Systemic Hypotension and the Development of Acute Sensorineural Hearing Loss in Young Healthy Subjects

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Background: Sudden sensorineural hearing loss (SSHL) is an acute disorder whose origin is often unclear. A vascular disorder may be a causative factor.

Objective: To determine whether hypotension influences the genesis of SSHL in healthy subjects.

Design: To investigate the role of a 24-hour blood pressure (BP) profile in a population of young subjects with SSHL from January 1, 1996, to December 31, 1999, by a nonrandomized controlled trial.

Setting: The Ear, Nose and Throat Section of the Department of Surgical and Anaesthesiological Sciences and the Department of Internal Medicine, S. Orsola Hospital, University of Bologna, Bologna, Italy.

Patients: The study population consisted of 23 untreated healthy patients diagnosed as having SSHL compared with 20 age- and sex-matched normotensive control subjects. Both groups underwent 24-hour BP monitoring, and their BP profiles were analyzed and compared with routine BP values. The data were analyzed with the Statistical Package for the Social Sciences, version 7.1, and the results are expressed as mean±SD.

Main Outcome Measures: The mean BP values were expected to be lower in the study population.

Results: The average clinic and ambulatory BP values were significantly lower in patients with SSHL, for systolic (clinic, $P = .004$; ambulatory BP, $P = .02$) and diastolic (clinical, $P = .03$; ambulatory BP, $P = .03$) values. The occurrence of persistent hypotension (the presence of ≥2 consecutive recordings of systolic BP of ≤105 mm Hg and/or diastolic BP of ≤60 mm Hg) was increased in the population with SSHL.

Conclusion: Systemic hypotension must be considered as the possible cause responsible for the development of SSHL in young healthy subjects.


SUDDEN sensorineural hearing loss (SSHL) is an acute disorder that affects a considerable proportion of the adult population of both sexes.1 Its etiology is still uncertain, and many different possibilities have been suggested, ranging from viral infections to systemic or local circulatory defects.2-7 As for the latter, a causative role is generally accepted for a sudden increase in systemic blood pressure (BP) values either in normotensive patients or in subjects with arterial hypertension in whom the sustained BP increase could be responsible for the local development of thrombotic and/or hemorrhagic complications at the site of an end organ. From an opposite viewpoint, to further investigate the mechanism(s) responsible for the onset of SSHL, the role of systemic hypotension has been considered.8-11 A preliminary report11 suggested that SSHL is common in young subjects who frequently experience a complete recovery of hearing function.7 In these patients, who are usually completely free from the more common vascular risk factors, a possible functional origin of SSHL11 related to the negative hemodynamic effects of arterial hypotension over the terminal-type cochlear vascularization has been hypothesized. Indeed, the routine BP values recorded in our young adult patients complaining of SSHL were significantly ($P = .005$) lower when compared with those of an age-matched control group, thus suggesting the need for further and more complete investigations in this field. In particular, these observations, if confirmed, could have some important implications for the therapeutic approach to SSHL, with a strong limitation in the use of vasodilatory and antihypertensive drugs that could further decrease the local cochlear perfusion pressure and enhance the sensorial loss, thus reducing the probability of a complete functional recovery.

This study investigates the characteristics of the 24-hour BP profile of young subjects with SSHL.
PATIENTS AND METHODS

Forty-seven patients (30 women and 17 men) younger than 50 years (mean±SD age, 37.10±7.94 years; range, 22-49 years), admitted, from January 1, 1996, to December 31, 1999, to the Ear, Nose and Throat Section of the S. Orsola Hospital of the University of Bologna, Bologna, Italy, with the diagnosis of SSHL, were subsequently studied. The diagnosis of SSHL was based on the widely accepted definition of a sensorineural hearing loss of 30 dB or more over at least 3 contiguous audiometric frequencies occurring within 3 days or less and without any other otologic cause for hearing impairment. Our diagnostic protocol included a complete clinical examination, including anamnesis, otoscopy, and an audiometric test battery. Evoked-response audiometry was always used; when the ipsilateral pattern was absent or evocative for retrocochlear involvement, a cerebral magnetic resonance image with gadolinium was obtained. In 12 patients, the type of audiometric curve suggested performing a glycerol perfusion, which proved effective in 7 (partial [5 patients] or complete [2 patients] immediate recovery). However, as in previous studies,1,2,13 we considered these patients to be included in our series based on the possibility of an involvement of the same hypotensive mechanisms as in menieric hearing loss.8,12 In any case, no vestibular symptoms were observed at follow-up in these patients.

Patients affected by arterial hypertension, diabetes (insulin dependent and non–insulin dependent), and peripheral vascular disease and those with a history of coronary or cerebrovascular accidents were excluded from the study. We also excluded the patients unable to cooperate with the study protocol, those who refused to give informed consent, and those showing a poor capacity to comply with the procedures for 24-hour BP monitoring. Patients were also excluded if they were taking any kind of cardiovascular, vasoactive, and/or antiplatelet drug.

The final population examined included 23 untreated normotensive patients (17 women and 6 men) whose age ranged between 22 and 49 years (mean±SD age, 36.4±8.0 years). The study group was compared with a group of 20 age- and sex-matched untreated normotensive control subjects (12 females and 8 males), comparable for age (mean±SD age, 34.3±7.0 years; age range, 16-44 years), admitted to the Department of Internal Medicine of the S. Orsola Hospital of the University of Bologna for reasons other than cardiovascular diseases. Both groups of patients were examined according to the same study protocol, which was approved by the Ethical Committee of the University of Bologna, and informed consent was obtained from each subject before inclusion in the trial.

Clinic BP measurements were obtained with a conventional mercury sphygmomanometer using standardized criteria13 after patients had been in the seated position for 5 minutes and again 2 minutes after patients had assumed the standing position. The fifth Korotkoff sound was used to define the diastolic BP (DBP).

Ambulatory BP monitoring was performed with an automated portable commercial instrument (model 90207; SpaceLabs Inc, Bellevue, Wash). The principles of this technique have been described elsewhere in more detail,14,15 and the device was programmed to automatically record systolic BP (SBP) and DBP values with the cuff placed on the nondominant arm. The monitor was programmed to measure the BP at 20-minute intervals between 6 AM and 11 PM and at 30-minute intervals between 11 PM and 6 AM. This schedule was chosen to reduce the number of measurements during sleep but to ensure adequate observations during the day when the hearing loss occurs more commonly.

The data were analyzed with the Statistical Package for the Social Sciences, version 7.1 (SPSS Inc, Chicago, Ill), and the results are expressed as mean±SD. Hourly BP values were obtained from each patient, and the results were averaged to achieve a final BP profile. Separated average values were computed for daytime (6 AM-11 PM) and nighttime (11 PM-6 AM). The main statistical analysis was the comparison between clinical and 24-hour SBP and DBP values between the 2 groups of patients. Two-way analysis of variance was used to compare the results of 24-hour BP monitoring in the 2 groups of patients. The t test was used to compare the baseline clinical characteristics of the populations.

The baseline characteristics of the 2 populations of patients are reported in Table 1. Average clinic and ambulatory BP values were significantly lower in the population with SSHL when compared with controls for SBP and DBP values. Conversely, no significant differences were observed between the 2 groups of patients for the other demographic variables or risk factors for cardiovascular disease.

Within the population of patients with SSHL, the average 24-hour daytime and nighttime SBP and DBP values were slightly reduced in the female subgroup (Table 2), and the difference reached formal statistical significance for SBP values. Conversely, the extent of the absolute decrease in SBP and DBP values between daytime and nighttime is comparable in both sexes for SBP (−10.6±4.0 mm Hg in females vs −10.3±4.0 mm Hg in males) and DBP (−11.1±5.0 mm Hg in females vs −11.2±4.0 mm Hg in males) values and largely comparable with that observed in the control population for SBP and DBP (SBP of −11.2±4.0 and DBP of −11.5±5.0 mm Hg in females vs SBP of −11.6±4.0 and DBP of −11.4±3.0 in males [P=.44]), despite a significant difference in baseline absolute BP values (SBP, P=.004; DBP, P=.03). This suggests the possibility that patients with SSHL are at great risk of developing an impairment of the perfusion of the ear vascular bed that could contribute to the development of abnormalities of the cochlear function. To investigate the hypothesis that a relatively short period of reduced perfusion of the inner ear could have been responsible for transient or permanent cochlear damage, we analyzed the individual 24-hour BP profiles with the aim of identifying those subjects with persistent hypotension, defined by the presence of more than 2 consecutive recordings of SBP of 105 mm Hg or less and/or of DBP of 60 mm Hg or less. This cutoff value for hypotension has been calculated by subtracting 1 SD from the average value of nighttime BP recordings in the entire study population. The proportion of patients who complied with such criteria was 70% (16/23) during the daytime and 87%
Among the possible pathophysiological mechanisms that could be responsible for the development of SSHL, most of the available studies have emphasized the possible causative role of abnormalities in the cochlear circulation, whose threshold level for functional damage in response to BP changes is still largely unknown. These findings could have some implications in general practice, particularly for the clinical and pharmacological approaches to the problem of SSHL. In particular, patients complaining of SSHL should be better examined in terms of BP profile because any condition leading to a further BP decrease (even if mostly transitory) could jeopardize the possibility of even a partial recovery of the cochlear function. Furthermore, the involvement of systemic hypotension in the pathophysiological features of SSHL should influence the use of drugs during the early phases of the disease, suggesting, for instance, a limited use of drugs with vasodilatory activity that could further impair the inner ear perfusion.
was published by Ross et al., who examined a population of patients with SSHL of “idiopathic” origin, but they did not consider the age of the patients and the presence of concomitant risk factors for cardiovascular diseases. In this study, in which only 24-hour SBP values were considered, the occurrence of hypotension was arbitrarily defined by the presence of a single value of SBP lower than 90 mm Hg and/or by the detection of a value of SBP lower than 100 mm Hg in at least 10% of the recordings. According to these criteria, hypotension was observed in 25 (31%) of the 81 patients with SSHL, and this proportion was significantly lower that that observed in the present study (79%). The differences observed between the 2 studies can be reasonably explained by the less conservative criteria used in our study to define hypotension. The decision to select a higher cutoff level of BP to define hypotension was based on the assumption that this could enable us to definitely understand the role of even mild systemic hypotension in the development of SSHL.

Lehnhardt and Hesch suggested that the effects of hypotension on cochlear function might not uniformly apply to the whole range of hearing frequencies. They reported a stronger relationship between systemic hypotension and sensorineural hearing loss affecting lower frequencies. This observation was recently confirmed, showing a selective loss of lower hearing frequencies in 20 young (aged <50 years) asymptomatic hypotensive subjects, free of cardiovascular complications and enrolled among the population of a large epidemiological survey, the Brisighella Heart Study. In this study, the presence of hypotension (casual SBP of <105 mm Hg and/or DBP of <60 mm Hg) was associated with a 35% prevalence of sensorineural hearing loss involving low frequencies, and this proportion was significantly higher than that observed in a control, age-matched, normotensive population (3%) (P<.001). These data seem to confirm the relationship between systemic hypotension and sensorineural hearing loss, and suggest that the phenomenon can be more evident for low frequencies, thereby providing more support for the clinical approach to the disease.

As far as the mechanistic hypothesis linking hypotension to the loss of cochlear function is concerned, the present data are sufficiently in agreement with those published by Maass in an animal model of hemorrhagic hypotension. In this experimental setting, Maass showed a decrease in intracochlear Po2 proportional to the extent of hypotension and associated with a marked precapillary vasocostriction caused by an increase in sympathetic tone. These observations, together with those published by Hultcrantz et al in the same field, clearly suggest a dependence of the inner ear circulation on the modifications in sympathetic tone that can be associated with a decrease in systemic BP. The effects of hypotension can also be enhanced by the reduced ability for autoregulation of the inner ear circulation compared with cerebral blood flow. As reported before, a similar pathophysiological mechanism has been hypothesized in most patients with Meniere disease who have a combination of systemic hypotension and a history of stress: this could elicit a situation of exaggerated sympathetic activation involving the inner ear circulation. This is why responders to glycerol infusion were included in this series; on the other hand, an osmotic mechanism could feasibly be responsible for peripheral vasoconstriction.

Taken together, all this evidence could have some important practical clinical implications. First, patients presenting with SSHL should be examined for a history of hypotension and undergo a careful BP evaluation during the early phase of the disease. Second, in patients who prove to be hypotensive, the use of vasoactive drugs could have some negative effects and promote relapses that have been shown to be rather common, and could probably be related to a condition of hemodynamic imbalance at the level of the inner ear circulation. Finally, patients who have had SSHL should adopt any available preventive measure to avoid further potential damage. In particular, we suggest that 24-hour ambulatory BP monitoring be carried out to identify the presence of significant hypotensive episodes that could contribute to provide a reliable explanation for the occurrence of the disease.

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