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Sudden hypoacusis treated with hyperbaric oxygen therapy: A controlled study

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Abstract

The term sudden hypoacusis *describes a hearing loss of rapid onset and unknown origin that can progress to severe deafness. Of the many therapeutic protocols that have been proposed for treating sudden hypoacusis, hyperbaric oxygen therapy (HOT) plays a leading role. We studied 50 patients who had been referred to our ENT unit within 48 hours of the onset of sudden hypoacusis. We randomly assigned 30 of these patients to undergo once-daily administration of HOT for 10 days; the other 20 patients were treated for 10 days with an intravenous vasodilator. Response to therapy in all patients was evaluated by calculating the mean hearing threshold at frequencies between 500 and 4,000 Hz and by assessing liminal tonal audiometry results recorded at baseline and 10 days after the cessation of treatment. These results, plus the findings of other audiologic and otoneurologic examinations, revealed that the patients in the HOT group experienced a significantly greater response to treatment than did those in the vasodilator group, regardless of age and sex variables. Significantly more patients in the HOT group experienced a* good *or* significant *response. In both groups, patients with pantonal hypoacusis responded significantly better than did those with a milder condition. Based on our findings, coupled with the fact that oxygen therapy is well tolerated and produces no side effects, we conclude that HOT should be considered the preferred treatment for patients with sudden hypoacusis.*

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Introduction

Sudden hypoacusis is defined as a sensorineural hearing loss of rapid progression and unknown etiology that can progress to severe deafness.[1,2] Tinnitus is often associated with this condition, but nonrecurrent vertigo is rare. Although the etiopathogenic mechanism of sudden hypoacusis is not known, the most common causes are believed to be either viral[2-6] or vascular in nature.[3,7] Other proposed causes include autoimmune disorders[8-10] and a parcellar rupture of the labyrinthine membrane.[11]

The difficulty in pinpointing the etiology of sudden hypoacusis has hindered efforts to treat it.[12] Many treatment protocols have been proposed by various authors. Among the variety of drug therapies that have been used are vasodilators (e.g., carbogen, alpha blockers, and calcium antagonists) to improve oxygenation of the cochlear tissues,[13-16] corticosteroids to counteract a possible autoimmune mechanism and to exert antiphlogistic and antiedematogenic effects,[10,17] and osmotic drugs (e.g., mannitol) to modify the hydrostatic pressure of the perilymph and increase the amount of blood in the cochlea by creating an osmotic gradient between plasma and perilymph.[10,18-21] The use of dextran has also been suggested as a means of creating a hypervolemic hemodilution, thus causing a decrease in blood viscosity and an increase in cochlear blood flow.[22-24] Some authors have employed normovolemic hemodilution to produce a decrease in hematocrit without reducing volemia.[25-28]

In addition to all these therapies, another effective treatment for sudden hypoacusis is hyperbaric oxygen therapy (HOT).[29-38] During HOT, a significant increase in

Table. Pretreatment audiologic and vestibular findings in the HOT* and vasodilator groups (n [%])

Group	Hearing loss			Audiometric curve			VOR†	VSR§
	Mild	Moderate	Severe	Flat	Upward	Downward		
HOT (n = 30)	10 (33.3)	11 (36.7)	9 (30.0)	13 (43.3)	6 (20.0)	11 (36.7)	4 (13.3)	5 (16.7)
Vasodilator (n = 20)	6 (30.0)	9 (45.0)	5 (25.0)	10 (50.0)	4 (20.0)	6 (30.0)	5 (25.0)	5 (25.0)

** Hyperbaric oxygen therapy.*
† Vestibulo-ocular reflex; unilateral canal paresis.
§ Vestibulospinal reflex; pathologic postural sway.

the amount of oxygen that is physically dissolved in plasma leads to an improvement of oxygen distribution throughout the tissues. The acoustic ciliate cells and the peripheral fibers of the acoustic nerve receive not only direct oxygenation via the bloodstream, but supplemental oxygenation through the perilymph and cortilymph.[38] Moreover, oxygen therapy also facilitates cell metabolism, even when the blood supply is poor, and thus promotes a more rapid and complete functional recovery.

In this article, we describe our comparison of outcomes in a group of 50 patients with sudden hypoacusis who were treated with either HOT or intravenous vasodilation within 48 hours of the onset of their symptoms.

Patients and methods

We studied 50 patients with sudden hypoacusis of unknown etiology who had been referred to the ENT Clinic at Pisa University within 48 hours of the onset of acute symptoms. Thirty of these patients (19 men and 11 women; mean age: 44 yr) were randomly assigned to undergo treatment with HOT, and 20 (11 men and 9 women; mean age: 47 yr) were selected for treatment with intravenous vasodilation. In the HOT group, pathologic anamnesis revealed that two patients had type 2 diabetes and one had mild arterial hypertension that was kept well under control with medication. In the vasodilator group, one patient had type 2 diabetes and two had arterial hypertension that was well controlled with drug therapy. We did not include in this study any patient who manifested a probable cause of sudden hypoacusis, such as acoustic trauma, a recent viral episode, or an important cardiovascular condition.

Pretreatment. A diagnosis of sudden hypoacusis was confirmed in both groups by a series of baseline tests, including liminal tonal audiometry, tympanometry, and auditory evoked potentials (table). Assessment of hearing loss in the tonal audiometric test was performed after classifying patients as having one of three categories of hypoacusis according to their pure-tone average (PTA) at frequencies of 500, 1,000, 2,000, and 4,000 Hz. The three categories were *mild* (PTA: ≤40 dB), *moderate* (PTA: >40 and ≤70 dB), and *severe* (PTA: >70 dB).

At baseline, these tests revealed that 10 HOT patients (33.3%) had mild hypoacusis, 11 (36.7%) had moderate hypoacusis, and nine (30%) had severe hypoacusis. The audiometric curve was flat in 13 patients (43.3%), upward-sloping in six (20%), and downward-sloping in 11 (36.7%). Twenty-five of the 30 patients (83.3%) complained of tinnitus in the affected ear.

In the vasodilator group, six patients (30%) had mild hypoacusis, nine (45%) had moderate hypoacusis, and five (25%) had severe hypoacusis. The liminal tonal audiometric test revealed a flat curve in 10 patients (50%), an upward-sloping curve in four (20%), and a downward-sloping curve in six (30%). Eighteen of these patients (90%) reported tinnitus in the affected ear.

All patients also underwent four other pretreatment tests: videonystagmography to evaluate the vestibulo-ocular reflex, static posturography to test the vestibulospinal reflex, computed tomography (CT) and/or magnetic resonance imaging (MRI) of the head to look for pathology, and Doppler echo testing on the cerebroafferent vessels. Baseline videonystagmography revealed that four HOT patients (13.3%) and five vasodilator patients (25%) had a peripheral vestibular deficit on the side of the hearing loss. Static posturography revealed that five patients in each group had postural instability with peripheral characteristics.

Treatment. The patients in the oxygen group underwent 10 once-daily sessions of HOT delivered in a multiperson chamber. Pure oxygen was administered through a mouth-and-nose mask at a constant level of 2.2 atmospheric pressures (ATA) for 90 minutes per session.

The patients in the medical therapy group underwent a 10-day course of intravenous vasodilator treatment (i.e., single injections of 200 mg/day of buflomedil in 250 ml of physiologic solution). Our intention was to suspend

treatment only if hearing recovered completely before the completion of the series (relative gain: 100%), which occurred in two patients.

Post-treatment. Baseline testing was repeated on all patients 10 days following the cessation of therapy. Assessments of both HOT and pharmacologic therapy were based on a comparison of baseline and post-treatment PTA values. Responses were quantified by calculating the relative gain (i.e., the value of the mean total gain divided by the value of the mean initial loss). Responses were classified as either *good* (relative gain: ≥50%), *significant* (≥25 and <50%), or *insignificant* (<25%).[1,28]

Statistical calculations were made according to analysis of covariance, using baseline PTA values, age, and sex as covariants. When differences were statistically significant ($p \leq 0.05$), a post hoc multiple comparison (Bonferroni test) was performed.

Results

Liminal tonal audiometric test results showed that the HOT group experienced a significantly greater mean improvement in PTA values than did the vasodilator group (relative gain: 61.3 [±33.6] vs 24.0% [±22.5]; $p = 0.005$) (figure 1). Improvement was independent of age and sex.

Analysis of relative gains showed that 17 HOT patients (56.7%) experienced a *good* recovery and eight (26.7%) had a *significant* recovery, while only five (16.7%) had an *insignificant* response (figure 2). By contrast, only five vasodilator patients (25%) had a *good* response and six (30%) had a *significant* response; nine (45%) had an *insignificant* response.

In both groups, patients with *severe* hypoacusis (PTA: >70 dB) experienced a significantly greater improvement ($p<0.05$) than did those with *mild* hypoacusis (PTA: ≤40 dB). We did not find any significant differences in either group in the degree of improvement between those with *mild* and *moderate* hypoacusis or between those with *moderate* and *severe* hypoacusis.

Patients in both groups who had a pantonal type of hearing loss achieved a significantly higher relative gain than did those whose hearing loss was mainly in the medium to high frequencies ($p<0.05$). On the other hand, we found no statistically significant difference in improvement in either group between those who had pantonal sensorineural hypoacusis and those who had hypoacusis mainly at the low frequencies (upward-rising curve). Likewise, there was no significant difference in improvement in either group between those who had sensorineural hypoacusis in the medium to high frequencies and those who had upward-sloping curves. Analysis of auditory evoked potentials confirmed the cochlear pattern and the behavior of hearing recovery that was detected by liminal tonal audiometry.

Post-treatment videonystagmography showed that three HOT patients (10%) and two vasodilator patients (10%) maintained persistent signs of a peripheral, compensating deficit. Static posturography values showed practically no change in either group.

Findings on CT and MRI were negative in all patients, as were all neurologic evaluations and all Doppler echo investigations of the cerebroafferent vessels (with the exception of five patients who exhibited slight intimal thickening).

Discussion

Even though the etiology of sudden hypoacusis is unknown, the therapeutic approach to it is substantially the same as that taken for patients for whom a cause of sudden deafness can be traced.

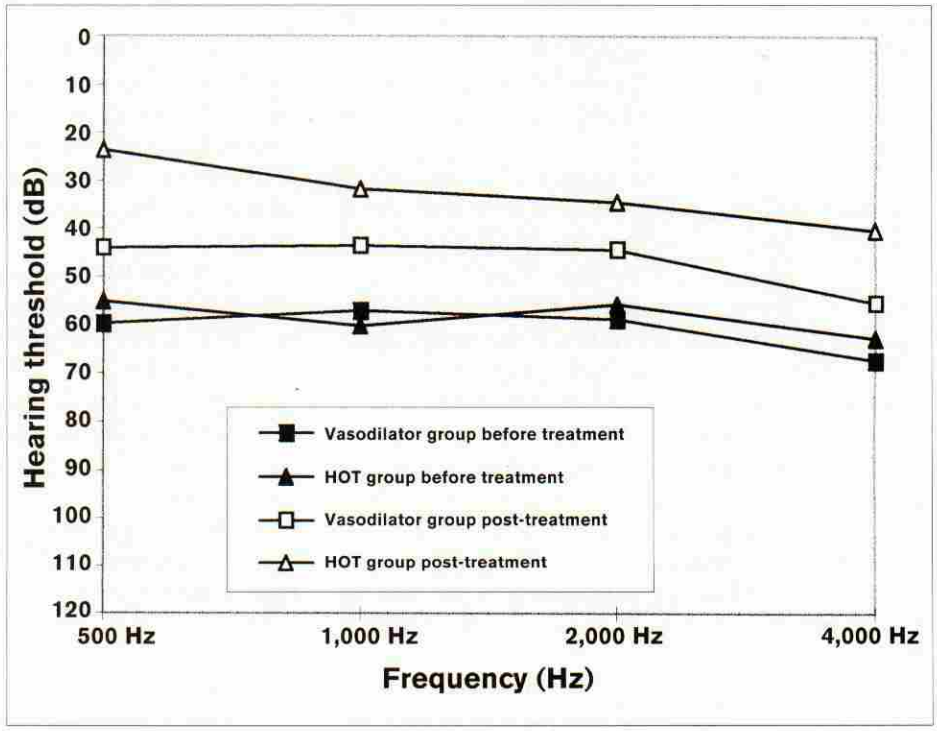

Figure 1. Mean hearing-threshold values at frequencies between 500 and 4,000 Hz in the HOT and vasodilator groups before and after treatment.

Viral and hydropic factors have been proposed by several authors as possible causes of sudden hypoacusis, while other researchers believe that a vascular component plays an important role.[2-7] It is well known that prolonged hypoxia can give rise to important perturbations in the auditory cells and even destroy them. In such cases, hearing damage can be reversed if the vascular obstruction is incomplete or if the labyrinth exerts a protective mechanism against the anoxic condition.

Sudden hypoacusis demands urgent diagnosis and therapy in an otolaryngology environment. Treatment should be started within 48 hours of the onset of symptoms to achieve the best chance of recovery. First introduced at the Marseille School in France, HOT has been used as a treatment for sudden hypoacusis for several decades.[29,30] HOT is based on the principle that a rise of at least 20% (1.2 bar) in barometric pressure will cause a partial rise in the blood oxygen level. High concentrations of pure oxygen in inspired air produce a considerable increase in the amount of oxygen that is physically dissolved in the blood—perhaps as much as 15 times more than what is dissolved at normal atmospheric pressure (1.0 ATA). When air at 1.0 ATA is inhaled, only 0.32% of the volume of oxygen is physically dissolved; when pure oxygen at 1.0 ATA is inhaled, the volume can rise to as much as 2.09%, and at 2.0 ATA, it can increase to 4.44%.

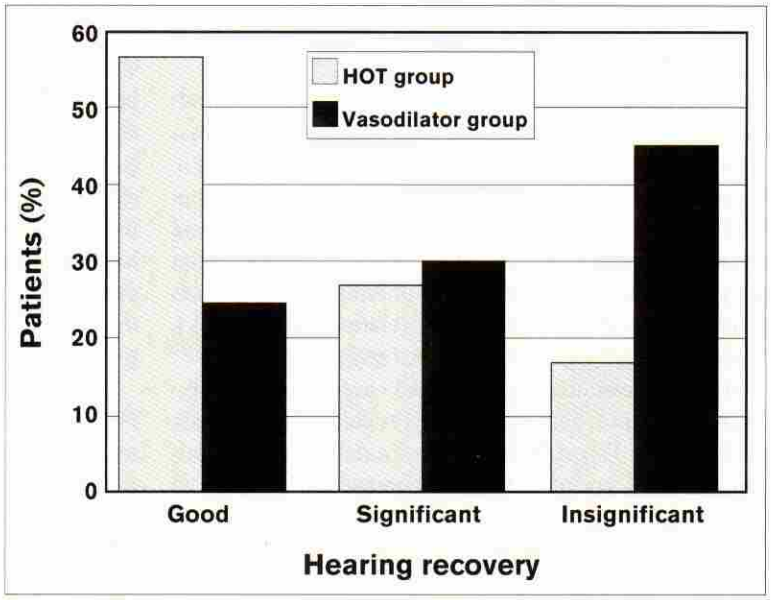

Figure 2. Percentages of patients in the HOT and vasodilator groups who experienced a good response (relative gain: ≥50%), a significant response (≥25 and <50%), and an insignificant response (<25%), as determined by tonal audiometric testing.

HOT not only raises the amount of oxygen that is physically dissolved in the blood, it also increases the level of PO_2 in the inner ear by means of diffusion through the round window and a subsequent increase in the proportion of oxygen that is dissolved in the labyrinthine liquids. The improved oxygenation of the inner ear activates cell metabolism and the Na^+K^+ pump, and it leads to a restoration of the ionic balance and the electrophysiologic functions of the cochlea. HOT is also capable of causing a reduction in hematocrit and blood viscosity, and this can have a rheologic effect in the cochlear region.[39] During HOT, therefore, hypoxic areas of the cochlea can be infused with high partial pressures of oxygen, and this infusion gives rise to an acceleration of the biologic cellular mechanisms that are involved in functional recovery.

In our study, statistical analysis showed that the HOT group had a significantly greater recovery of hearing than did the vasodilator group (p=0.005). The improvement in hearing threshold was associated with both the type of audiometric curve and the degree of hypoacoustic symptoms. Moreover, in both groups of patients, the treatment response was better in those whose hearing loss was pantonal and in those who had *severe* hypoacusis (PTA: >70 dB).

Recovery in both groups was independent of age, sex, and the presence of any concomitant diseases. In addition, any association between hearing damage and vestibular damage (i.e., unilateral canal paresis, which was present in 13.3% of the HOT patients and 25% of the vasodilator patients) was not considered to be predictive of a negative prognosis.

Not only did HOT produce a greater relative auditory gain than did pharmacologic treatment, but a large majority of HOT patients (25 of 30 [83.3%]) experienced either a *good* (relative gain: ≥50%) or *significant* (≥25 and <50%) response. On the other hand, a large minority (nine of 20 [45%]) of the vasodilator patients experienced an *insignificant* response, and only five of them (25%) had a *good* response.

On the basis of our findings and in light of the high degree of tolerability of this procedure, we believe that HOT must be considered the preferred treatment for sudden hypoacusis.

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