Deafness

Table 1 shows the mean airway hearing thresholds, before and after treatment, of patients treated with medication and those treated with medication plus sound therapy.

Speech Audiometry

Patients treated with medication plus sound therapy presented a mean recovery rate of 87%, compared with 58% by patients treated only with medication (P<.003).

Patients With Normal Hearing and Hypoacusis, Before the Onset of Sudden Deafness

The results reported in Table 2 and Fig. 3 consider hearing before sudden deafness. The most notable observation is a better recovery in patients treated with medication plus sound therapy. The recovery was of 33.4 dB in patients treated with medication plus sound therapy and prior normal hearing, compared to a recovery of 20 dB in patients treated only with medication and normal hearing. Similarly, the recovery was of 22.4 dB in patients treated with medication plus sound therapy and prior hearing loss, compared to a recovery of 11.7 dB in patients treated only with medication and prior hearing loss.

Discussion

The action of appropriate sounds in deafness can promote recovery, especially if hearing loss has occurred acutely. All these mechanisms express neural plasticity and may explain the recovery of sensorineural hearing losses such as sudden deafness recovered with sound therapy. The reason for this improvement could be because direct stimulation exerted by sounds on ciliated cells would act as a rehabilitating mechanism, transforming mechanical energy into electrical impulses which would reach the auditory cortex through the auditory nerve.

In this study, 8 out of 10 patients regained hearing during the first month of treatment with medication and sound therapy, with this improvement persisting for at least 1 year after the end of the therapy. A favourable prognosis for sudden deafness was associated with the following factors: treatment during the first 15 days, patient age (<25 years), absence of vertigo, remission of tinnitus after treatment and the application of sound therapy. There was no statistical significance in relation to gender, prior hearing (normal hearing or hypoacusis), type of audiometric configuration or degree of hearing loss.

One aspect to consider may be the use of sounds as the only treatment for sudden deafness, but this raises ethical issues regarding the use of medication. During the study, 3 patients were not included because they did not follow the protocol. Specifically, they did not take the prescribed medication and only underwent sound therapy. The first case was a 28-year-old woman who refused the medication due to its side effects. She was only treated with sound therapy 60 days after the onset of sudden deafness. We obtained a recovery rate of 43%. The second case was a 63-year-old man...
who did not take corticosteroids because he suffered duodenal ulcer. Sound therapy was initiated within 30 days of onset of sudden deafness. We obtained a recovery rate of 11%. The third case was an 80-year-old woman who suffered from heart disease, hypertension, arrhythmia and cervicarthrosis. She was only treated with sound therapy 2 days after the onset of sudden deafness. We obtained a recovery rate of 50%. It is important to start sound therapy from the time of onset of sudden deafness in order to obtain better recovery of hearing.

There is a theoretical-computational\textsuperscript{12} model for the application of sounds in hearing loss. This model is based on neuronal hyperactivity and homeostatic plasticity after hearing loss. In this model, sounds are applied to cover the entire hearing loss, as would a hearing aid. Since sounds above 80 dB could cause acoustic trauma, sounds are applied with a limit between 60 and 70 dB, and only to the area affected by hearing loss. The difference between this protocol and the type of sound therapy applied in this study was that the sounds applied stimulated all frequencies.

Table 1 Pure Tone Audiometry in Patients With Sudden Deafness, Before and After Treatment.

<table>
<thead>
<tr>
<th>Mean air threshold</th>
<th>Control</th>
<th></th>
<th>Sounds</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before treatment</td>
<td>After treatment</td>
<td>Before treatment</td>
<td>After treatment</td>
</tr>
<tr>
<td>&lt;25 dB</td>
<td>0 (0%)</td>
<td>9 (14%)</td>
<td>0 (0%)</td>
<td>19 (28%)</td>
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<tr>
<td>26–34 dB</td>
<td>5 (8%)</td>
<td>5 (8%)</td>
<td>4 (6%)</td>
<td>12 (18%)</td>
</tr>
<tr>
<td>35–54 dB</td>
<td>10 (15%)</td>
<td>11 (17%)</td>
<td>11 (17%)</td>
<td>12 (18%)</td>
</tr>
<tr>
<td>55–74 dB</td>
<td>17 (26%)</td>
<td>18 (28%)</td>
<td>19 (28%)</td>
<td>25 (37%)</td>
</tr>
<tr>
<td>&gt;74 dB</td>
<td>33 (51%)</td>
<td>22 (33%)</td>
<td>33 (49%)</td>
<td>5 (8%)</td>
</tr>
</tbody>
</table>

Control: patients treated with medication.
Sounds: patients treated with medication plus sound therapy.
Chi-square: comparing control before and after treatment, $P<.030$; sounds before and after treatment, $P<.001$; control after treatment and sounds after treatment, $P<.020$.

Figure 3 Pure tone audiometry of patients with sudden deafness. (A) Patients with normal hearing before sudden deafness who were treated with medication. (B) Patients with sensorineural hearing loss before sudden deafness who were treated with medication. (C) Patients with normal hearing before sudden deafness who were treated with medication plus sound therapy. (D) Patients with sensorineural hearing loss before sudden deafness who were treated with medication plus sound therapy. Results are expressed as mean±standard error (■-■: hearing before sudden deafness; ▼-▼: hearing after treatment of sudden deafness; □-□: hearing at the time of diagnosis of sudden deafness).
### Table 2  Tone Audiometry of Patients With Sudden Deafness, Before and After Treatment.

<table>
<thead>
<tr>
<th>Frequency, Hz</th>
<th>Normal (n=43)</th>
<th>Hearing Loss (n=22)</th>
<th>Prior Hearing</th>
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<tr>
<td></td>
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<td>38.8±3.9</td>
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</tr>
<tr>
<td>Day of Diagnosis</td>
<td>30 Days After Treatment</td>
<td>Improvement in Decibels</td>
<td>Percentage Recovery</td>
</tr>
<tr>
<td>Patients treated with medication n=65</td>
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<td></td>
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<tr>
<td>125</td>
<td>73.6±8.5</td>
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<td>250</td>
<td>74.4±9.2</td>
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<td>500</td>
<td>76.2±9.4</td>
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<td>8000</td>
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<tr>
<td>Mean</td>
<td>74.7±0.5</td>
<td>54.8±0.6</td>
<td>20.0±0.6&lt;sup&gt;a&lt;/sup&gt;</td>
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<table>
<thead>
<tr>
<th>Frequency, Hz</th>
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<th>Hearing Loss (n=25)</th>
<th>Percentage Recovery</th>
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<tr>
<td></td>
<td>22.5±0.3</td>
<td>38.7±3.8</td>
<td></td>
</tr>
<tr>
<td>Day of Diagnosis</td>
<td>30 Days After Treatment</td>
<td>Improvement in Decibels</td>
<td>Percentage Recovery</td>
</tr>
<tr>
<td>Patients treated with medication plus sound therapy n=67</td>
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<tr>
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<td>74.1±9.3</td>
<td>41.1±3.4</td>
<td>32.9</td>
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<td>8000</td>
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<td>42.8±3.8</td>
<td>32.5</td>
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<tr>
<td>Mean</td>
<td>75.8±0.5</td>
<td>42.4±0.2</td>
<td>33.4±1.0&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are expressed in decibels, mean±standard error, and hearing recoveries as indicated.

Student’s t test: comparing<sup>a</sup> with<sup>e</sup>,<sup>b</sup> with<sup>f</sup>,<sup>c</sup> with<sup>g</sup>, and<sup>d</sup> with<sup>h</sup>, all presenting statistical significance of<sup>P<.001</sup>.
This alternative methodology could be considered in future studies.

Based on the results of these studies in animals,8–11 sound stimulation inhibits degenerative processes, influences the partial repair of damaged ciliated cells, modulates the hearing recovery process or prevents the reorganisation of the auditory cortical tonotopic map. The neurophysiological influence of musical training on speech perception has also been studied, even in noisy environments and in cases of deafness.13

In the present study we conducted 3 different types of evaluation: recovery rate, pure tone audiometry and speech audiometry. The limitations of the different determinations have been discussed in the literature.14

In conclusion, this new, scientifically based methodology can be applied to sudden deafness, with acoustic stimulation being administered for 1 month. Hearing recovery is much higher in cases of sudden deafness stimulated with sound therapy when treatment is started immediately.

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Conflict of Interests

The authors have no conflicts of interest to declare.

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