

REVIEW

Consensus on diagnosis and treatment of sudden hearing loss[☆]

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KEYWORDS

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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 tonal audiometry, as improvement in the recovery rate in unilateral cases, and as improvement in speech audiometry.

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PALABRAS CLAVE

Sordera súbita;
Acumetría;
Audiometría;
Corticoides;
Efectos secundarios

Consenso sobre el diagnóstico y tratamiento de la sordera súbita

Resumen La sordera súbita idiopática es aquella hipoacusia neurosensorial de inicio súbito, en menos de 72 horas, sin otros antecedentes otológicos previos.

Presentamos un consenso sobre el diagnóstico, tratamiento y seguimiento de la sordera súbita, surgido desde la Asociación Madrileña de ORL.

Mediante revisión sistemática de la literatura sobre sordera súbita, desde 1966 hasta junio de 2010, sobre los términos MESH “(acute or sudden) hearing loss”, llegando a las siguientes sugerencias: en cuanto al diagnóstico, ante una sospecha clínica de sordera súbita, las pruebas diagnósticas que se consideran necesarias son: otoscopia, acumetría, audiometría tonal, audiometría verbal y timpanograma.

Una vez hecho el diagnóstico clínico de sordera súbita, antes de comenzar el tratamiento, se solicitará una batería analítica, debiendo completarse más tarde el estudio con RM de oído interno.

Se recomienda que el tratamiento de la sordera súbita esté basado fundamentalmente en los corticoides sistémicos, generalmente por vía oral, apoyados en los corticoides intratimpánicos como rescate.

Respecto al seguimiento, se realizará un control a la semana del inicio del mismo, incluyendo audiometría tonal y verbal, y a los 15, 30 y 90 días del diagnóstico, y 12 meses después.

Como consenso, el resultado de los tratamientos aplicados debería presentarse, tanto en cuanto a la cantidad de dB recuperados en el umbral auditivo tonal como siguiendo la tasa de recuperación en los casos unilaterales, así como con parámetros de audiometría verbal.

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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,^{1,2} 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.¹⁻⁵ 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.^{6,7}

Epidemiology

Since its first description in 1944,⁸ the incidence of SD has been increasing steadily over the years, and currently represents 1.2% of the cases at ENT Emergency Units.^{1,3,9} Looking at the literature on its epidemiology, its impact can be established in 5-20 cases per 100,000 inhabitants per year.^{1,6,9,10} Recently, other authors have reported an even higher incidence, of up to 160 cases per 100,000 inhabitants per year,^{11,12} 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.^{1,5,7,12}

Aetiopathogenesis and physiopathology

Three possible causes have been postulated for idiopathic SD.^{1-7,10,13,14:}

Viral theory

This is the most documented theory, both anatomically-pathologically, with post-mortem findings related to infection by type 1 herpes simplex virus,¹⁵ and by the frequent history of upper respiratory tract catarrh or its higher incidence at certain times of the year.¹⁶ However, no specific serological profiles¹⁷ or response to common antiviral treatment such as acyclovir have been reported.^{15,18-24}

Theory of alteration in inner ear microcirculation

Some studies have found a certain genetic prothrombotic susceptibility,²⁵ while others have shown a higher incidence of SD in patients with cardiovascular risk,²⁶⁻³² especially with mitral prolapse^{33,34} or antiphospholipid syndrome.³⁵ On the other hand, SD has been reported after transient ischemic events in the inner ear, such as during general anesthesia,^{36,37} or confirmed after an episode of intralabyrinthine haemorrhage, objectified by MRI imaging.³⁸⁻⁴⁰ It has also been reported that the frequency of stroke is greater in the 5 years after having suffered SD.⁴¹ These alterations in the microcirculation of the inner ear have been the basis of treatments such as administration of low molecular weight heparins,⁴² plasmapheresis for the cleaning of plasma LDL,⁴³⁻⁴⁹ the use of inhaled carbogen⁵⁰⁻⁵² or hyperbaric oxygen chambers⁵³⁻⁶² and the administration of prostaglandins such as PGE1,^{52,63,64} with mixed results.

Theory of immune-mediated disease

This theory is supported by pathological studies, spontaneous recoveries and the response to treatment with steroids.^{1-7,14,65,66} 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.^{67,68} Moreover, it is common for SD cases initially classified as idiopathic to be diagnosed as a specific autoimmune disorder with the passage of time.⁶⁹

Conversely, although it would not be an idiopathic SD, the "theory of cochlear membrane rupture" has been described,^{70,71} 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 tympanostomy to seal the rupture.^{72,73}

Diagnosis

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

In primary healthcare and ENT emergency services, the two tests required are otoscopy and acoumetry:

Otoscopy 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.^{1,5,13}

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).^{1,5,74} 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).⁷⁵

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.⁷⁶

Series of analyses,^{66,68,77-79} whose extraction should be performed prior to treatment, including at least the following parameters: blood count, erythrocyte sedimentation rate (ESR), luetic serology (VDRL and FTAabs) and antinuclear antibodies (ANA), according to prior systematic reviews.^{66,77} It may also be useful to request immune phenotype by CD4+ and CD8+ lymphocyte subpopulations, and the CD45RO+ and CD45RA+ isoforms.^{66,80}

MRI of the inner ear with gadolinium to exclude retrocochlear pathology or show intracochlear haemorrhage.⁸¹⁻⁸⁶

For a more extensive review on the diagnosis process for SD, we recommend reviewing the work of Chau et al¹⁴ and Nosrati-Zarenoe et al.⁸³

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.

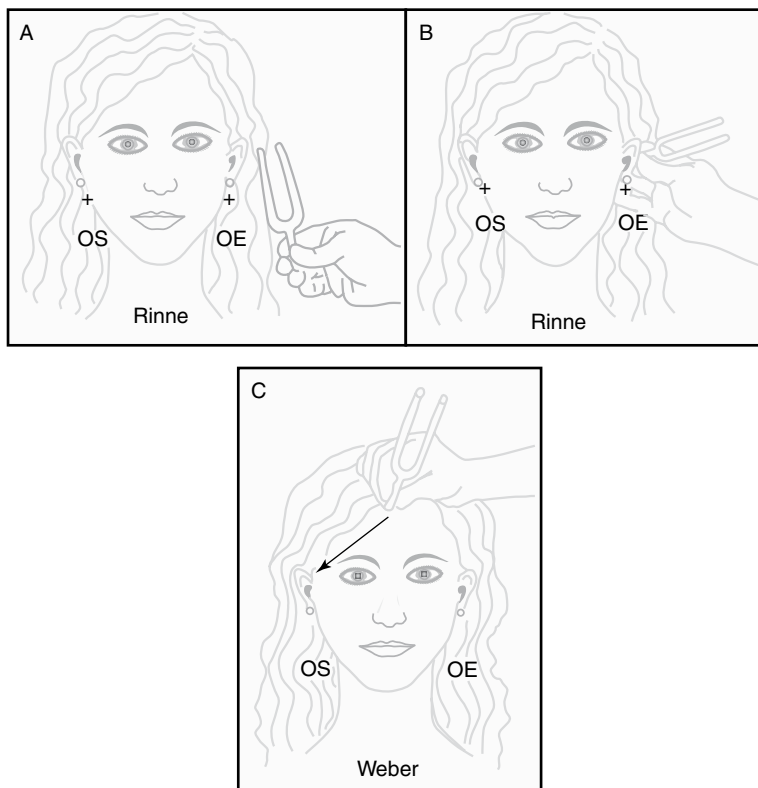


Figure 1 Acoumetry in sudden deafness: A: Rinne applying the tuning fork to the ear to explore the air pathway. B: Rinne applying the tuning fork on the mastoid bone to explore bone conduction. C: Weber, perceiving bone conduction to the healthy ear. OS: healthy ear, OE: diseased ear.

Table 1 Causes of sudden deafness

Cochlear

- Inflammatory: virus, bacteria, spirochetes (syphilis)
- Traumatic
- Vascular
- Haematological (anaemia, stroke, alterations of circulation)
- Immune-mediated disease (Cogan, scleroderma, ulcerative colitis, sarcoidosis), vasculitis
- Endolymphatic *hydrops*, including Ménière’s disease
- Metabolic diseases (diabetes)
- Bone diseases of the otic capsule (metastases, myeloma, histiocytosis X)
- Ototoxic agents

Retrocochlear

- Meningitis
- Multiple sclerosis
- Friederich ataxia
- Amyotrophic lateral sclerosis
- Vogt-Koyanagi-Harada syndrome
- Xeroderma pigmentosum
- Posterior fossa tumours (acoustic neurinoma)
- Central deafness

Idiopathic

In Table 1, we present the relationship of pathology and anatomical location,^{1,7} 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.^{1,5} 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 implications⁸⁷ (Figure 2), since those affecting lower tones usually show a better response to treatment.

Moreover, there are three atypical forms of SD.⁸⁸

Paediatric forms

In the event of traumatic injury, however slight, we must investigate a dilated vestibular aqueduct syndrome, through an ear CT scan.^{89,90}

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

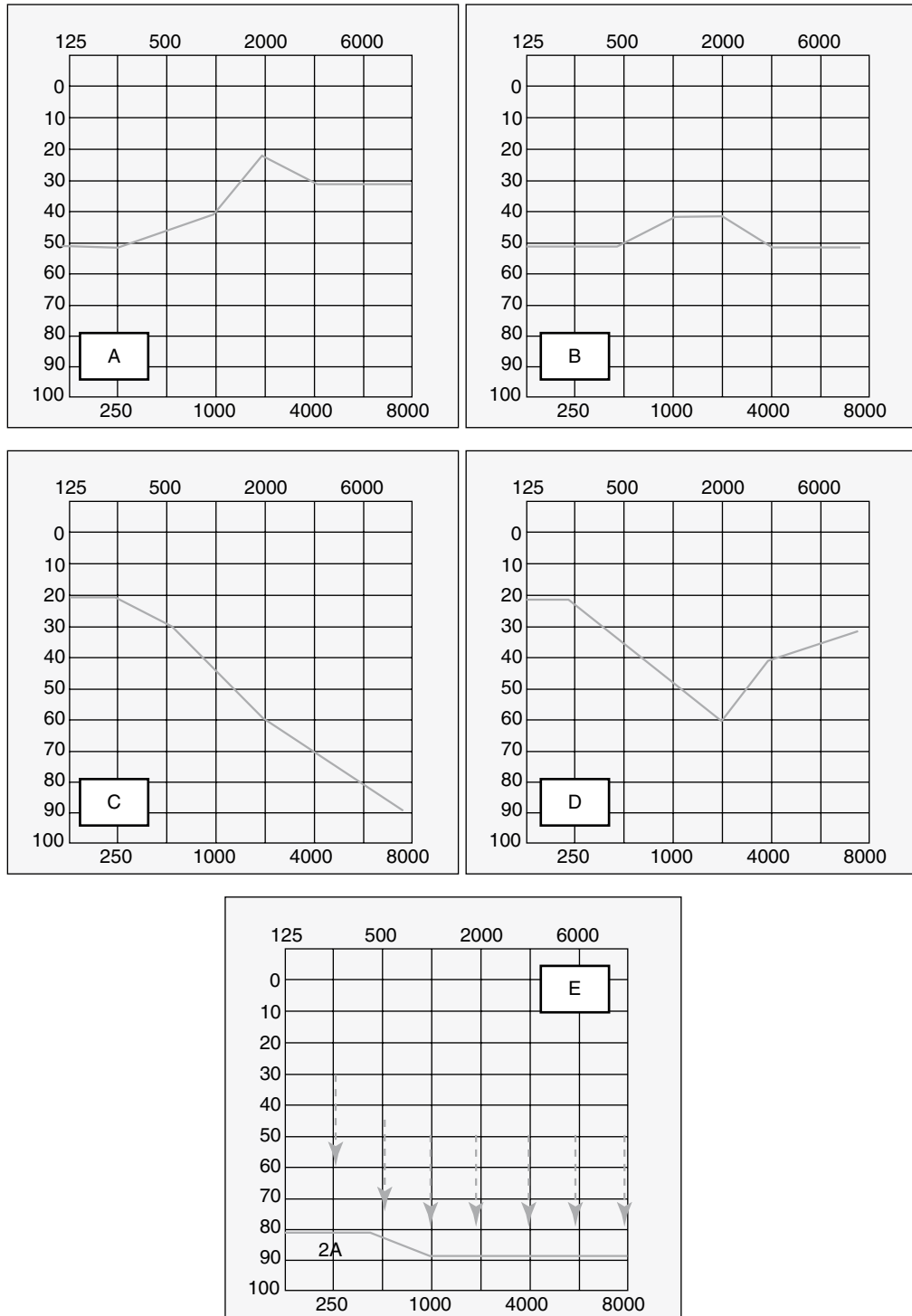


Figure 2 Forms of presentation of sudden deafness. A: Sudden deafness with low frequency SNHL (better prognosis). B: Sudden deafness with pantonal SNHL. C: Sudden deafness with SNHL at high frequencies. D: Sudden deafness with SNHL at medium frequencies. E: Sudden deafness with residual hearing (cophosis).

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).

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}

Table 2 Prognostic factors in sudden deafness

Prognosis	Audiometry		Symptoms			Start of treatment	Age
Worse	SNH in high and medium frequencies	Higher auditory affectation (and cophosis)	Vertigo	Tinnitus	Headache	Late	Elderly
Better	SNH in low and pantonal frequencies	Lower auditory affectation	Absent	Absent	Absent	Early	Young

However, when it affects mostly high tones, SD can be the first symptom of an acoustic neurinoma,^{81,82} even when hearing is restored completely.

Prognosis

The natural history of 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.⁹³⁻⁹⁵ 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.⁹⁶⁻⁹⁹ 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.¹⁰⁰

Table 2 describes the signs of good and bad prognosis.¹⁰⁰⁻¹⁰²

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.

Treatment

Treatment of SD is very controversial, due to the absence of solid evidence to clearly endorse any of the options raised.¹⁰³ The doses employed are also very variable.^{104,105}

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.^{1,2}

Despite the excess of existing literature on SD, there are only a few randomized double-blind controlled trials, which have been reviewed by Cochrane¹⁰⁴ and summarised in a meta-analysis.¹⁰⁶ The most outstanding include the classic work of Wilson et al,¹⁰⁷ 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 al⁵⁰ 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 1998,²⁰ Tucci et al in 2002²¹ and Westerlaken et al in 2003²³ 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-analysis⁵⁵ and by Cochrane.⁶⁰ 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,^{50,51,102,107-113} supported by intratympanic corticosteroids as rescue, with a large volume of recent publications in this respect:¹¹⁴⁻¹⁵¹

- 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^{152,153}:
 - Prednisone (Prednisone 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,^a 1.5 mg/kg bw/day, in decreasing doses every 5 days (for example, a patient weighing 80 kg, 120 mg x

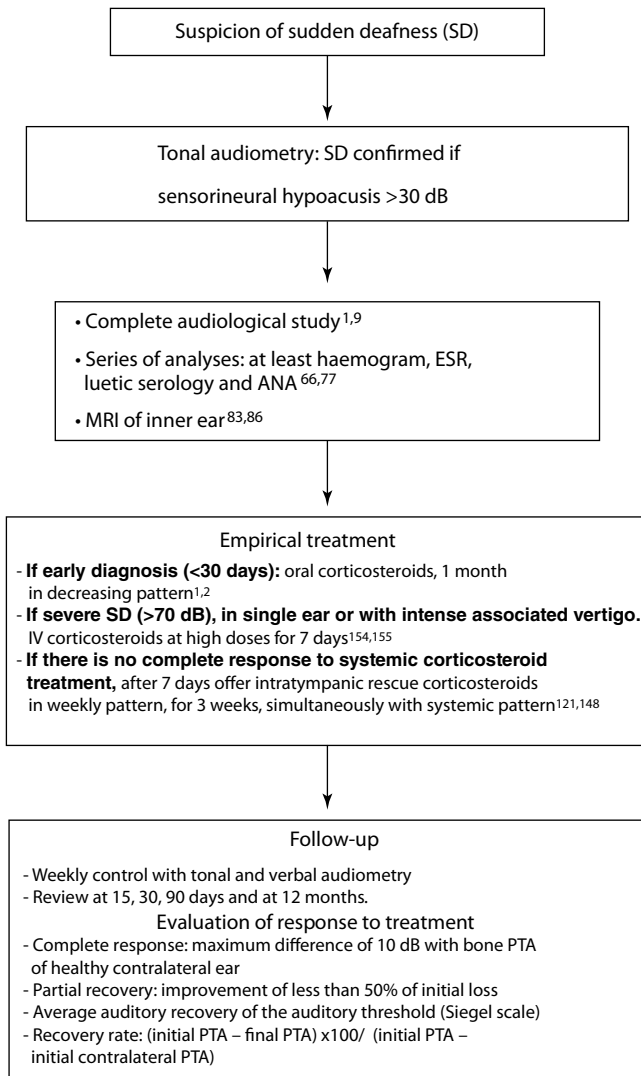


Figure 3 Diagnostic and therapeutic algorithm for sudden deafness.

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.^{154,155} 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,^{121,148} 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)¹⁵⁰:

- 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.

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).^{156,157}

Moreover, given the relative frequency of adverse effects associated to standard treatments for SD,¹⁵⁸⁻¹⁶² 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,^{42,163-167} 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.

- If the diagnosis is late, between 30 days and 90 days after the onset of symptoms, treatment should be with oral corticosteroids for 1 month, following the previous scheme.
- If the diagnosis is very late, more than 90 days from the onset of symptoms, treatment should be discussed individually.

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.

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

Table 3 Criteria for hearing improvement in SD, based on the average hearing threshold

Response	Average auditory recovery
Full recovery	Until obtaining a final PTA <25 dB, or independently of dB gained
Partial improvement	Improvement >15 dB, but final PTA between 25-45 dB
Slight improvement	Improvement >15 dB, but final PTA >45 dB
No improvement	Improvement <15 dB

(Taking the threshold average at 0.25, 0.5, 1, 2, 4 and 8 kHz as PTA) according to Siegel.¹⁷²

Table 4 Criteria for hearing improvement in SD, based on the average hearing threshold

Response	Average auditory recovery
Full recovery	Until obtaining a PTA <20 dB, or until the threshold of the contralateral healthy ear
Good improvement	Improvement >30 dB
Slight improvement	Improvement 10-30 dB
No improvement	Improvement <10 dB

(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.¹⁷³⁻¹⁷⁵

Control should be performed, including tonal and verbal audiometry, at 15, 30 and 90 days of diagnosis.¹⁶⁷⁻¹⁷⁰ 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.^{89,171}

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 8kHz as PTA) of more than 30 dB, or following gradual schemes as proposed in the U.S. by Siegel in the 70s (Table 3),¹⁷² by the Japanese Committee on Sudden Deafness in the 80s (Table 4),¹⁷³⁻¹⁷⁵ or by the Swedish National Register.¹⁷⁶

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:

$$\frac{\text{Rate of recovery}}{\text{Initial PTA} - \text{Final PTA}} \times 100 = \frac{\text{Initial PTA} - \text{Contralateral Initial PTA}}{\text{Initial PTA} - \text{Contralateral Initial PTA}}$$

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.^{101,104,177}

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 lateralised 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 5 Sudden Hearing Loss Registry: initial data

Case	Initial
Hospital	
Date of SD	
Gender	
Date of birth	
Age	
General history	AHT, DM, etc.
ENT history	Single ear, otorrhea sequel
Side	Right, left
Recurrence	Yes/No
Tinnitus	Yes /No
Hyperacusis	Yes /No
Vertigo	Yes /No
Worst PTA	Arithmetical mean of frequencies
Best PTA	0.5, 1, 2, 4 y 8 KHz
Worst URV	
Best URV	
Worst DMax	
Best DMax	
Type of curve	Low/Pantonal/High/Medium/Cophosis
Degree of deafness	Slight/Moderate/Severe

Table 6 Register of sudden deafness: evolution data

	Worst ear	Best ear
7 days	Worst PTA Best PTA Worst URV Best URV Worst DMax Best DMax	
30 days	Worst PTA Best PTA Worst URV Best URV Worst DMax Best DMax	
90 days	Worst PTA Best PTA Worst URV Best URV Worst DMax Best DMax	

Recovery in mean dB and recovery rate.
Aetiological study: MRI, analyses, etc.

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 Sweden¹⁷⁶ or Japan,¹⁷³ 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

1. Rauch SD. Clinical practice. Idiopathic sudden sensorineural hearing loss. *N Engl J Med.* 2008;359:833-40.
2. Schreiber BE, Agrup C, Haskard DO, Luxon LM. Sudden sensorineural hearing loss. *Lancet.* 2010;375:1203-11.
3. O'Malley MR, Haynes DS. Sudden hearing loss. *Otolaryngol Clin North Am.* 2008;41:633-49.
4. Hughes GB, Freedman MA, Haberkamp TJ, Guay ME. Sudden sensorineural hearing loss. *Otolaryngol Clin North Am.* 1996;29:393-405.
5. Wilson WR. Why treat sudden hearing loss. *Am J Otol.* 1984;5:481-3.
6. Fetterman BL, Luxford WM, Saunders JE. Sudden bilateral sensorineural hearing loss. *Laryngoscope.* 1996;106:1347-50.
7. Weber PC. Sudden sensorineural hearing loss. In: UpToDate, Dechler DJ (Ed.), UpToDate, Waltham, MA, 2009.
8. De Kleyn A. Sudden complete or partial loss of function of the octavus system in apparently normal persons. *Acta Otol.* 1944;32:407-29.
9. Tran Ba Huy P, Manach Y. Les Urgences en ORL. Conduite à tenir en Urgence face à une surdit e bruaque. *Rapport de la Soci et e fran aise d'ORL.* 2002:207-25.
10. Merchant SN, Adams JC, Nadol JB. Pathology and pathophysiology of idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2005;26:151-60.
11. Teranishi M, Katayama N, Uchida Y, Tominaga M, Nakashima T. Thirty-year trends in sudden deafness from four nationwide epidemiological surveys in Japan. *Acta Otolaryngol.* 2007;127:1259-65.
12. Klemm E, Deutscher A, M ogges R. Aktuelle Stichprobe zur Epidemiologie des idiopathischen H orsturzes. *Laryngorhinootologie.* 2009;88:524-7.
13. Guillemany JM, Garc a-Pi nero A. Sordera Brusca. *Jano* 23-29 de noviembre 2007; no. 1673:42-5.
14. Chau JK, Lin JR, Atashband S, Irvine RA, Westerberg BD. Systematic review of the evidence for the etiology of adult sudden sensorineural hearing loss. *Laryngoscope.* 2010;120:1011-21.
15. Merchant SN, Durand ML, Adams JC. Sudden deafness: is it viral?. *ORL J Otorhinolaryngol Relat Spec.* 2008;70:52-62.
16. Jourdy DN, Donatelli LA, Victor JD, Selesnick SH. Assessment of variation throughout the year in the incidence of idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2010;31:53-7.

17. García Berrocal JRG, Ramírez-Camacho R, Portero F, Vargas JA. Role of viral and Mycoplasma pneumoniae infection in idiopathic sudden sensorineural hearing loss. *Acta Otolaryngol.* 2000;120:835-59.
18. Kanemaru S, Fukushima H, Nakamura H, Tamaki H, Fukuyama Y, Tamura Y. Alpha-Interferon for the treatment of idiopathic sudden sensorineural hearing loss. *Eur Arch Otorhinolaryngol.* 1997;254:158-62.
19. Pitkaranta A, Julkunen I. Sudden deafness: lack of evidence for systemic viral infection. *Otolaryngol Head Neck Surg.* 1998;118:397-9.
20. Stokroos RJ, Albers FW, Tenvergert EM. Antiviral treatment of idiopathic sudden sensorineural hearing loss: a prospective, randomized, double-blind clinical trial. *Acta Otolaryngol.* 1998;118:488-95.
21. Tucci DL, Farmer JC, Kitch RD, Witsell DL. Treatment of sudden sensorineural hearing loss with systemic steroids and valacyclovir. *Otol Neurotol.* 2002;23:301-8.
22. Uri N, Doweck I, Cohen-Kerem R, Greenberg E. Acyclovir in the treatment of idiopathic sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2003;128:544-9.
23. Westerlaken BO, Stokroos RJ, Dhooge IJ, Wit HP, Albers FW. Treatment of idiopathic sudden sensorineural hearing loss with antiviral therapy: a prospective, randomized, double-blind clinical trial. *Ann Otol Rhinol Laryngol.* 2003;112:993-1000.
24. Zadeh MH, Storper IS, Spitzer JB. Diagnosis and treatment of sudden-onset sensorineural hearing loss: a study of 51 patients. *Otolaryngol Head Neck Surg.* 2003;128:92-8.
25. Capaccio P, Cuccharini V, Ottaviani F, Fracchiolla NS, Bossi A, Pignataro L. Prothrombotic gene mutations in patients with sudden sensorineural hearing loss and cardiovascular thrombotic disease. *Ann Otol Rhinol Laryngol.* 2009;118:205-10.
26. Marcucci R, Alessandrello Liotta A, Cellai AP, Rogolino A, Berloco P, Leprini E, et al. Cardiovascular and thrombophilic risk factors for idiopathic sudden sensorineural hearing loss. *J Thromb Haemost.* 2005;3:929-34.
27. Rudack C, Langer C, Stoll W, Rust S, Walter M. Vascular risk factors in sudden hearing loss. *Thromb Haemost.* 2006;95:454-61.
28. Aimoni C, Bianchini C, Borin M, Ciorba A, Fellin R, Martini A, et al. Diabetes, Cardiovascular Risk Factors and Idiopathic Sudden Sensorineural Hearing Loss: A Case-Control Study. *Audiol Neurootol.* 2009;15:111-5.
29. Ballesteros F, Alobid I, Tassies D, Reverter JC, Scharf RE, Guilemany JM, et al. Is there an overlap between sudden neurosensorial hearing loss and cardiovascular risk factors? *Audiol Neurootol.* 2009;14:139-45.
30. Cadoni G, Scorpecci A, Cianfrone F, Giannantonio S, Paludetti G, Lippa S. Serum fatty acids and cardiovascular risk factors in sudden sensorineural hearing loss: a case-control study. *Ann Otol Rhinol Laryngol.* 2010;119:82-8.
31. Mosnier I, Stepanian A, Baron G, Bodenez C, Robier A, Meyer B, and Thromboembolic Risk Factors in Idiopathic Sudden Sensorineural Hearing Loss: A Case-Control Study. *Audiol Neurootol.* 2010;16:55-66.
32. Nagaoka J, Anjos MF, Takata TT, Chaim RM, Barros F, Penido Nde O. Idiopathic sudden sensorineural hearing loss: evolution in the presence of hypertension, diabetes mellitus and dyslipidemias. *Braz J Otorhinolaryngol.* 2010;76:363-9.
33. Sancho Alvarez A, de Nicolás López R, Sancho Calvo RM. Sordera súbita y prolapso mitral. *Acta Otorrinolaringol Esp.* 1988;39:281-3.
34. Vázquez R, Solanellas J, Alfageme I, et al. Mitral valve prolapse and sudden deafness. *Int J Cardiol.* 2008;124:370-1.
35. Wiles NM, Hunt BJ, Callanan V, Chevretton EB. Sudden sensorineural hearing loss and antiphospholipid syndrome. *Haematologica.* 2006;91(12 suppl):ECR46.
36. Punj J, Pandey R, Darlong V. Sensorineural hearing loss after general anaesthesia: 52 cases reported until now!. *Anaesthesia.* 2009;64:226.
37. Pau H, Selvadurai D, Murty GE. Reversible sensorineural hearing loss after non-otological surgery under general anaesthetic. *Postgrad Med J.* 2000;76:304-6.
38. Herrero Agustín J, González Martín FM, Pinilla Urraca M, Laguna Ortega D, de la Fuente Hernández R. Hemorragia coclear. Causa excepcional de sordera súbita sensorineural. *Acta Otorrinolaringol Esp.* 2002;53:363-8.
39. Salomone R, Abu TA, Chaves AG, Bocalini MC, Vicente Ade O, Riskalla PE. Sudden hearing loss caused by labyrinthine hemorrhage. *Braz J Otorhinolaryngol.* 2008;74:776-9.
40. Rosado WM, Palacios E. Sudden onset of sensorineural hearing loss secondary to intralabyrinthine hemorrhage: MRI findings. *Ear Nose Throat J.* 2008;87:130-1.
41. Lin HC, Chao PZ, Lee HC. Sudden sensorineural hearing loss increases the risk of stroke: a 5-year follow-up study. *Stroke.* 2008;39:2744-8.
42. Agarwal L, Pothier DD. Vasodilators and vasoactive substances for idiopathic sudden sensorineural hearing loss. *Cochrane Database Syst Rev.* 2009;4:CD003422.
43. Yue WL, Li P, Qi PY, Li HJ, Zhou H. Role of low-molecular-weight heparins in the treatment of sudden hearing loss. *Am J Otolaryngol.* 2003;24:328-33.
44. Klingel R, Heibges A, Uygun-Kiehne S, Fassbender C, Mösges R. Rheopheresis for sudden sensorineural hearing loss. *Atheroscler Suppl.* 2009;10:102-6.
45. Heigl F, Hettich R, Suckfuell M, Luebbers CW, Osterkorn D, Osterkorn K, et al. Fibrinogen/LDL apheresis as successful second-line treatment of sudden hearing loss: a retrospective study on 217 patients. *Atheroscler Suppl.* 2009;10:95-101.
46. Julius U, Tselmin S, Fischer S, Passauer J, Bornstein SR. The Dresden Apheresis Center - experience with LDL apheresis and immunoabsorption. *Atheroscler Suppl.* 2009;10:12-6.
47. Mösges R, Köberlein J, Heibges A, Erdtracht B, Klingel R, Lehmacher W, RHEO-ISHL Study Group. Rheopheresis for idiopathic sudden hearing loss: results from a large prospective, multicenter, randomized, controlled clinical trial. *Eur Arch Otorhinolaryngol.* 2009;266:943-53.
48. Canis M, Suckfüll M. Rheopheresis as a successful second-line treatment for sudden hearing loss. *HNO.* 2010;58:443-4.
49. Uygun-Kiehne S, Straube R, Heibges A, Klingel R, Davids H. Rheopheresis for recurrent sudden hearing loss: therapeutic options for patients refractory to infusion therapy. *HNO.* 2010;58:445-51.
50. Cinamon U, Bendet E, Kronenberg J. Steroids, carbogen or placebo for sudden hearing loss: a prospective double-blind study. *Eur Arch Otorhinolaryngol.* 2001;258:477-80.
51. Echarrri RM, Rivera T, Mate MA, Cobeta I. Sordera súbita: eficacia de un protocolo terapéutico. *Acta Otorrinolaringol Esp.* 2000;51:490-4.
52. Kim MG, Jung YG, Eun YG. Effect of steroid, carbogen inhalation, and lipoprostaglandin E(1) combination therapy for sudden sensorineural hearing loss. *Am J Otolaryngol.* 2009 Dec 18. [Epub ahead of print].
53. Aslan I, Oysu C, Veyseller B, Baserer N. Does the addition of hyperbaric oxygen therapy to the conventional treatment modalities influence the outcome of sudden deafness? *Otolaryngol Head Neck Surg.* 2002;126:121-6.
54. Cadoni G, Agostino S, Scipione S, Ippolito S, Caselli A, Marchese R, Paludetti G. Sudden sensorineural hearing loss: our experience in diagnosis, treatment, and outcome. *J Otolaryngol.* 2005;34:395-401.
55. Bennett M, Kertesz T, Yeung P. Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss and tinnitus: a systematic review of randomized controlled trials. *J Laryngol Otol.* 2005;119:791-8.

56. Horn CE, Himel HN, Selesnick SH. Hyperbaric oxygen therapy for sudden sensorineural hearing loss: a prospective trial of patients failing steroid and antiviral treatment. *Otol Neurotol*. 2005;26:882-9.
57. Dundar K, Gumus T, Ay H, Yetiser S, Ertugrul E. Effectiveness of hyperbaric oxygen on sudden sensorineural hearing loss: prospective clinical research. *J Otolaryngol*. 2007;36:32-7.
58. Domachevsky L, Keynan Y, Shupak A, Adir Y. Hyperbaric oxygen in the treatment of sudden deafness. *Eur Arch Otorhinolaryngol*. 2007;264:951-3.
59. Fujimura T, Suzuki H, Shiomori T, Udaka T, Mori T. Hyperbaric oxygen and steroid therapy for idiopathic sudden sensorineural hearing loss. *Eur Arch Otorhinolaryngol*. 2007;264:861-6.
60. Bennett MH, Kertesz T, Yeung P. Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and tinnitus. *Cochrane Database Syst Rev*. 2007;1:CD004739.
61. Cekin E, Cincik H, Ulubil SA, Gungor A. Effectiveness of hyperbaric oxygen therapy in management of sudden hearing loss. *J Laryngol Otol*. 2009;123:609-12.
62. Körpınar S, Alkan Z, Yiğit O, Gör AP, Toklu AS, Cakir B, et al. Factors influencing the outcome of idiopathic sudden sensorineural hearing loss treated with hyperbaric oxygen therapy. *Eur Arch Otorhinolaryngol*. 2010 Jul 14. [Epub ahead of print].
63. Zhuo XL, Wang Y, Zhuo WL, Zhang XY. Is the application of prostaglandin E1 effective for the treatment of sudden hearing loss? An evidence-based meta-analysis. *J Int Med Res*. 2008;36:467-70.
64. Suzuki H, Fujimura T, Shiomori T, Ohbuchi T, Kitamura T, Hashida K, et al. Prostaglandin E1 versus steroid in combination with hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss. *Auris Nasus Larynx*. 2008;35:192-7.
65. García-Berrocal JR, Ramírez-Camacho R, Millán I, Górriz C, Trinidad A, Arellano B, et al. Sudden presentation of immune-mediated inner ear disease: characterization and acceptance of a cochleovestibular dysfunction. *J Laryngol Otol*. 2003;117:775-9.
66. García-Berrocal JR, Trinidad A, Ramírez-Camacho R, Verdager JM, Ibañez A. Immunologic work-up study for inner ear disorders: looking for a rational strategy. *Acta Otolaryngol*. 2005;125:814-8.
67. Toubi E, Ben-David J, Kessel A, Halas K, Sabo E, Luntz M. Immune-mediated disorders associated with idiopathic sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol*. 2004;113:445-9.
68. García Callejo FJ, Velert Vila MM, Laporta P, Orts Alborch MH, de Paula Vernetta C, Marco Algarra J. Titulación de anticuerpos anticocleares mediante western-blot y grado de recuperación auditiva tras corticoterapia en pacientes con sordera súbita. *Acta Otorrinolaringol Esp*. 2004;55:463-9.
69. Wilson H, Alderson DJ. Sudden sensorineural hearing loss: when is it idiopathic?. *J Laryngol Otol*. 2010;124:690-3.
70. Goodhill V. Sudden deafness and window rupture. *Laryngoscope*. 1971;81:1462-74.
71. Simmons FB. Theory of membrane breaks in sudden hearing loss. *Arch Otolaryngol*. 1968;88:41-8.
72. Maier W, Fradis M, Kimpel S, Schipper J, Laszig R. Results of exploratory tympanotomy following sudden unilateral deafness and its effects on hearing restoration. *Ear Nose Throat J*. 2008;87:438-51.
73. Gedlicka C, Formanek M, Ehrenberger K. Analysis of 60 patients after tympanotomy and sealing of the round window membrane after acute unilateral sensorineural hearing loss. *Am J Otolaryngol*. 2009;30:157-61.
74. Yueh B, Shapiro N, MacLean CH, Shekelle PG. Screening and management of adult hearing loss in primary care: scientific review. *JAMA*. 2003;289:1976-85.
75. AEDA. *Auditio: Revista Electrónica de Audiología*. 2002 [accessed 2009 Nov 24]. Available form: <http://www.auditio.com>.
76. Chen CN, Young YH. Differentiating the cause of acute sensorineural hearing loss between Ménière's disease and sudden deafness. *Acta Otolaryngol*. 2006;126:25-31.
77. Lobo D, López FG, García-Berrocal JR, Ramírez-Camacho R. Diagnostic tests for immunomediated hearing loss: a systematic review. *J Laryngol Otol*. 2008;122:564-73.
78. Süslü N, Yılmaz T, Gürsel B. Utility of anti-HSP 70, TNF-alpha, ESR, antinuclear antibody, and antiphospholipid antibodies in the diagnosis and treatment of sudden sensorineural hearing loss. *Laryngoscope*. 2009;119:341-6.
79. Gross M, Eliashar R, Ben-Yaakov A, Ulmanský R, Elidan J. Prevalence and clinical significance of anticardiolipin, anti-beta2-glycoprotein-1, and anti-heat shock protein-70 autoantibodies in sudden sensorineural hearing loss. *Audiol Neurootol*. 2008;13:231-8.
80. García-Berrocal JR, Vargas JA, Ramírez-Camacho RA, González FM, Gea-Banacloche JC, Vergara J, Durántez A. Deficiency of naive T cells in patients with sudden deafness. *Arch Otolaryngol Head Neck Surg*. 1997;123:712-7.
81. Weber PC, Zbar RI, Gantz BJ. Appropriateness of magnetic resonance imaging in sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg*. 1997;116:153-6.
82. Lin YT, Young YH. Retrocochlear mass lesion in mid-frequency sudden deafness. *Otolaryngol Head Neck Surg*. 2008;138:13-7.
83. Nosrati-Zarenoe R, Hansson M, Hultcrantz E. Assessment of diagnostic approaches to idiopathic sudden sensorineural hearing loss and their influence on treatment and outcome. *Acta Otolaryngol*. 2010;130:384-91.
84. Cadoni G, Cianfoni A, Agostino S, Scipione S, Tartaglione T, Galli J, et al. Magnetic resonance imaging findings in sudden sensorineural hearing loss. *J Otolaryngol*. 2006;35:310-6.
85. Aarnisalo AA, Suoranta H, Ylikoski J. Magnetic resonance imaging findings in the auditory pathway of patients with sudden deafness. *Otol Neurotol*. 2004;25:245-9.
86. Chatard-Baptiste S, Martin C, Pouget JF, Veyret C. Surdités brusques: intérêt de l'imagerie: à propos d'une étude prospective de 37 cas. *J Radiol*. 2009;90:1823-35.
87. Tran Ba Huy P, Sauvaget E. La surdit e brusque idiopathique n'est pas, aujourd'hui, une urgence sensorielle. *Ann Otolaryngol Chir Cervicofac*. 2007;124:66-71.
88. Sauvage JP, et al. Sorderas bruscas y fluctuantes. *Enciclopedia M dico-Quir rgica* 2002; ed. Elsevier E-20-183-A-10.
89. Sugiura M, Naganawa S, Ishida IM, Teranishi M, Nakata S, Yoshida T, et al. Vestibular aqueduct in sudden sensorineural hearing loss. *J Laryngol Otol*. 2008;122:887-92.
90. Can IH, G cmen H, Kurt A, Samim E. Sudden hearing loss due to large vestibular aqueduct syndrome in a child: should exploratory tympanotomy be performed?. *Int J Pediatr Otorhinolaryngol*. 2004;68:841-4.
91. Fushiki H, Junicho M, Aso S, Watanabe Y. Recurrence rate of idiopathic sudden low-tone sensorineural hearing loss without vertigo: a long-term follow-up study. *Otol Neurotol*. 2009;30:295-8.
92. Fushiki H, Junicho M, Kanazawa Y, Aso S, Watanabe Y. Prognosis of sudden low-tone loss other than acute low-tone sensorineural hearing loss. *Acta Otolaryngol*. 2010;130:559-64.
93. Finger RP, Gostian AO. Idiopathic sudden hearing loss: contradictory clinical evidence, placebo effects and high spontaneous recovery rate-where do we stand in assessing treatment outcomes? *Acta Otolaryngol*. 2006;126:1124-7.
94. Jeyakumar A, Francis D, Doerr T. Treatment of idiopathic sudden sensorineural hearing loss. *Acta Otolaryngol*. 2006;126:708-13.

95. Alexiou C, Arnold W, Fauser C, Schratzenstaller B, Gloddek B, Fuhrmann S, et al. Sudden sensorineural hearing loss: does application of glucocorticoids make sense?. *Arch Otolaryngol Head Neck Surg.* 2001;127:253-8.
96. Mattox DE, Simmons FB. Natural history of sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol.* 1977;86:463-80.
97. Byl FM. Sudden hearing loss: eight years' experience and suggested prognosis table. *Laryngoscope.* 1984;94:647-61.
98. Simmons FB. Sudden idiopathic sensorineural hearing loss: some observations. *Laryngoscope.* 1973;83:1221-7.
99. Ceylan A, Celenk F, Kemaloğlu YK, Bayazit YA, Gökse N, Ozbilen S. Impact of prognostic factors on recovery from sudden hearing loss. *J Laryngol Otol.* 2007;121:1035-40.
100. Chao TK, Hsiu-Hsi Chen T. Predictive model for improvement of idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2010;31:385-93.
101. Cvorović L, Deric D, Probst R, Hegemann S. Prognostic model for predicting hearing recovery in idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2008;29:449-64.
102. Hikita-Watanabe N, Kitahara T, Horii A, Kawashima T, Doi K, Okumura SI. Tinnitus as a prognostic factor of sudden deafness. *Acta Otolaryngol.* 2010;130:79-83.
103. Conlin AE, Parnes LS. Treatment of sudden sensorineural hearing loss: I. A systematic review. *Arch Otolaryngol Head Neck Surg.* 2007;133:573-81.
104. Wei, B, Mubiru, S, O'leary, S. Steroids for idiopathic sudden sensorineural hearing loss. *Cochrane Database Syst Rev* 2006;CD003998.
105. Shemirani NL, Schmidt M, Friedland DR. Sudden sensorineural hearing loss: An evaluation of treatment and management approaches by referring physicians. *Otolaryngol Head Neck Surg.* 2009;140:86-91.
106. Conlin AE, Parnes LS. Treatment of sudden sensorineural hearing loss: II. A Meta-analysis. *Arch Otolaryngol Head Neck Surg.* 2007;133:582-6.
107. Wilson WR, Byl FM, Laird N. The efficacy of steroids in the treatment of idiopathic sudden hearing loss. A double blind clinical study. *Arch Otolaryngol.* 1980;106:772-6.
108. Moskowitz D, Lee KJ, Smith HW. Steroid use in idiopathic sudden sensorineural hearing loss. *Laryngoscope.* 1984;94:664-6.
109. Kitajiri S, Tabuchi K, Hiraumi H, Hirose T. Is corticosteroid therapy effective for sudden-onset sensorineural hearing loss at lower frequencies. *Arch Otolaryngol Head Neck Surg.* 2002;128:365-7.
110. Chen CY, Halpin C, Rauch SD. Oral steroid treatment of sudden onset sensorineural hearing loss: a ten year retrospective analysis. *Otol Neurotol.* 2003;24:728-33.
111. Slattery WH, Fisher LM, Iqbal Z, Liu N. Oral steroid regimens for idiopathic sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2005;132:5-10.
112. Arellano B, García Berrocal JR, Górriz C, González FM, Vicente J, Ramírez Camacho R. Protocolo de tratamiento de la sordera súbita. *Acta Otorrinolaringol Esp.* 1997;48:513-6.
113. Pino Rivero V, Trinidad Ruíz G, Marcos García M, Montero García C, González Palomino A, Pantoja Hernández CG, et al. Estudio estadístico y resultados clínicos de la comparación de dos protocolos diferentes en el tratamiento de la hipoacusia brusca. *Acta Otorrinolaringol Esp.* 2005;56:63-7.
114. Hu A, Parnes LS. Intratympanic Steroids for Inner Ear Disorders: A Review. *Audiol Neurootol.* 2009;14:373-82.
115. Hamid M, Trune D. Issues, indications, and controversies regarding intratympanic steroid perfusion. *Curr Opin Otolaryngol Head Neck Surg.* 2008;16:434-40.
116. Rauch SD. Intratympanic steroids for sensorineural hearing loss. *Otolaryngol Clin North Am.* 2004;37:1061-74.
117. Parnes LS, Sun AH, Freeman DJ. Corticosteroid pharmacokinetics in the inner ear fluids: an animal study followed by clinical application. *Laryngoscope.* 1999;109:1-17.
118. Chandrasekhar SS. Intratympanic dexamethasone for sudden sensorineural hearing loss: clinical and laboratory evaluation. *Otol Neurotol.* 2001;22:1823.
119. Alles MJ, der Gaag MA, Stokroos RJ. Intratympanic steroid therapy for inner ear diseases, a review of the literature. *Eur Arch Otorhinolaryngol.* 2006;263:791-7.
120. Gianoli GJ, Li JC. Transtympanic steroids for treatment of sudden hearing loss. *Otolaryngol Head Neck Surg.* 2001;125:142-6.
121. Guan-Min H, Hung-Ching L, Min-Tsan S, Tsai HT. Effectiveness of intratympanic dexamethasone injection in sudden-deafness patients as salvage treatment. *Laryngoscope.* 2004;114:1184-9.
122. Gouveris H, Selivanova O, Mann W. Intratympanic dexamethasone with hyaluronic acid in the treatment of idiopathic sudden sensorineural hearing loss after failure of intravenous steroid and vasoactive therapy. *Eur Arch Otorhinolaryngol.* 2005;262:131-4.
123. Herr BD, Marzo SJ. Intratympanic steroid perfusion for refractory sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2005;132:527-31.
124. Battista RA. Intratympanic dexamethasone for profound idiopathic sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2005;132:902-5.
125. Choung YH, Park K, Shin YR, Cho MJ. Intratympanic dexamethasone injection for refractory sudden sensorineural hearing loss. *Laryngoscope.* 2006;116:747-52.
126. Dallan I, Bruschini L, Nacci A, Bruschini P, Traino C, Rognini F, et al. Transtympanic steroids as a salvage therapy in sudden hearing loss: preliminary results. *ORL J Otorhinolaryngol Relat Spec.* 2006;68:247-52.
127. Kopke RD, Hoffer ME, Wester D, O'Leary MJ, Jackson RL. Targeted topical steroid therapy in sudden sensorineural hearing loss. *Otol Neurotol.* 2001;22:475-9.
128. Lefebvre PP, Staecker H. Steroid perfusion of the inner ear for sudden sensorineural hearing loss after failure of conventional therapy: a pilot study. *Acta Otolaryngol.* 2002;122:698-702.
129. Lutterman J, Sudhoff, Junker R. Transtympanic corticoid therapy for acute profound hearing loss. *Eur Arch Otorhinolaryngol.* 2005;262:587-91.
130. Plontke S, Lowenheim H, Preyer S, Leins P, Dietz K, Koitschev A, et al. Outcomes research analysis of continuous intratympanic glucocorticoid delivery in patients with acute severe to profound hearing loss: basis for planning randomized controlled trials. *Acta Otolaryngol.* 2005;125:830-9.
131. Slattery WH, Fisher LM, Iqbal Z, Friedman RA, Liu N. Intratympanic steroid injection for treatment of idiopathic sudden hearing loss. *Otolaryngol Head Neck Surg.* 2005;133:251-9.
132. Roebuck J, Chang CY. Efficacy of steroid injection on idiopathic sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2006;135:276-9.
133. Banerjee A, Parnes LS. Intratympanic corticosteroids for sudden idiopathic sensorineural hearing loss. *Otol Neurotol.* 2005;26:878-81.
134. Xenellis J, Papadimitriou N, Nikolopoulos T, Maragoudakis P, Segas J, Tzagaroulakis A, et al. Intratympanic steroid treatment in idiopathic sudden sensorineural hearing loss: a control study. *Otolaryngol Head Neck Surg.* 2006;134:940-5.
135. Kakehata S, Sasaki A, Oji K, Futai K, Ota S, Makinae K, et al. Comparison of intratympanic and intravenous dexamethasone treatment on sudden sensorineural hearing loss with diabetes. *Otol Neurotol.* 2006;27:604-8.
136. Haynes DS, O'Malley M, Cohen S, Watford K, Labadie RF. Intratympanic dexamethasone for sudden sensorineural hearing loss after failure of systemic therapy. *Laryngoscope.* 2007;117:3-15.
137. Fitzgerald DC, McGuire JF. Intratympanic steroids for idiopathic sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol.* 2007;116:253-6.

138. Van Wijck F, Staecker H, Lefebvre PP. Topical steroid therapy using the Silverstein Microwick in sudden sensorineural hearing loss after failure of conventional treatment. *Acta Otolaryngol.* 2007;127:1012-7.
139. Kara E, Cetik F, Tarkan O, Sürmelioglu O. Modified intratympanic treatment for idiopathic sudden sensorineural hearing loss. *Eur Arch Otorhinolaryngol.* 2010;267:701-7.
140. Hong SM, Park CH, Lee JH. Hearing outcomes of daily intratympanic dexamethasone alone as a primary treatment modality for ISSHL. *Otolaryngol Head Neck Surg.* 2009;141:579-83.
141. Kiliç R, Safak MA, Oğuz H, Kargin S, Demirci M, Samim E, et al. Intratympanic methylprednisolone for sudden sensorineural hearing loss. *Otol Neurotol.* 2007;28:312-6.
142. Han CS, Park JR, Boo SH, Jo JM, Park KW, Lee WY, et al. Clinical efficacy of initial intratympanic steroid treatment on sudden sensorineural hearing loss with diabetes. *Otolaryngol Head Neck Surg.* 2009;141:572-8.
143. Ahn JH, Yoo MH, Yoon TH, Chung JW. Can intratympanic dexamethasone added to systemic steroids improve hearing outcome in patients with sudden deafness?. *Laryngoscope.* 2008;118:279-82.
144. Battaglia A, Burchette R, Cueva R. Combination therapy (intratympanic dexamethasone + high-dose prednisone taper) for the treatment of idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2008;29:453-60.
145. Plontke SK, Lowenheim H, Mertens J, Engel C, Meisner C, Weidner A, et al. Randomized, double blind, placebo controlled trial on the safety and efficacy of continuous intratympanic dexamethasone delivered via a round window catheter for severe to profound sudden idiopathic sensorineural hearing loss after failure of systemic therapy. *Laryngoscope.* 2009;119:359-69.
146. Zernotti ME, Paoletti OA, Zernotti M, Martínez ME, Roques-Revil M, Prina AC. Dexametasona intratimpánica como opción terapéutica en hipoacusia neurosensorial súbita. *Acta Otorrinolaringol Esp.* 2009;60:99-103.
147. Lee HS, Kim JM, Kim YJ, Chung DH, Seo BS, Kim SH. Results of intratympanic dexamethasone injection as salvage treatment in idiopathic sudden hearing loss. *J Otolaryngol Head Neck Surg.* 2008;37:263-8.
148. Plaza G, Herráiz C. Intratympanic steroids for treatment of sudden hearing loss after failure of intravenous therapy. *Otolaryngol Head Neck Surg.* 2007;137:74-8.
149. Dallan I, De Vito A, Fattori B, Casani AP, Panicucci E, Berrettini S, Marchetti M, et al. Intratympanic Methylprednisolone in Refractory Sudden Hearing Loss: A 27-Patient Case Series With Univariate and Multivariate Analysis. *Otol Neurotol.* 2010;31:25-30.
150. Herraiz C, Aparicio JM, Plaza G. La vía intratimpánica en el tratamiento de la patología de oído interno. *Acta Otorrinolaringol Esp.* 2010;61:225-32.
151. She W, Dai Y, Du X, Yu C, Chen F, Wang J, Qin X. Hearing evaluation of intratympanic methylprednisolone perfusion for refractory sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2010;142:266-71.
152. Deflazacort como glucocorticoide por vía oral. Institut Català de Farmacologia. 2002 [accessed 2009 Nov 24]. Available from: <http://www.icf.uab.es/HOSPITAL/informes/cast/2002/ IPI802deflazacort-es.pdf>.
153. Rhen T, Cidlowski J. Anti-inflammatory action of glucocorticoids: new mechanisms for old drugs. *N Engl J Med.* 2005;353:1711-23.
154. Aoki D, Takegoshi H, Kikuci S. Evaluation of super-highdose steroid therapy for sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg.* 2006;134:783-878.
155. Westerlaken BO, de Kleine E, van der Laan B, Albers F. The treatment of idiopathic sudden sensorineural hearing loss using pulse therapy: a prospective, randomized, double-blind clinical trial. *Laryngoscope.* 2007;117:684-90.
156. Bone and Tooth Society of Great Britain, National Osteoporosis Society and Royal College of Physicians. Guidelines on the prevention and treatment of glucocorticoid induced osteoporosis [accessed 2009 Nov 24]. Available from: <http://www.rcplondon.ac.uk/pubs/books/glucocorticoid/-glucocortConcise.pdf>.
157. Sosa Henriquez M. Grupo de Trabajo en Osteoporosis de la Sociedad Española de Medicina Interna. (GTO-SEMI). Guía de prevención y tratamiento de la osteoporosis inducida por corticoides [accessed 2009 Nov 24]. Available from: http://www.fesemi.org/grupos/osteoporosis/-publicaciones/guia_gio_semi.pdf.
158. García-Berrocal JR, Ramírez-Camacho R, Lobo D, Trinidad A, Verdaguer JM. Adverse effects of glucocorticoid therapy for inner ear disorders. *ORL J Otorhinolaryngol Relat Spec.* 2008;70:271-4.
159. Buttgereit F, da Silva JA, Boers M, Burmester GR, Cutolo M, Jacobs J, et al. Standardised nomenclature for glucocorticoid dosages and glucocorticoid treatment regimens: current questions and tentative answers in rheumatology. *Ann Rheum Dis.* 2002;61:718-22.
160. Hoes JN, Jacobs JW, Verstappen SM, Bijlsma JW, Van der Heijden GJ. Adverse events of low- to medium-dose oral glucocorticoids in inflammatory diseases: a meta-analysis. *Ann Rheum Dis.* 2009;68:1833-8.
161. Free RH, Smale ND, De Kleine E, Van Der Laan BFAM. Side effects of oral dexamethasone pulse therapy for idiopathic sudden sensorineural hearing loss. *Otol Neurotol.* 2009;30:691.
162. Poetker DM, Reh DD. A comprehensive review of the adverse effects of systemic corticosteroids. *Otolaryngol Clin North Am.* 2010;43:753-68.
163. Shaia F, Sheehy J. Sudden sensorineural hearing impairment: a report of 1220 cases. *Laryngoscope.* 1976;86:389-98.
164. Probst R, Tschopp K, Lüdin E, Kellerhals B, Podvinec M, Pfaltz CR. A randomized, double-blind, placebo-controlled study of dextran/pentoxifylline medication in acute acoustic trauma and sudden hearing loss. *Acta Otolaryngol.* 1992;112:435-43.
165. Burschka MA, Hassan HA, Reineke T, van Bebbler L, Caird DM, Mösges R. Effect of treatment with Ginkgo biloba extract EGb 761 (oral) on unilateral idiopathic sudden hearing loss in a prospective randomized double-blind study of 106 outpatients. *Eur Arch Otorhinolaryngol.* 2001;258:213-9.
166. Kronenberg J, Almagor M, Bendet E, Kushnir D. Vasoactive therapy versus placebo in the treatment of sudden hearing loss: a double-blind clinical study. *Laryngoscope.* 1992;102:65-8.
167. Moon IS, Kim J, Lee SY, Choi HS, Lee WS. How long should the sudden hearing loss patients be followed after early steroid combination therapy? *Eur Arch Otorhinolaryngol.* 2009;266:1391-5.
168. Yeo SW, Lee DH, Jun BC, Park SY, Park YS. Hearing outcome of sudden sensorineural hearing loss: long-term follow-up. *Otolaryngol Head Neck Surg.* 2007;136:221-4.
169. Canis M, Osterkorn D, Osterkorn K, Suckfuell M. A Retrospective Assessment of 741 Patients with Sudden Hearing Loss. *The Open Otorhinolaryngology Journal.* 2009;3:5-10. 5
170. Psifidis AD, Psillas GK, Daniilidis JCh. Sudden sensorineural hearing loss: long-term follow-up results. *Otolaryngol Head Neck Surg.* 2006;134:809-15.
171. Kallinen J, Laurikainen E, Bergroth L, Grénman R. A follow-up study of patients suffering from sudden sensorineural hearing loss. *Acta Otolaryngol.* 2001;121:818-22.
172. Siegel LG. The treatment of idiopathic sudden sensorineural hearing loss. *Otolaryngol Clin North Am.* 1975;8:467-73.

173. Yanagita N, Nakashima T, Ohno Y, Kanzaki J, Shitara T. Estimated Annual Number of Patients Treated for Sensorineural Hearing Loss in Japan Results of a Nationwide Epidemiological Survey in 1987. *Acta Otolaryngol (Stockh)*. 1994;9-13.
174. Kanzaki J. Diagnostic Criteria for Acute Profound Deafness. *Acta Otolaryngol (Stockh)*. 1994;7-8.
175. Kanzaki J, Inoue Y, Ogawa K, et al. Effect of single-drug treatment on idiopathic sudden sensorineural hearing loss. *Auris Nasus Larynx*. 2003;30:123-7.
176. Nosrati-Zarenoe R, Arlinger S, Hultcrantz E. Idiopathic sudden sensorineural hearing loss: results drawn from the Swedish national database. *Acta Otolaryngol*. 2007;127:1168-75.
177. Plontke SK, Bauer M, Meisner C. Comparison of pure-tone audiometry analysis in sudden hearing loss studies: lack of agreement for different outcome measures. *Otol Neurotol*. 2007;28:753-63.