The Journal of Laryngology & Otology
October 2005, Vol. **119,** pp. 791–798

Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss and tinnitus: a systematic review of randomized controlled trials

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Abstract

Background: Idiopathic sudden sensorineural hearing loss (ISSHL) and tinnitus are common. Hyperbaric oxygen therapy (HBOT) may improve hearing loss and/or reduce the intensity of tinnitus.

Methods: We performed a systematic search of the literature for randomized controlled trials, and made pooled analyses of pre-determined clinical outcomes where possible.

Results: Six trials contributed to this review (304 subjects). Pooled analysis suggested a significantly increased chance of a 25 per cent improvement in hearing threshold on pure tone average with HBOT (relative risk (RR) 1.39, 95 per cent confidence interval (CI) 1.05–1.84, $p = 0.02$; number-needed-to-treat 5, 95 per cent CI 3–20), but not a 50 per cent increase (RR 1.53, 95 per cent CI 0.85–2.78, $p = 0.16$). The significance of any improvement in tinnitus following HBOT could not be assessed due to poor reporting.

Conclusions: HBOT improved hearing, but the clinical significance of the level of improvement is not clear. Routine application of HBOT to patients with ISSHL is not justified by this review. More research is needed.

Key words: Hyperbaric Oxygenation; Hearing Loss, Sensorineural; Tinnitus

Introduction

Idiopathic sudden sensorineural hearing loss (ISSHL) is an acute hearing impairment with an incidence of about 8–15 per 100 000 of the population per year.[1] Although the aetiology and pathophysiology remain unclear,[2] ISSHL is most commonly defined as a greater than 30 decibels (dB) sensorineural hearing loss occurring in at least three contiguous audiometric frequencies over 72 hours or less.[3] Tinnitus may be defined as the perception of sound in the absence of external acoustic stimulation. The incidence is probably around 10–20 per cent of adults in the developed countries.[4,5] Brief episodes of tinnitus are probably normal, and clinically significant tinnitus is usually defined by applying one of several proposed classification systems.[6,7]

Because of the abrupt onset in many patients, a vascular cause for ISSHL has been suggested,[8] but other possibilities include viral infection, autoimmune disease and inner ear membrane rupture.[9,10] The cause of tinnitus is equally obscure, although it is often associated with ISSHL; up to 90 per cent of patients suffering from ISSHL also complain of tinnitus.[11] The most widely discussed theories include excessive or abnormal spontaneous activity in the auditory system and related cerebral areas[12] and abnormal signal processing with 'feedback'.[13,14]

Treatments for ISSHL, often aimed at improving the oxygenation of the inner ear, include vasodilators, plasma expanders, steroids, anticoagulants, diuretics and antivirals. None have been proven of benefit in large randomized trials or meta-analyses, although a Cochrane review is underway of the use of vasodilators for ISSHL.[15] Assessment of the effectiveness of therapy is complicated by a high rate of spontaneous recovery, as much as 65 per cent in some studies,[16] and the very variable periods for which hearing loss has been present before the institution of therapy. Specific therapies for tinnitus have tended to focus on the impact of the noise on quality of life and mood, and include antidepressants, anticonvulsants and benzodiazepines, or on trying to mask the noise itself with white-noise generators. A variety of psychotherapeutic and 'habituation' programs are also advocated to help the sufferer deal with the problem.[17] A Cochrane review of antidepressants for tinnitus is underway.[18]

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Accepted for publication: 5 May 2005.

Hyperbaric oxygen therapy (HBOT) is a further, usually adjunctive, therapy that has been proposed to improve both ISSHL and tinnitus. This is the therapeutic administration of oxygen at environmental pressures greater than one atmosphere absolute (ATA). Administration involves placing the patient in an airtight vessel, increasing the pressure within that vessel, and administering 100 per cent oxygen for respiration. In this way, it is possible to deliver a greatly increased partial pressure of oxygen to the tissues. Typically, treatments involve pressurization to between 1.5 and 3.0 ATA for periods of between 60 and 120 minutes once or twice daily. A typical course will involve 20–40 such treatments.

HBOT was first reported to improve the outcome following ISSHL and tinnitus in the late 1960s by both French and German workers.[19] The administration of hyperbaric oxygen is based on the argument that both hearing loss and tinnitus may result from a hypoxic event in the cochlear apparatus, and that hyperbaric oxygen therapy may be able to reverse that oxygen deficit.[19] Despite more than 30 years of interest in the application of hyperbaric oxygen therapy to these patients, however, little clinical evidence exists for the assertion that such an intervention improves outcome. The purpose of this review is to assess the randomized clinical evidence for the benefit of HBOT in the treatment of both acute and chronic sensorineural hearing loss and/or tinnitus.

This paper is based on a Cochrane review first published in The Cochrane Library 2005, Issue 2, Chichester, UK: John Wiley & Sons Ltd (www.thecochranelibrary.com). Cochrane reviews are regularly updated as new evidence emerges and in response to comments and criticisms. The Cochrane Library should be consulted for the most recent version of the review.

Methods

It was our intention to identify and review all randomized controlled trials (RCTs) concerning the treatment with HBOT of any patient with ISSHL and/or tinnitus. We included all trials using hyperbaric oxygen administered in a compression chamber above 1.2 ATA and for treatment times between 30 and 120 minutes on at least one occasion. For the comparator therapy, we accepted any standard treatment regimen designed to maximize hearing loss recovery or reduction in tinnitus, or where the comparator was designed to improve quality of life for appropriate patients. Subgroup analysis was considered to evaluate the impact of different comparator strategies.

Specific search strategies were developed to identify eligible reports from database inception to December 2004 in MEDLINE, EMBASE, the Cochrane Central Register of Controlled Trials (CENTRAL) and the Database of Randomised Controlled Trials in Hyperbaric Medicine (DORCTIHM). The latter is a specifically targeted database of clinical evidence in the field (http://www.hboevidence.com).

Medical subject headings (MeSH) and main key words used were 'hyperbaric oxygenation', 'hearing loss, sensorineural', 'hearing loss, sudden' and 'tinnitus', with variants of the main key words and free text terms also applied. No restrictions to language were made. Relevant hyperbaric textbooks, journals and conference proceedings were hand searched. Experts in the field were contacted for published, unpublished and ongoing RCTs. Additional trials were identified from the citations within obtained papers.

We pre-determined the following clinically important outcomes for assessment, and all included studies must have reported at least one of these: pure tone average (PTA) audiometric response to therapy, subjective tinnitus score, activities of daily living, improvements in depression or other mood disturbance, or hearing handicap inventory. Any reported adverse events of HBOT were also recorded.

Each reviewer independently assessed the electronic search results and selected potentially relevant studies. Disagreements were settled by examination of the full paper and consensus. To assess methodological quality and detect potential sources of bias we applied the quality scale of Jadad *et al*.[20] (see Table I). We also recorded the adequacy of allocation concealment. If any relevant data were missing from trial reports, we attempted to contact the authors. To allow an intention-to-treat analysis we extracted the data reflecting the original allocation group where possible. Disagreements were again settled by consensus.

Statistical analysis

Following agreement, the data were entered into Review Manager® 4.2.1 (Cochrane Collaboration,

TABLE I

SUMMARY OF JADAD SCORE

Criteria	Description
Randomization	The study is described as randomized, including using words such as 'random', 'randomized' or 'randomly'
Addition	The method of randomization is described and appropriate (e.g. use of random-number table)
Deduction	The method of randomization is described and is inappropriate (e.g. use of birth date)
Double-blinding	The study is described as double-blind
Addition	The method of double-blinding is described and appropriate (e.g. use of placebo or sham therapy)
Deduction	The method of double-blinding is described and is inappropriate (e.g. use of observably different placebo)
Description of withdrawals	There is a description of any dropouts or withdrawals during the course of the study

From Jadad *et al*.[20]
Each criteria scores or deducts one point if satisfied, giving a quality score from 0 to 5.

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Oxford, UK). For dichotomous outcomes such as the proportion of subjects with a greater than 20 dB improvement in hearing, we calculated the relative risk (RR) with a 95 per cent confidence interval (CI). A statistically significant difference from control was assumed when the 95 per cent CI of the RR did not include the value 1.0. For continuous outcomes, such as the mean change in PTA for each group, we calculated the weighted mean difference (WMD) between groups with 95 per cent CI. We used a fixed-effects model where problematic heterogeneity between the studies was not likely and a random-effects model where such heterogeneity was likely. Heterogeneity was deemed problematic if the I^2 analysis suggested more than 30 per cent of the variability in an analysis was due to systematic differences between trials rather than chance alone.[21] Consideration was then given to the appropriateness of pooling and meta-analysis. Number-needed-to-treat (NNT) with 95 per cent CI was calculated when the relative risk estimates were statistically significant.

We planned sensitivity analyses for missing data using best-case and worst-case scenarios for imputing outcome. We also considered subgroup analysis based on the time between onset of the problem and institution of therapy (acute versus chronic), aetiology, oxygen dose (pressure, time and number of treatments), the nature of comparator therapy, and severity of hearing loss.

Results

The included studies

The initial search in July 2004 yielded 68 articles of which 15 were considered to be suitable randomized human trials dealing with the treatment of ISSHL and/or tinnitus with HBOT. Appraisal of the full report of these papers led to the exclusion of 10 publications because they were reviews without new data,[22-24] comparative trials where all groups received HBOT,[25-27] not randomly allocated,[28,29] or were only case series[30,31]. Five publications therefore initially met our inclusion criteria.[32-36] Following a second search conducted in December 2004 prior to submission of the present paper, one further RCT was included.[37] Five of these studies included patients with acute presentation of ISSHL with or without tinnitus, while one enrolled

TABLE II

CHARACTERISTICS OF INCLUDED STUDIES

Study	Methods	Participants	Interventions	Outcomes
Cavallazzi *et al.* (1996)[34]	Method of allocation not clear, no blinding; Jadad score 0	64 subjects (30 control, 34 HBOT) with ISSHL, time course unknown; stratified into mild, moderate, severe and 'deep'	Control: multiple drug therapy (heparin, betamethasone, nicotinic acid, flunarizine, citidinephosphocoline, dextran, vitamins, neurotropic and antiviral drugs) HBOT: as above plus oxygen at 2.5 ATA for 60 mins daily for 15 sessions over three weeks	PTA recovery (%)
Fattori *et al.* (2001)[32]	Method of randomization not clear, no blinding; Jadad score 2	50 subjects (20 control, 30 HBOT) with ISSHL referred within 48 hours; stratified into mild, moderate and severe	Control: vasodilator therapy: 10-day course i.v. 200 mg/day buflomedil HBOT: oxygen at 2.2 ATA for 90 mins daily for 10 days	PTA recovery (%) Mean PTA recovery (%)
Hoffmann *et al.* (1995a)[35]	Method of randomization not clear, patients and outcome assessors assessors blinded; Jadad score 3	44 subjects (22 control, 22 HBOT) with ISSHL for longer than six months	Control: air breathing at 1.5 ATA for 45 mins daily, five days each week for three weeks HBOT: 100% oxygen on the same schedule as controls	Improved hearing (%) Tinnitus (%)
Hoffmann *et al.* (1995b)[36]	Method of randomization not clear, no blinding; Jadad score 2	20 subjects (10 control, 10 HBOT) with ISSHL not improved after 14 days of pharmacological treatment with hydroxyethyl starch, pentoxifylline and cortisone	Control: no treatment HBOT: oxygen at 1.5 ATA for 45 mins daily, five days each week for two to four weeks (10–20 sessions)	Mean PTA recovery (dB) Tinnitus (%)
Schwab *et al.* (1998)[33]	Method of randomization not clear, no blinding; Jadad score 2	75 subjects (38 control, 37 HBOT) with ISSHL seen within two weeks and without any prior therapy	Control: no treatment HBOT: oxygen at 1.5 ATA for 45 mins daily, five days each week for two to four weeks (10–20 sessions)	Mean PTA recovery (dB) Tinnitus (0-10)
Topuz *et al.* (2004)[37]	Method of randomization not clear, no blinding; Jadad score 2	51 subjects (21 control, 30 HBOT) with ISSHL seen within two weeks and without any prior therapy	Control: multiple drug therapy: prednisone (1 mg/kg/day/2 weeks), rheomacrodex (500 ml/day/5 days), diazepam (5 mg bd) and pentoxiphylline (200 mg bd) HBOT: as above plus oxygen at 2.5 ATA for 90 mins, 25 treatments in three weeks	Mean PTA recovery (dB)

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subjects with at least six months' history of ISSHL and/or tinnitus.[35] The total number of patients enrolled was 304, with 163 receiving HBOT, and 141 control.

Inclusion criteria varied between the five studies dealing with acute presentation. Hoffmann *et al.*[36] accepted only patients who had not improved after two weeks of pharmacological therapy, Fattori *et al.*[32] accepted patients untreated within 48 hours of hearing loss, while Schwab *et al.* and Topuz *et al.*[33,37] accepted patients up to two weeks after loss. Cavallazzi *et al.*[34] did not define entry criteria. Treatment pressure (1.5–2.5 ATA), time schedule (45–90 min), and number of sessions (10–25) of HBOT differed somewhat between studies. Similarly, there was some variation in comparator therapies. Three studies compared HBOT to a polypharmaceutical approach,[33,34,37] one to a vasodilator alone,[32] and one to no specific therapy.[36] Follow-up periods were generally short, with the longest being only three months following completion of therapy.[33,36]

Study quality was generally assessed as low and was not used as a basis for sensitivity analysis. No study described the method of randomization or clearly concealed allocation from the individual responsible for randomization. Only one study employed a sham therapy,[35] and that was the single study dealing with chronic presentation. The individual study characteristics are summarized in Table II.

Clinical outcomes

Statistical pooling was not possible for the majority of pre-planned outcome measures due to lack of suitable data. Problems included the small number of studies, modest number of patients, and the variability in outcome measures employed. The data are summarized in Table III.

Improvement in hearing with ISSHL (acute presentation). Proportion of subjects with more than 50 per cent return of hearing loss (Figure 1): two trials reported this outcome immediately following the course of therapy[32,34] and 114 subjects were involved. Thirty-five subjects (55 per cent) improved in the HBOT group versus 18 (36 per cent) in the control group. Pooled analysis suggests there is no significant difference between groups (RR with HBOT: 1.53, 95 per cent CI 0.85–2.78, $p = 0.16$). There was evidence of moderate heterogeneity between these studies ($I^2 = 38$ per cent), and this result is achieved with a random-effects model.

Proportion of subjects with more than 25 per cent return of hearing loss (Figure 1): the same two trials reported this outcome at the end of therapy.[32,34] There was a statistically significant increase in the proportion of subjects showing an improvement in PTA-assessed hearing loss over four frequencies following HBOT (RR 1.39, 95 per cent CI 1.05–1.84, $p = 0.02$). Subgroup analysis did not suggest a different response for different grades of severity on enrolment. The absolute risk difference of 22 per cent (78 versus 56 per cent) represents an NNT to achieve one extra good outcome of 5 (95 per cent CI 3–20).

Mean improvement in PTA as a percentage of baseline: this outcome was reported in only one trial.[32] There was a mean improvement in PTA of 61 per cent with the application of HBOT versus an improvement of 24 per cent in control subjects, and this difference was statistically significant (WMD 37 per cent in favour of HBOT, 95 per cent CI 22–53).

Mean improvement in hearing over all frequencies (dB): three trials reported on this outcome[33,36,37] involving 128 subjects (42 per cent of the total). All three reported greater mean improvement with HBOT, but only Topuz *et al.*[37] reported standard deviations, and so the other two studies could not contribute to this analysis. Topuz stratified the results by severity on entry, and analysis suggested that there was significantly improved return of hearing with HBOT for severe hearing loss (WMD in hearing gain 37.7 dB, 95 per cent CI 22.9–52.5, $p < 0.0001$) and

TABLE III

SUMMARY OF POOLED OUTCOMES

Outcome	Studies	HBOT /control (n)	Efficacy data with 95% CI, p-value and NNT
Acute presentation			
>50% return in hearing (proportion by PTA)	Cavallazzi *et al.* (1996)[34] Fattori *et al.* (2001)[32]	64/50	RR 1.53, 95% CI 0.85–2.78, $p = 0.16$
>25% return in hearing (proportion by PTA)	Cavallazzi *et al.* (1996)[34] Fattori *et al.* (2001)[32]	64/50	*RR 1.39, 95% CI 1.05–1.84, $p = 0.02$ NNT 5, 95% CI 3–20
Mean improvement in PTA (%)	Fattori *et al.* (2001)[32]	30/20	*WMD: 37.3, 95% CI 21.75–52.85, $p < 0.0001$
Mean hearing improvement (dB)	Hoffmann *et al.* (1995b)[36] Schwab *et al.* (1998)[33] Topuz *et al.* (2004)[37]	78/68	*WMD (severe loss): 37.7, 95% CI 22.9–52.5, $p < 0.0001$ *WMD (moderate): 19.3 dB, 95% CI 5.2–33.4, $p = 0.007$ WMD 0.2 (mild): 95% CI-10.0–10.4, $p = 0.97$
Mean improvement in tinnitus score (0–10)	Schwab *et al.* (1998)[33] Hoffmann *et al.* (1995b)[36]	36/37	Improved 3.1 and 4.0 units more in HBOT, respectively
Chronic presentation			
Some improvement in hearing (proportion)	Hoffmann *et al.* (1995a)[35]	22/22	RR: 0.64, 95% CI 0.30 to 1.33, $p = 0.23$
Some improvement in tinnitus (proportion)	Hoffmann *et al.* (1995a)[35]	22/22	RR: 0.44, 95% CI 0.16 to 1.23, $p = 0.12$

WMD = weighted mean difference; RR = relative risk; NNT = number-needed-to-treat. *Significant outcomes (statistical difference is assumed if the 95% CI does not include the value 1.0).

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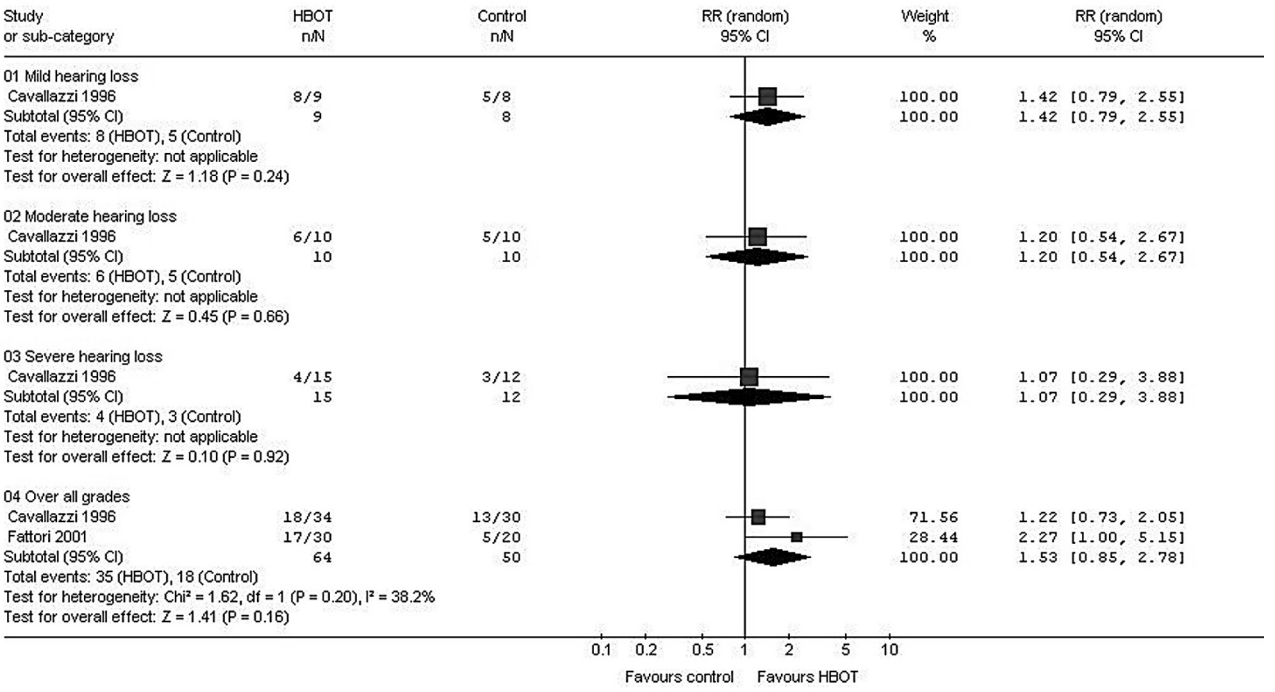

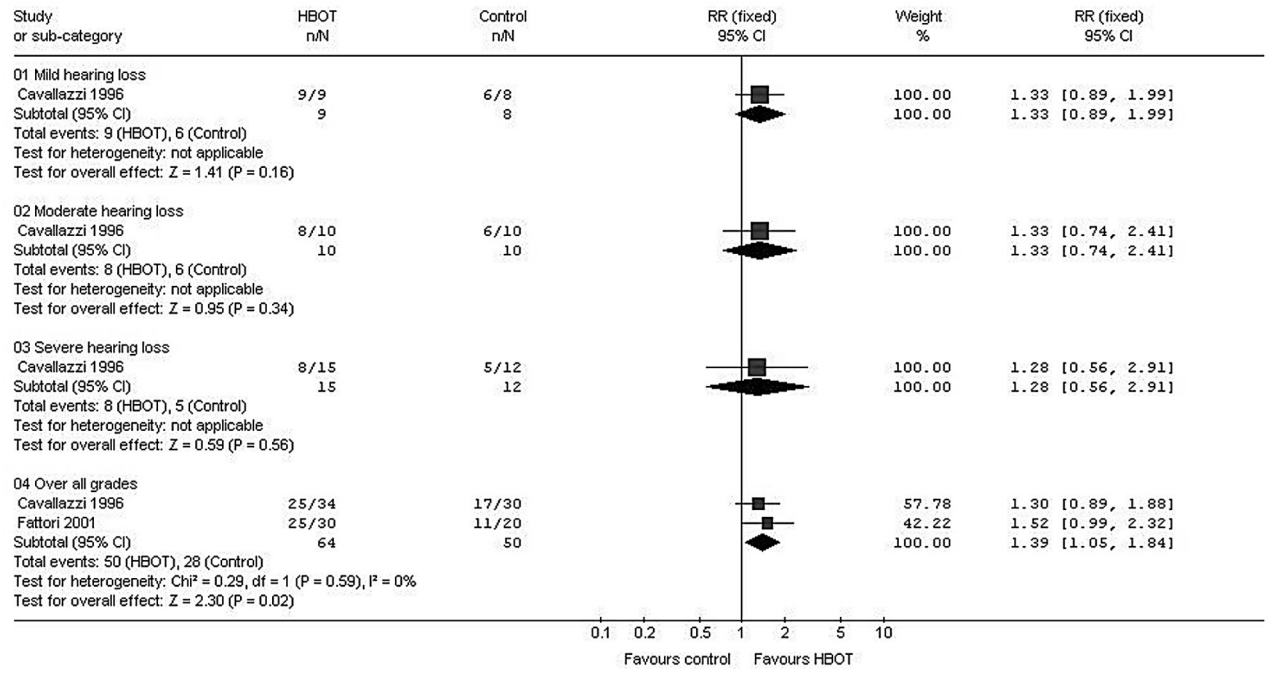

FIG. 1

Forest plot of treatment effect for acute presentation of ISSHL. Proportion of subjects attaining 50 per cent (top plot) and 25 per cent (bottom plot) improvement in PTA hearing loss at the completion of therapy with subgroup analysis by severity grade on enrolment.

moderate loss (WMD 19.3 dB, 95 per cent CI 5.2–33.4, $p = 0.007$), but no difference between groups for mild loss (WMD 0.2 dB, 95 per cent CI -10.0–10.4, $p = 0.97$). *Improvement in tinnitus (acute presentation)*. Two trials reported on this outcome[33,36] and enrolled 53 subjects. While these trials reported a greater mean improvement in tinnitus (using a visual analogue scale between 0 and 10) in the HBOT arm than the control (3.1 and 0.4 units, respectively), neither trial reported standard deviation around those means, making pooled analysis impossible.

Chronic presentation. The single trial that enrolled patients with a chronic presentation[35] did not suggest any statistically significant differences in recovery of hearing (RR for improvement with HBOT 0.64, 95 per cent CI 0.30–1.33, $p = 0.23$) or tinnitus (RR for improvement with HBOT 0.44, 95 per cent CI 0.16–1.23, $p = 0.12$).

No trials reported any outcomes related to activities of daily living, improvements in mood disturbance, hearing handicap inventory or adverse events of therapy in either arm.

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Discussion

This review includes data from six trials, and we believe these represent all randomized human trials in this area, both published and unpublished, at the time of searching the databases. We found limited evidence that HBOT improves hearing when applied as an early treatment in ISSHL. There was some indication from the analysis of pooled data from two trials[32,34] that HBOT increases the proportion of patients gaining more than a 25 per cent improvement in hearing, while one of those trials also suggested there was a greater mean improvement in PTA as a percentage of baseline following HBOT.[32] Three trials also suggested improvements in mean hearing measured in decibels following HBOT.[33,36,37] We found no evidence from the single relevant trial that HBOT was useful in those individuals with long-standing hearing loss or tinnitus of unknown aetiology.

Only six trials with 304 participants were available for evaluation using our planned comparisons, and meta-analysis was not appropriate or possible for a number of these. Other problems for this review were the poor methodological quality of many of these trials, variability and poor reporting of entry criteria, the variable nature and timing of outcomes, and poor reporting of both outcomes and methodology. In particular, given the high rate of spontaneous recovery from ISSHL, there is a possibility of bias due to different times to entry in these small trials, as well as from non-blinded management decisions in all trials.

These trials were published over a nine-year period up to 2004, and from a wide geographical area. We had planned to perform subgroup analyses with respect to the time between onset and therapy, the putative aetiology of the ISSHL or tinnitus, the dose of oxygen received (pressure, time and length of treatment course), and the nature of the comparative treatment modalities. None of these analyses were appropriate in the small number of pooled analyses. The one trial that enrolled subjects who had failed to respond to two weeks of intensive multiple pharmacotherapy did not contribute to any pooled analysis.[36] Response rates stratified by severity of hearing loss were only reported by Cavallazzi *et al*. and Topuz *et al*.,[34,37] and these suggest trends to greater treatment effect in opposite directions. Patient inclusion criteria were not standard, and poorly reported in some trials. No standard severity scale was employed across these trials, and the time to entry varied from within 48 hours[32] to two weeks.[33,36,37]

Pooled data for clinical outcomes of interest could only be performed with respect to the proportion of patients showing an audiometric improvement in hearing of 50 per cent or 25 per cent from baseline to the end of therapy. While the chance of a 50 per cent improvement was not significantly increased following HBOT, the chance of a 25 per cent improvement in hearing was ($p = 0.02$). This analysis suggests that we would need to treat five patients with HBOT in order to improve one extra person's hearing by 25 per cent (95 per cent CI 3–20) than if we used the control therapy. Given the small number of subjects and generally poor quality of these trials, this result needs to be interpreted with caution. Further, the clinical significance of a 25 per cent improvement in hearing from baseline is not clear, and will depend greatly on the starting level of impairment. No trial in this review has estimated any functional improvement.

Two trials reported on improvements in tinnitus for patients with an early presentation.[33,36] While both reported improvement in mean visual analogue scores for patients receiving HBOT, neither group of authors reported standard deviations around the mean and the significance of these changes is not clear. There was no suggestion that HBOT had a positive influence on chronic presentation of tinnitus in the single trial that reported this outcome.[35]

None of these trials systematically reported adverse effects with HBOT or control therapies, so we are unable to assess any negative impact of HBOT on the outcome of these patients. HBOT is regarded as a relatively benign intervention. There are few major adverse effects (pulmonary barotrauma, drug reactions, injuries or death related to chamber fire). There are a number of more minor complications that may occur commonly. Visual disturbance, usually reduction in visual acuity secondary to conformational changes in the lens, is very commonly reported; perhaps as many as 50 per cent of those having a course of 30 treatments.[38] While the great majority of patients recover spontaneously over a period of days to weeks, a small proportion of patients continue to require correction to restore sight to pre-treatment levels. The second most common adverse effect associated with HBOT is barotrauma, usually affecting the middle ear, although other sites include the respiratory sinuses and dental cavities. Most episodes of barotrauma do not require the therapy to be abandoned. Less commonly, perhaps once every 5000 treatments, HBOT may be associated with acute neurological toxicity manifesting as seizure.[39]

While we have made every effort to locate further unpublished data, it remains possible that this review is subject to a positive publication bias, with generally favourable trials more likely to achieve reporting. With regard to long-term outcomes following HBOT and any effect on the quality of life for these patients, we have located no relevant data.

We conclude that there is limited evidence that HBOT improves hearing in patients with ISSHL who present within two weeks of hearing loss, and some indication that HBOT might improve tinnitus presenting in the same time frame. However, there is no evidence that any improvement is functionally important. Thus, the routine use of HBOT in these patients cannot be justified by this review. The small number of studies, the modest numbers of patients, and the methodological and reporting inadequacies of the primary studies included in this review demand a cautious interpretation. Given the findings of improved hearing with the use of HBOT in these

https://doi.org/10.1258/002221505774481246 Published online by Cambridge University Press

patients, there is a case for large randomized trials of high methodological rigour in order to define the true extent of the benefit (if any) from administration of HBOT. Specifically, more information is required on the subset of disease severity and time of presentation most likely to be associated with a benefit from this therapy, the effect of differing oxygen dosage, and effect of other therapies administered simultaneously. Attention should be paid to the use of appropriate clinical outcomes designed to measure functional importance.

- **ISSHL and tinnitus are common problems with significant health impact on individuals**
- **The aetiology of these problems is not clear, but ISSHL may be associated with hypoxia**
- **No clearly effective therapy has been demonstrated**
- **In some centres, HBOT is routinely used for the treatment of ISSHL and tinnitus with claims of effectiveness**
- **HBOT is associated with an improvement in hearing loss of unknown clinical significance when compared to controls**
- **There is a case for methodologically sound randomized trials to confirm and quantify these findings**
- **HBOT does not seem to be effective for chronic presentations of ISSHL and tinnitus**

Acknowledgements

We acknowledge the assistance of the Cochrane Advanced Reviewer Support Service provided by the Australasian Cochrane Centre in undertaking literature location and reference management. We would also like to thank Jenny Bellorini and the editors of the Cochrane Ear Nose and Throat Group for their invaluable advice and guidance.

The results of the Cochrane review can be interpreted differently, depending on people's perspectives and circumstances. Please consider the conclusions presented carefully. They are the opinions of review authors, and are not necessarily shared by The Cochrane Collaboration.

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Dr M Bennett takes responsibility for the integrity of the content of the paper.
Competing interests: None declared