Consensus on diagnosis and treatment of sudden hearing loss

Guillermo Plaza, a,∗ Enrique Durio, b Carlos Herráiz, c† Teresa Rivera, d José Ramón García-Berrocal e

aServicio de Otorrinolaringología, Hospital Universitario de Fuenlabrada, Fuenlabrada, Madrid, Spain
bServicio de Otorrinolaringología, Hospital Universitario de Getafe, Getafe, Madrid, Spain
cServicio de Otorrinolaringología, Hospital Universitario de Alcorcón, Alcorcón, Madrid, Spain
dServicio de Otorrinolaringología, Hospital Universitario Príncipe de Asturias, Alcalá de Henares, Madrid, Spain
eServicio de Otorrinolaringología, Hospital Universitario Puerta de Hierro-Majadahonda, Majadahonda, Madrid, Spain

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Corticoids;
Adverse effects

Abstract  Idiopathic sudden sensorineural hearing loss is an unexplained unilateral hearing loss with onset over a period of less than 72 hours, without other known otological diseases.

We present a consensus on the diagnosis, treatment and follow-up of this disease, designed by AMORL, after a systematic review of the literature from 1966 to June 2010.

Diagnosis of sudden sensorineural hearing loss is based on mandatory otoscopy, acoumetry, tonal audiometry, speech audiometry, and tympanometry. After clinical diagnosis is settled, and before treatment is started, a full analysis should be done and an MRI should be requested later.

Treatment is based on systemic corticosteroids (orally in most cases), helped by intratympanic doses as rescue after treatment failures.

Follow-up should be done at day 7, with tonal and speech audiometries, and regularly at 15, 30, and 90 days after start of therapy, and after 12 months.

By consensus, results after treatment should be reported as absolute dBs recovered in pure tone audiometry, as improvement in the recovery rate in unilateral cases, and as improvement in speech audiometry.

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∗Work developed under the direction of the Asociacion Madrileña de Otorrinolaringologia (AMORL) (Otolaryngology Association of Madrid).
∗Corresponding author.
E-mail address: gplaza.hfr@salud.madrid.org (G. Plaza).
†Unfortunately deceased in March 2010.

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Introduction

During the Third Congress of the Madrid ENT Association (AMORL), held in 2008, the Round Table on Sudden Deafness saw the need to create a consensus document on its diagnosis and treatment among hospitals in Madrid, as this is a controversial and relatively common condition. Moreover, various reviews on sudden deafness (SD) have recently been published in journals of general interest, so it seems appropriate to review the subject, to propose unified criteria for this disease.

Regarding methodology, this consensus group, backed by AMORL, has conducted a systematic review of the literature published from 1966 to June 2010 on the terms MESH “(acute or sudden) hearing loss”, collecting 4,180 articles in Spanish, English, German and French. It is striking that among so many publications, there are no clinical practice guidelines published on the subject, and very few randomized trials and meta-analysis. Through various meetings, the group evaluated the literature independently, highlighting the studies with the highest scientific quality, especially with regard to treatment.

Our study work attempted to collect and combine the experiences of various hospitals, and to systematically review the available scientific evidence to reach a common consensus about this pathology, in to the benefit of adult patients with sudden hearing loss.

Definition

By consensus, SD is considered as sensorineural or perceptual hearing loss cases with a sudden onset, within 72 hours, with a loss of over 30 dB, in at least 3 consecutive frequencies of tonal audiometry, without other prior otological history. If a previous audiometry is available, this should be used as a baseline reference; if no previous audiometry is available, in unilateral cases the healthy contralateral ear should be used as a reference.

However, some studies also consider as “probable SD” cases of sensorineural or perceptual hearing loss that affect only 2 or 3 frequencies, with losses of 10-20 dB, appearing in less than 12 hours, including deafness appreciated upon waking in the morning, which recover rapidly.

Epidemiology

Since its first description in 1944, the incidence of SD has been increasing steadily over the years, and currently represents 1.2% of the cases at ENT Emergency Units. Looking at the literature on its epidemiology, its impact can be established in 5-20 cases per 100,000 inhabitants per year. Recently, other authors have reported an even higher incidence, of up to 160 cases per 100,000 inhabitants per year, especially in cases where there are national registers of SD, as in Japan.

Presumably, these differences are due to different inclusion criteria defined for sudden deafness and to the underdiagnosis of this entity at the primary healthcare level.

Aetiopathogenesis and physiopathology

Three possible causes have been postulated for idiopathic SD:
Viral theory

This is the most documented theory, both anatomically-pathologically, with post-mortem findings related to infection by type 1 herpes simplex virus, and by the frequent history of upper respiratory tract catarrh or its higher incidence at certain times of the year. However, no specific serological profiles or response to common antiviral treatment such as acyclovir have been reported.

Theory of alteration in inner ear microcirculation

Some studies have found a certain genetic prothrombotic susceptibility, while others have shown a higher incidence of SD in patients with cardiovascular risk, especially with mitral prolapse or antiphospholipid syndrome. On the other hand, SD has been reported after transient ischemic events in the inner ear, such as during general anesthesia, or confirmed after an episode of intralabyrinthine haemorrhage, objectified by MRI imaging. It has also been reported that the frequency of stroke is greater in the 5 years after having suffered SD. These alterations in the microcirculation of the inner ear have been the basis of treatments such as administration of low molecular weight heparins, plasmapheresis for the cleaning of plasma LDL, the use of inhaled carbogen or hyperbaric oxygen chambers and the administration of prostaglandins such as PGE1, with mixed results.

Theory of immune-mediated disease

This theory is supported by pathological studies, spontaneous recoveries and the response to treatment with steroids. However, in some patients with SD, there is no evidence of impaired immunity and the clinical evolution is not always compatible with an autoimmune case. Moreover, it is common for SD cases initially classified as idiopathic to be diagnosed as a specific autoimmune disorder with the passage of time.

Conversely, although it would not be an idiopathic SD, the "theory of cochlear membrane rupture" has been described, due to a possible perilymph fistula, appearing in connection with physical exercise, barotrauma or a Valsalva manoeuvre. This theory could justify the spontaneous recovery of some patients, and for some authors it determines the indication for early exploratory tympanotomy to seal the rupture.

Diagnosis

Faced with a clinical suspicion of SD, and before considering a possible treatment, the diagnostic tests required are:

**Otoscop y** should be normal in both ears; however, the finding of a wax plug does not exclude a possible SD. The plug should be removed and test whether hearing becomes normal.

**Acoumetry** (tuning forks) will give us a sensorineural pattern: positive Rinne in the diseased ear (Figures 1A and 1B) and Weber lateralised to the healthy ear (Figure 1C), allowing us to rule out causes of SD due to middle ear diseases: otitis media with effusion, etc. with a transmission pattern (negative Rinne in diseased ear and Weber towards the diseased ear). However, in cases of severe SD, cophosis, there can be a false negative Rinne (the patient does not hear the tuning fork at all).

In outpatient ENT consultations, in addition to confirming the normal otoscopy and sensorineural acoumetry, it is necessary to carry out a tonal and verbal audiometry and a tympanogram

Tonal audiometry will determine the pure tone audiometry (PTA), taking the average dB threshold at frequencies 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA, which must be greater than 30 dB in bone conduction to confirm the diagnosis of SD.

Verbal audiometry will assess verbal comprehension, determining the verbal reception threshold (VRT) and the maximum discrimination (DMax).

With these tests, we may conduct an initial diagnosis, and thus begin treatment; in addition, they will serve as baseline data to evaluate the response.

The audiological examination may be selectively extended to include supraliminal tonal audiometry, otoacoustic emissions or auditory potentials (BAEP, ASSR) or to include vestibular, caloric, and VEMP tests.

It is advisable to supplement the diagnostic process with:

- Stapedial reflex, including the Metz test, to rule out cochlear recruitment and Ménière’s disease.

- Series of analyses, whose extraction should be performed prior to treatment, including at least the following parameters: blood count, erythrocyte sedimentation rate (ESR), luetic serology (VDRL and FTAab) and antinuclear antibodies (ANA), according to prior systematic reviews. It may also be useful to request immune phenotype by CD4+ and CD8+ lymphocyte subpopulations, and the CD45RO+ isoforms.

- MR of the inner ear with gadolinium to exclude retrocochlear pathology or show intracochlear haemorrhage.

For a more extensive review on the diagnosis process for SD, we recommend reviewing the work of Chau et al and Nosrati-Zarenoe et al.

Differential diagnosis

Sudden deafness can be a symptom of many diseases, both as their debut form and during the course of their evolution. The most difficult part is determining the causal relationship in each individual patient.
In Table 1, we present the relationship of pathology and anatomical location, with the aim of reducing the cases which are labelled as idiopathic SD.

It is compulsory to make a correct differential diagnosis between SD and other entities with a similar presentation, such as catarrhal ototubaritis, barotrauma, etc. This requires an acoumetry and otoscopy.

**Clinical forms**

Among the presentation forms, we can find the following audiometric curves of sensorineural hearing loss (SNHL), with different prognostic implications (Figure 2), since those affecting lower tones usually show a better response to treatment.

Moreover, there are three atypical forms of SD.

**Paediatric forms**

In the event of traumatic injury, however slight, we must investigate a dilated vestibular aqueduct syndrome, through an ear CT scan.

**Bilateral forms**

In these cases, it is common to find elevated ANA in the case of autoimmune forms, such as sarcoidosis, multiple sclerosis, Crohn’s disease, Cogan syndrome, immune-mediated inner
ear disease, etc. Other possible causes of bilateral SD are infectious (infectious mononucleosis, syphilis, meningitis, HIV infection, etc.), neoplastic (malignant lymphomatosis, carcinomatous meningitis, leukaemia, etc.) or vascular (intracranial aneurysm, hydrocephalus, stroke, periarteritis nodosa, history of spinal anaesthesia and so on).

**Figure 2** Forms of presentation of sudden deafness. A: Sudden deafness with low frequency SNHL (better prognosis). B: Sudden deafness with pantonal SNHL. C: Sudden deafness with SNHL at high frequencies. D: Sudden deafness with SNHL at medium frequencies. E: Sudden deafness with residual hearing (cophosis).

**Evolving forms**

These are cases of SD as a presentation symptom of fluctuating sensorineural hearing loss or Ménière’s disease, especially when it affects low frequencies. 83, 91, 92
The natural history of sudden deafness (SD) is variable, since its causes are multiple. Some patients recover completely without medical intervention, usually during the first three days (spontaneous recovery) and generally do not see a doctor. Others improve slowly over a period of 1 to 2 weeks, with reports of improvement or spontaneous recovery in up to 65% of cases in the most classical series of the 70s and 80s. However, most patients do not recover their hearing without treatment, and up to 10% of patients experience a worsening of their hearing over time, despite the introduction of adequate treatment. 

Table 2 describes the signs of good and bad prognosis. 

The most commonly accepted factors of poor prognosis in SD in the literature are the following:

- Advanced age of the patient.
- Cardiovascular risk factors (arterial hypertension, etc.).
- Exposure to noise trauma.
- Intensity of the initial loss: the greater the degree of loss, the worse the prognosis for recovery of hearing function.
- Type of audiometric curve: pantonal or high frequency deafness cases have a lower percentage of recovery.
- Healthy ear auditory function: when the contralateral ear suffers a different pathology, the SD of the affected ear has a poorer prognosis for recovery.
- Associated symptoms: the presence of vestibular symptoms represents a greater involvement of the entire labyrinth, and has a worse prognosis.
- Promptly initiated treatment: the sooner the process is treated, the greater the chances of recovery.
- Speed of onset of clinical improvement: the earlier the onset of the improvement of clinical symptoms, the better the functional outcome of the SD.

Table 2  Prognostic factors in sudden deafness

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Audiometry</th>
<th>Symptoms</th>
<th>Start of treatment</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worse</td>
<td>SNH in high and medium frequencies</td>
<td>Higher auditory affectation (and cophosis)</td>
<td>Vertigo, Tinnitus, Headache</td>
<td>Late, Elderly</td>
</tr>
<tr>
<td>Better</td>
<td>SNH in low and pantonal frequencies</td>
<td>Lower auditory affectation</td>
<td>Absent, Absent, Absent</td>
<td>Early, Young</td>
</tr>
</tbody>
</table>

However, when it affects mostly high tones, SD can be the first symptom of an acoustic neurinoma, even when hearing is restored completely.

Prognosis

Traditional general measures such as bed rest or diets with a restricted salt intake have not demonstrated effectiveness, so hospital admission to maintain bed rest is debatable. For this reason, there is no agreement on the need for a first phase of hospital treatment for 4 to 7 days, followed by outpatient treatment.

Despite the excess of existing literature on SD, there are only a few randomized double-blind controlled trials, which have been reviewed by Cochrane and summarised in a meta-analysis. The most outstanding include the classic work of Wilson et al, who compared oral steroids versus placebo in 1980, laying the foundation for their use (OR 3.22; 1.18-8.76). However, the group of Cinamon et al found no significant effect with steroids (0.89; 0.1-7.86).

With regard to antiviral therapy, although antiviral agents should theoretically have a positive effect on SD, randomized clinical trials conducted by Stokross et al in 1998, Tucci et al in 2002, and Westerlaken et al in 2003 were unable to find statistically-significant differences between antiviral agents and placebo.

Other widely used treatments, on the basis of vascular aetiology, such as vasodilators, carbogen or hyperbaric oxygen have been reviewed recently in a meta-analysis and by Cochrane. However, there were no findings of significant effectiveness in SD.

Consequently, having reviewed the literature, once SD has been diagnosed, this consensus group advises the following therapeutic scheme (Figure 3), based mainly on systemic corticosteroids, supported by intratympanic corticosteroids as rescue, with a large volume of recent publications in this respect.

- If the diagnosis was possible within 30 days of onset of symptoms, the treatment should be with oral steroids for 1 month, with 3 main options:
  - Prednisone (Prednison Alonga®, Dacortin®), 1 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 80 mg x 5 days, 60 mg x 5 days, 40 mg x 5 days, 20 mg x 5 days, 10 mg x 5 days, 5 mg x 5 days).
  - Methylprednisolone (Urbason®), 1 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 80 mg x 5 days, 60 mg x 5 days, 40 mg x 5 days, 20 mg x 5 days, 10 mg x 5 days, 5 mg x 5 days).
  - Deflazacort (Dezacor®, Zamene®), a similar decreasing pattern, 1.5 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 120 mg x...
Susception of sudden deafness (SD)

- Tonal audiometry: SD confirmed if sensorineural hypoacusis >30 dB
  - Complete audiological study
  - Series of analyses: at least haemogram, ESR, luetic serology and ANA
  - MRI of inner ear

Empirical treatment
- If early diagnosis (<30 days): oral corticosteroids, 1 month in decreasing pattern
- If severe SD (>70 dB), in single ear or with intense associated vertigo.
- IV corticosteroids at high doses for 7 days
- If there is no complete response to systemic corticosteroid treatment, after 7 days offer intratympanic rescue corticosteroids in weekly pattern, for 3 weeks, simultaneously with systemic pattern

Follow-up
- Weekly control with tonal and verbal audiometry
- Review at 15, 30, 90 days and at 12 months.
- Evaluation of response to treatment
  - Complete response: maximum difference of 10 dB with bone PTA of healthy contralateral ear
  - Partial recovery: improvement of less than 50% of initial loss
  - Average auditory recovery of the auditory threshold (Siegel scale)
  - Recovery rate: (initial PTA – final PTA) x100/ (initial PTA – initial contralateral PTA)

- If the patient has fully recovered hearing and presents acusia, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month).
- If the patient has regained partial hearing, with an improvement of less than 15 dB, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month), and simultaneous intratympanic treatment may be recommended individually.
- If hearing has deteriorated, intravenous rescue treatment will be recommended.

During systemic steroid treatment, either orally or intravenously, there should be gastro-duodenal prophylaxis with proton pump inhibitors (such as omeprazole) at doses of 40 mg/day, for 1 month.

In patients over 65 years, if glucocorticoid treatment is prolonged beyond 15 days, it will be necessary to associate vitamin D (800 IU/day) and calcium (800-1,000 mg/day) as a preventive regimen of bone loss and osteoporosis (requesting a bone density test is optional).

Moreover, given the relative frequency of adverse effects associated to standard treatments for SD, this consensus group suggests the use of specific informed consent, especially when it uses intravenous corticosteroids at high doses (validated informed consent, which is available from the authors).

In cases of suspected vascular aetiology (known cardiovascular risk factors, etc.), it may be associated with vasodilators, such as nimodipine, intravenously (5-15 cc in 500 ml of saline solution, passing slowly, every 8 hours), or such as oral trimetazidine (especially if the patient is already in treatment for hypertension, 1 comp/8 h for 1 month), and then refer the patient to the internal medicine department for evaluation and possible antiplatelet therapy.

Figure 3 Diagnostic and therapeutic algorithm for sudden deafness.

5 days, 90 mg x 5 days, 60 mg x 5 days, 30 mg x 5 days, 15 mg x 5 days).
- If SD is severe (> 70 dB), in a single ear or with severe associated vertigo (suspected vestibular neuritis), treatment should be offered with intravenous corticosteroids for 7 days, in a day hospital regime or through hospital admission, with a dose of 500 mg methylprednisolone per day, slowly passing to one dose in serum in 30 minutes. Subsequently, the oral regime described above should be reintroduced.
- If there is contraindication for the use of systemic corticosteroids, or if there is no response to treatment with systemic corticosteroids, oral or intravenous, after 7 days of its establishment, a rescue treatment with intratympanic corticosteroids should be offered, with 1 dose weekly for 3 weeks in outpatient ENT consultation (and maintaining the decreasing oral pattern), with two commonly used guidelines (after topical anaesthesia with phenol on the tympanic membrane, with a no. 22 Abocath needle):
  - Methylprednisolone, 0.9 cc of a 40 mg vial, mixed with 1% lidocaine at 0.1 ml.
  - Dexamethasone, 0.9 cc of an 8 mg vial.

Please note the equivalence between the two steroids in terms of anti-inflammatory effect: 4 mg methylprednisolone equals 6 mg deflazacort.

Follow-up

Once the treatment has been established, there should be a control after the first week, including tonal and verbal audiometry, to assess tolerance to treatment and its results:

- If the patient has fully recovered hearing and presents acusia, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month).
- If the patient has regained partial hearing, with an improvement of less than 15 dB, the prescribed scheme should be followed (oral corticosteroids in decreasing doses for one month), and simultaneous intratympanic treatment may be recommended individually.
- If hearing has deteriorated, intravenous rescue treatment will be recommended.
Control should be performed, including tonal and verbal audiometry, at 15, 30 and 90 days of diagnosis. However, to rule out autoimmune hearing loss or Ménière's disease, it would be advisable to monitor patients until 12 months after diagnosis, especially in cases of SD affecting low frequencies.

Evaluation of results

After treatment, recovery would be complete if a maximum difference of 10 dB is reached with the bone PTA of the healthy contralateral ear. Partial response is considered when there is an improvement of less than 50% of the initial loss.

For most authors, successful treatment of SD is defined arbitrarily, as an average auditory recovery of the auditory threshold (taking the average of the threshold at 0.25, 0.5, 1, 2, 4, and 8 kHz as PTA) of more than 30 dB, or following gradual schemes as proposed in the U.S. by Siegel in the 70s (Table 3), by the Japanese Committee on Sudden Deafness in the 80s (Table 4), or by the Swedish National Research Committee of the Ministry of Health and Welfare, Japan.

Other authors use more stringent criteria such as the recovery rate, described in publications in the 70s. This rate is established taking into account the degree of recovery of the diseased ear with respect to the healthy ear, so it only applies in cases with a normal contralateral hearing (<25 dB), according to the following ratio:

\[
\text{Rate of recovery} = \frac{\text{Initial PTA – Final PTA}}{\text{Initial PTA – Contralateral Initial PTA}} \times 100
\]

Moreover, it is essential to confirm the improvement in verbal comprehension through serial verbal audiometry, determining the verbal reception threshold (VRT) and the maximum discrimination (DMax or SDS).

As a consensus, the result of treatments applied should be presented in both forms, with respect to the dB recovered in PTA (Siegel scale), and following the recovery rate in unilateral cases, as well as with speech audiometry parameters such as VRT and DMax, as postulated in the most recent studies.

Conclusions

Sudden deafness (SD) is a perceptual or sensorineural hearing loss with sudden onset, within 72 hours, with a loss of over 30 dB, in at least 3 consecutive frequencies in a tone audiometry, with no other previous otological history. It is an entity of increasing prevalence, affecting 5-20 cases per 100,000 inhabitants per year, and its diagnosis requires a greater involvement of the primary healthcare network.

Faced with a clinical suspicion of SD, and before considering possible treatment, diagnostic tests required in the field of primary healthcare and emergency ENT would be: normal otoscopy and acoumetry, showing a sensorineural pattern (positive Rinne in diseased ear and Weber lateralisated to the healthy ear). In ENT outpatient consultations: it would also be necessary to obtain tonal audiometry, determine hearing threshold, administer verbal audiometry, find the verbal reception threshold and the maximum discrimination and perform a tympanogram.

Once the clinical diagnosis of SD has been carried out, before starting treatment; a series of analyses should be requested; their extraction should be performed prior to treatment and should include at least the following parameters: blood count, erythrocyte sedimentation rate, luetic serology and antinuclear antibodies. The study should be completed subsequently with an MRI scan of the inner ear.

Treatment of SD is very controversial; however, as consensus, once SD has been diagnosed, we can recommend treatment based primarily on systemic corticosteroids, usually taken orally, supported by intratympanic corticosteroids as rescue. Moreover, given the relative frequency of adverse effects associated with standard treatments of SD, this consensus group suggests the use of a specific informed consent.

With respect to follow-up, once the treatment has been established, it should be checked after one week, including tonal and verbal audiometry, to assess tolerance to treatment and its results, and at 15, 30 and 90 days of diagnosis. It would be advisable to monitor patients until 12 months after diagnosis.

As a consensus, the result of treatments applied to patients with SD should be presented both in terms of the number of dB recovered in the tonal auditory threshold and

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Criteria for hearing improvement in SD, based on the average hearing threshold</th>
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<tbody>
<tr>
<td>Response</td>
<td>Average auditory recovery</td>
</tr>
<tr>
<td>Full recovery</td>
<td>Until obtaining a final PTA &lt;25 dB, or independently of dB gained</td>
</tr>
<tr>
<td>Partial improvement</td>
<td>Improvement &gt;15 dB, but final PTA between 25-45 dB</td>
</tr>
<tr>
<td>Slight improvement</td>
<td>Improvement &gt;15 dB, but final PTA &gt;45 dB</td>
</tr>
<tr>
<td>No improvement</td>
<td>Improvement &lt;15 dB (Taking the threshold average at 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA) according to Siegel.</td>
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</tbody>
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<tr>
<th>Table 4</th>
<th>Criteria for hearing improvement in SD, based on the average hearing threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response</td>
<td>Average auditory recovery</td>
</tr>
<tr>
<td>Full recovery</td>
<td>Until obtaining a PTA &lt;20 dB, or until the threshold of the contralateral healthy ear</td>
</tr>
<tr>
<td>Good improvement</td>
<td>Improvement &gt;30 dB</td>
</tr>
<tr>
<td>Slight improvement</td>
<td>Improvement 10-30 dB</td>
</tr>
<tr>
<td>No improvement</td>
<td>Improvement &lt;10 dB</td>
</tr>
</tbody>
</table>

(Taking the threshold average at 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA). Criteria established in 1981 by the Sudden Deafness Research Committee of the Ministry of Health and Welfare, Japan.
following the recovery rate in unilateral cases, as well as with verbal audiometry parameters.

### Sudden deafness registry

Following the initiatives of other countries such as Sweden\(^7\) or Japan,\(^1,3\) it would be advisable to create a register of SD at our hospitals, acting as a unit within the Community of Madrid or in those communities deeming it appropriate.

To do this, there is a minimum set of data to be collected for each patient (Table 5, Table 6) and sent to the community register, by e-mail to SEORL (e-mail can be sent to the authors).

### Conflict of interest

The authors declare no conflict of interest.

### References


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