fees in academic institutions are approximately $600 to $700 per session, including both technical and professional fees. Typical treatments have consisted of 5 to 10 sessions.

Given the small number of patients in the trials reviewed, methodological shortcomings, and poor reporting, the reported findings of benefit should be interpreted cautiously. The substantial cost, the potential adverse effects (including barotrauma), a question of the clinical significance of reported benefits, and the confounding effect of cointerventions (steroids, antivirals, rheologic agents) make it difficult to weigh benefits and harms. The evidence supports possible benefit of HBOT as an adjuvant treatment in cases of acute SSNHL when used within 3 months of the onset of the hearing loss, with potentially more benefit noted in cases of severe to profound loss.

STATEMENT 10. OTHER PHARMACOLOGIC THERAPY: C[illegible] ISSNHL. [illegible] C[illegible].

Action Statement Profile for Statement 10

- Aggregate evidence quality: Grade B
- Benefit: Avoidance of unnecessary treatment, avoid adverse events of unnecessary treatment, cost saving
- Risk, harm, cost: None as the recommendation is against the use of these therapies
- Benefit-harm assessment: Preponderance of benefit
- Value judgments: None
- Intentional vagueness: The word [illegible] is used to avoid setting a legal standard, recognizing that patient-specific indications for 1 or more of these therapies may be reasonable to try on an individualized basis, with shared decision making
- Role of patient preferences: None
- Exclusions: None
- Policy level: Recommendation against

Supporting Text

The purpose of this statement is to discourage clinicians from using pharmacologic agents that have potential side effects and no documented efficacy, despite the fact that many of these agents are advocated.

One of the proposed etiologies of SSNHL is inflammation caused by a viral infection. Proposed mechanisms of action include direct viral invasion of the cochlea or cochlear nerve, reactivation of a latent virus within the spiral ganglion, and immune-mediated mechanisms once an infection becomes systemic.[178] Theoretically, initiation of antiviral agents may be valuable for aiding in the recovery of hearing. Because direct sampling of inner ear fluids is impractical and potentially harmful to the patient, hematologic serologic testing is the only avenue for viral testing.

Multiple trials have been carried out and failed to find any benefit of the addition of antiviral therapies. Conlin and Parnes performed both a systematic review[7] and meta-analysis[6] of treatments for SSNHL and found only 4 RCTs[179-182] comparing antiviral therapy and steroid therapy vs placebo and steroid therapy. None of the studies reported statistically significant results. In addition, antiviral agent use is not without consequences, and reported side effects include nausea, vomiting, photosensitivity, and, rarely, reversible neurologic reactions, including mental status changes, dizziness, and seizures.

Another proposed etiology of SSNHL is cochlear ischemia. Because the blood supply to the inner ear has no collateral circulation, it is tenuous at best. As with most vascular disorders, hemorrhage, embolism, and vasospasm may affect the inner ear negatively and cause damage, resulting in SSNHL. Fisch et al[183] demonstrated a 30% reduction of perilymphatic oxygen tension in patients with SSNHL and demonstrated that treatment with carbogen resulted in a mean increase in perilymph oxygen tension of 175%. Hypercoagulability has also been seen in blood samples of patients with SSNHL. There is contradictory histopathological and clinical evidence against the vascular theory of SSNHL.[12,184-186] Most patients with SSNHL probably do not have a solely ischemic etiology, which is difficult to disprove based on clinical features and testing.

Vasoactive agents have been tried to enhance cochlear blood flow. Prostaglandin E_1 has shown benefit as a vasodilator and an inhibitor of platelet aggregation. Naftidrofuryl acts to dilate vessels by antagonizing the effect of serotonin and thromboxane A2. Calcium antagonists act to dilate vessels by antagonizing contraction of the smooth muscle cells in the vessel walls. [illegible] extract contains flavones and terpenes, which prevent the development of free radicals in cases of ischemic-related metabolic disturbances and thus counteract secondary vessel contraction. Antihypoxidotic and antiedematous effects, as well as platelet-activating factor (PAF)–antagonistic properties, have been described. Pentoxifylline increases the flexibility of erythrocytes and leukocytes and thus improves blood viscosity, particularly in the capillaries. In addition, pentoxifylline also inhibits platelet aggregation by means of prostaglandin synergy. Dextran may improve microcirculation due to an antithrombotic effect. Hydroxyethyl starch carrier solution reduces the hematocrit level and platelet aggregation.[187,188]

These therapies have considerable side effects. Different types of treatment entail different risks. The clinician should be aware of these potential adverse drug events, including allergic reactions, bleeding, hypotension, arrhythmias, seizures, circulatory collapse, and drug interactions.

The use of vasodilators and vasoactive substances for ISSNHL was reviewed by the Cochrane Collaborative in 2009.[189] Only 3 RCT studies were worthy of evaluation. All 3 of these were considered to have a high risk of bias because their overall methodology was poor and sample sizes were small. The reviewers noted differences in the type, dosage, and duration of vasodilator treatment used in each of these studies. Because of the degree of heterogeneity in methodology and outcomes assessment, the results could not be

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