Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and tinnitus

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A substantive amendment to this systematic review was last made on 06 September 2004. Cochrane reviews are regularly checked and updated if necessary.

Abstract

Background: Idiopathic sudden sensorineural hearing loss (ISSHL) with or without tinnitus is common and presents a health problem with significant effect on quality of life. Hyperbaric oxygen therapy (HBOT) may improve oxygen supply to the inner ear and thereby result in an improvement in hearing and/or a reduction in the intensity of tinnitus.

Objective: To assess the benefits and harms of HBOT for treating ISSHL and tinnitus.

Search strategy: We searched the Cochrane ENT Specialist Register (June 2004), CENTRAL (The Cochrane Library Issue 3, 2004), MEDLINE (1966 to 2004), EMBASE (1974 to 2004), CINAHL (1982 to 2004), DORCTHIM (1996 to 2004), and reference lists of articles. Researchers in the field were contacted.

Selection criteria: Randomised studies comparing the effect on ISSHL and/or tinnitus of therapeutic regimens which include HBOT with those that exclude HBOT.

Data collection and analysis: Three reviewers independently evaluated the quality of the relevant trials using the validated Jadad 1996 Oxford-Scale and extracted the data from the included trials.

Main results: Five trials contributed to this review (254 subjects, 133 receiving HBOT and 120 control). Pooled data from two trials involving 114 patients (45% of the total) suggested there was a trend towards, but no significant increase in, the chance of a 50% increase in hearing threshold on Pure Tone Average (PTA) over four frequencies when HBOT was used (relative risk (RR) for good outcome with HBOT 1.53, 95% confidence interval (CI) 0.85 to 2.78, P = 0.16). The chance of achieving a 25% increase with HBOT was, however, statistically significant (RR 1.39, 95% CI 1.05 to 1.84, P = 0.02). Fifty-six per cent of the control subjects achieved this outcome versus 78% of the HBOT subjects, with the number-needed-to-treat (NNT) to achieve one extra good outcome being 5 (95% CI 3 to 20). A single trial involving 50 subjects (20% of the total) also suggested a significant improvement in the mean PTA threshold expressed as a percentage of baseline (61% improvement with HBOT, 24% with control, WMD 37%, 95% CI 22% to 53%).The effect of HBOT in tinnitus could not be assessed due to poor reporting.There were no significant improvements in hearing or tinnitus reported in the single study to examine the effect of HBOT on a chronic presentation (six months) of ISSHL and/or tinnitus.

Reviewers' conclusions: For people with early presentation of ISSHL, the application of HBOT significantly improved hearing loss, but the clinical significance of the level of improvement is not clear. We could not assess the effect of HBOT on tinnitus by pooled analysis. The routine application of HBOT to these patients cannot be justified from this review. In view of the modest

number of patients, methodological shortcomings and poor reporting, this result should be interpreted cautiously, and an appropriately powered trial of high methodological rigour is justified to define those patients (if any) who can be expected to derive most benefit from HBOT.There is no evidence of a beneficial effect of HBOT on chronic presentation of ISSHL and/or tinnitus.

Background

Idiopathic sudden sensorineural hearing loss (ISSHL) is an acute hearing impairment, with an incidence of about 8 to 15 per 100,000 of the population per year (Stokroos 1996). Although, the aetiology and pathophysiology remain unclear (Haberkamp 1999), ISSHL is most commonly defined as a greater than 30 dB sensorineural hearing loss occurring in at least three contiguous audiometric frequencies over 72 hours or less (Hughes 1996). Tinnitus can be described as the perception of sound in the absence of external acoustic stimulation, and in many cases it is associated with some degree of hearing loss, particularly in those individuals who have been exposed to excessive noise. The incidence is probably around 10% to 20% of adults in the developed countries (ATA 2001; Coles 1990). For the patient it may be trivial or it may become a debilitating illness (Luxon 1993). Sufferers from tinnitus hear a noise that apparently arises from the ears or within the head and may be continuous or intermittent. Brief episodes of tinnitus are probably normal, and clinically significant tinnitus is usually defined by applying one of several classification systems proposed (Dauman 1992; Stephens 1991).

Because of the abrupt onset in many patients, a vascular cause for ISSHL has been suggested (Belal 1980), but other possibilities include viral infection, autoimmune disease and inner ear membrane rupture (Thurmond 1998; Yoon 1990). The cause of tinnitus is equally obscure, although it is often associated with ISSHL - up to 90% of patients suffering from ISSHL also complain of tinnitus (Parnes 1997). The most widely discussed theories include excessive or abnormal spontaneous activity in the auditory system and in related cerebral areas (Kaltenbach 2000) and abnormal processing of a signal generated in the auditory system with 'feedback' (Jastreboff 1990). Recent work confirms that a broad multimodal network of neurons, often operating from a site remote to that of the initial pathology, is involved in generating and sustaining the tinnitus perception in some forms of the disorder (Cacace 2003). Tinnitus has, in fact, been compared to chronic pain of central origin in some regards, and when symptoms are severe, tinnitus can be associated with major depression, anxiety and other psychological disturbances, leading to a progressive deterioration of quality of life (Sullivan 1992; Sullivan 1994).

Treatments for ISSHL have mostly been designed to improve the blood circulation and oxygenation of the inner ear and include vasodilators, plasma expanders, steroids, anticoagulants, diuretics, contrast dye and antivirals. None have been proven of benefit in large randomised trials or meta-analyses, although a Cochrane review is underway in the use of vasodilators for ISSHL (Liang 2002). Assessment of the effectiveness of therapy is further complicated by a high rate of spontaneous recovery, as much as 65% in some studies (Mattox 1977), and the very variable periods for which hearing loss has been present before the institution of therapy. While the impact of therapy will vary with individual circumstances, we have selected a 50% return of hearing following therapy as a clinically significant improvement when considering appropriate power for included studies in this review. Specific therapies for tinnitus have tended to focus either 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 (Noell 2003). A Cochrane review of antidepressants for tinnitus is underway (Baldo 2001).

Hyperbaric oxygen therapy is a further, usually adjunctive, therapy that has been proposed to improve both ISSHL and tinnitus. This is the therapeutic administration of 100% 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% oxygen for respiration. In this way, it is possible to deliver a greatly increased partial pressure of oxygen to the tissues. Typically, treatments involve pressurisation to between 1.5 and 3.0 ATA for periods between 60 and 120 minutes once or twice daily. A typical course will involve 20 to 40 such treatments.

Hyperbaric oxygen therapy was first reported to improve the outcome following ISSHL and tinnitus in the late 1960s by both French and German workers (translations unavailable at present). The administration of hyperbaric oxygen is based on the argument that both hearing loss and tinnitus may result from an hypoxic event in the cochlear apparatus, and that hyperbaric oxygen therapy may be able to reverse that oxygen deficit (Lamm 1998). Despite more than 30 years of interest in the delivery of hyperbaric oxygen therapy in these patients, however, little clinical evidence exists for the assertion that such an intervention improves outcome.

Hyperbaric oxygen therapy is associated with some risk of adverse effects including damage to the ears, sinuses and lungs from the effects of pressure, temporary worsening of short-sightedness, claustrophobia and oxygen poisoning. Although serious adverse events are rare, hyperbaric oxygen therapy cannot be regarded as an entirely benign intervention.

Objectives

To assess the evidence for the benefit of hyperbaric oxygen therapy in the treatment of both acute and chronic sensorineural hearing loss and/or tinnitus. We compared treatment regimens including hyperbaric oxygen against similar regimens excluding hyperbaric oxygen. Where regimens differed significantly between studies, this was clearly stated and the implications discussed. All comparisons were made using an intention to treat analysis where possible, and they reflect efficacy in the context of randomised trials rather than true effectiveness in any particular clinical context. Specifically, we wished to address the following questions:

1. Does the administration of hyperbaric oxygen to people with idiopathic sensorineural hearing loss (whether early or late presentation) result in an increase in the proportion attaining a useful improvement in hearing? We also intended to investigate both binaural hearing recovery and speech discrimination recovery where possible.

2. Does the administration of hyperbaric oxygen to people with tinnitus (whether early or late presentation) result in an increase in the proportion experiencing a useful reduction in tinnitus?

Criteria for considering studies for this review

Types of studies

Randomised and pseudo-randomised controlled trials that compared the effect of treatment for either acute or chronic idiopathic sensorineural hearing loss and/or tinnitus where hyperbaric oxygen administration is included, with the effect of similar treatment in the absence of hyperbaric oxygen. Studies were considered irrespective of allocation concealment or blinding status.

Types of participants

Any adult with acute onset sensorineural hearing loss and/or tinnitus of any duration.

Types of intervention

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 were eligible. The comparator group was somewhat diverse. We accepted any standard treatment regimen designed to maximise 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.

Types of outcome measures

Studies were eligible for inclusion if they reported any of the following outcome measures at any time:

- Primary outcomes
- 1. Acute ISSHL: Pure tone audiometric documented change in hearing in response to treatment.
- 2. Chronic ISSHL: Pure tone audiometric documented change in hearing in response to treatment.
- 3. Acute ISSHL: Relief of tinnitus. Subjective assessment of tinnitus level.
- 4. Chronic ISSHL: Relief of tinnitus. Subjective assessment of tinnitus level.

- Secondary outcomes
- 5. Activities of daily living (ADL).
- 6. Subjective or objective improvements in depression or mood disturbance.
- 7. Hearing handicap inventory change (and similar tool for tinnitus).
- 8. Adverse events associated with hyperbaric oxygen therapy and comparators.

Search strategy for identification of studies

See: Cochrane Ear, Nose and Throat Disorders Group search strategy

It was our intention to identify both published and unpublished studies.

- Electronic searches
- Searches were performed in July 2004 for randomised controlled trials and controlled clinical trials in the following databases, in combination with the randomised controlled trial filter validated by the Cochrane Collaboration, using the strategies detailed in Table 01:

- ENT Specialist Register
- CENTRAL
- MEDLINE
- PUBMED
- EMBASE
- CINAHL
- LILACS
- AMED

MeSH terms appear in uppercase and are all exploded. Free text terms appear in lowercase. * indicates truncation.

We also searched KOREAMED, mRC, and The Database of Randomised Controlled Trials in Hyperbaric Medicine (in the latter: tinnitus OR sudden* OR sshl OR snhl OR ishl OR isshl OR issnhl OR ssnhl).

In addition we made a systematic search for relevant controlled trials in specific hyperbaric literature sources:

- 1. Experts in the field and leading hyperbaric therapy centres (as identified by personal communication and searching the Internet) were contacted and asked for additional relevant data in terms of published or unpublished randomised trials.
- 2. Relevant hyperbaric textbooks (Kindwall, Jain, Marroni, Bakker, Bennett and Elliot), journals (Undersea and Hyperbaric Medicine, Hyperbaric Medicine Review, South Pacific Underwater Medicine Society (SPUMS) Journal, European Journal of Hyperbaric Medicine and Aviation, Space and Environmental Medicine Journal) and conference proceedings (Undersea and Hyperbaric Medical Society, SPUMS, European Undersea and Baromedical Society, International Congress of Hyperbaric Medicine) published since 1980 were hand searched.
- 3. Authors of relevant studies were contacted to request details of unpublished or ongoing investigations.

All languages were considered. Authors were contacted to settle any ambiguity about the published data.

Methods of the review

- Data retrieval and management
- One reviewer (MB) was responsible for hand searching and identification of appropriate studies for consideration. Three reviewers (MB, TK and PY) examined the electronic search results and identified studies that may have been relevant and these studies were entered into a bibliographic software package (Review Manager) when any of the reviewers considered the study might satisfy the inclusion criteria. All comparative clinical trials identified by this process were retrieved with the assistance of the Cochrane Advanced Reviewer Support Service of the Australasian Cochrane Centre and reviewed independently by the three reviewers, two with content expertise in sensorineural hearing loss and tinnitus, one with content expertise in hyperbaric oxygen. In addition, one of the reviewers (MB) has expertise in clinical epidemiology. Reviewers recorded data using the data extraction form developed for this review.

Where reporting methods differed between trials for the same outcome, we attempted to contact the principal authors to request further data. Our intention was to convert reported data to a form that enabled meta-analysis. However, no suitable further data were forthcoming from any author.

- Data extraction
- Each reviewer extracted relevant data, graded the studies for methodological quality using the method of Jadad (Jadad 1996), and made a recommendation for inclusion or exclusion from the review. The method of Jadad scores trials on three criteria (randomisation, double-blinding and description of withdrawals), each of which, if present, is given a score of one. Further points are available for description of a reliable randomisation method and use of a placebo (modified for our analysis to include a sham hyperbaric oxygen session). The scores are totaled as an estimate of overall quality. Any differences were settled by consensus, and further information was sought from the authors where not stated explicitly in the report. In addition, we ranked studies on sample size and identified those with sufficient power to determine the clinically important effect for which the trial was designed. All data extracted reflected original allocation group where possible to allow an intention to treat analysis. Dropouts were identified where this information was given.

- Analyses
- For proportions (dichotomous outcomes), relative risk (RR) was used. We used a fixed-effect model where there was no evidence of significant heterogeneity between studies

(see below), and employed a random effects model when such heterogeneity was likely. All analyses were undertaken with RevMan 4.2 software.

- Primary outcomes
- 1. There were two approaches to improvement in hearing loss analysis depending on the nature of the data presented:

- a) Proportion of subjects with good hearing loss resolution (e.g. PTA improvement > 20 dB). Subjects were dichotomised into good outcome and poor outcome. The RR for good outcome with hyperbaric oxygen therapy was established using the intention to treat data of the hyperbaric oxygen therapy versus the control group. As an estimate of the statistical significance of a difference between experimental interventions and control interventions we calculated RR for benefit using hyperbaric oxygen therapy with 95% confidence intervals (CI). A statistically significant difference between experimental intervention and control intervention was assumed if the 95% CI of the RR did not include the value 1.0. As an estimate of the clinical relevance of any difference between experimental intervention and control intervention we calculated the number-needed-to-treat (NNT) and number-needed-to-harm (NNH) with 95% CI as appropriate.
- b) Comparison of the difference between the mean change in PTA in each group, hyperbaric oxygen versus non-hyperbaric oxygen. The weighted mean differences (WMD) in hearing loss recovery between hyperbaric oxygen and control groups were compared using RevMan 4.2. A statistically significant difference was defined as existing if the 95% CI did not include a zero WMD.

2. Relief of tinnitus. Was treated similarly to 1. above.

- Secondary outcomes
- 3. Activities of daily living (ADL). The weighted mean differences (WMD) in ADL between hyperbaric oxygen and control groups were to be compared as in 1. b) above.

4. Depression and mood disturbance. Methods were to depend on the nature of the data as in 1. b) above.

5. Adverse events. Dichotomous data were considered for adverse events (number of patients with adverse events versus number of patients without them in both groups) in the hyperbaric oxygen groups of the included studies.

- Sensitivity analyses
- We intended to perform sensitivity analyses for missing data and study quality.

- Missing data
- We employed sensitivity analyses using different approaches to input missing data. The best-case scenario assumed that none of the originally enrolled patients missing from the primary analysis in the treatment group had the negative outcome of interest whilst all those missing from the control group did. The worst-case scenario was the reverse.

- Study quality
- If appropriate, we intended to conduct a sensitivity analysis by study quality based on the Jadad score and an assessment of adequate sample size to detect the clinically important difference in outcome for which the study was designed.

- Subgroups
- Where appropriate data exist, we considered subgroup analysis based on:

- 1. Time between onset and therapy - early versus late presentation for treatment in the trial.
- 2. Aetiology of the ISSHL or tinnitus.

- 3. Dose of oxygen received (pressure, time and length of treatment course).
- 4. Nature of the comparative treatment modalities.
- 5. Severity of hearing loss and/or tinnitus.

Heterogeneity was explored and subgroup analyses performed when appropriate. Statistical heterogeneity was estimated using the I2 statistic and consideration was given to the appropriateness of pooling and meta-analysis.

Description of studies

We identified 68 publications apparently dealing with the use of HBOT for the treatment ISSHL and/or tinnitus. Initial examination confirmed 19 were case reports or case series, 18 were reviews without new data, nine were dealing with a different condition (acoustic trauma) and four were non-random comparative studies. These reports were excluded. One report was unobtainable but deemed unlikely to be a randomised or pseudo-randomised trial (Blagovesh 1990), leaving 15 possible comparative trials. After appraisal of the full reports we further excluded three reports as reviews without new data, three as comparative trials where all groups received HBOT, two as non-random comparative trials with historical controls or sequential treatment, and two as case series (see table 'Characteristics of excluded studies'). The other five trials were accepted into the review (Cavallazzi 1996; Fattori 2001; Hoffmann 1995; Hoffmann 1995b; Schwab 1998).

The included trials were published between 1995 and 2001, and the reviewers are unaware of any on-going RCTs in the area. In total, these trials include data on 254 participants, 133 receiving HBOT and 120 control (one participant was lost without information on allocation). The largest (Schwab 1998) accounts for 30% of cases. (See table: 'Characteristics of included studies').

Both the dose of oxygen per treatment session and for the total course of treatment varied between studies. The lowest dose administered was 1.5 ATA for 45 minutes daily for 15 days (Hoffmann 1995; Hoffmann 1995b), while the highest dose was 2.5 ATA for 60 minutes daily for 15 days (Cavallazzi 1996). All authors used between 1.5 and 2.5 ATA as a maximum oxygen pressure and the total number of individual treatment sessions varied from 10 (Fattori 2001; Schwab 1998) to 20 for some participants in Hoffmann 1995b.

All trials except Hoffmann 1995 included participants with acute hearing loss with or without tinnitus. Hoffmann 1995b accepted only patients who had not improved after two weeks of pharmacological therapy, Fattori 2001 accepted patients untreated within 48 hours of hearing loss, while Schwab 1998 accepted patients up to two weeks after loss. Cavallazzi 1996 did not define entry criteria. Hoffmann 1995 was the only trial to examine the effect of HBOT on chronic presentation and this trial accepted participants with at least six months of hearing loss. There was little information on exclusion criteria. Schwab 1998 specifically excluded candidates with contra-indications to therapy, while Fattori 2001 specifically excluded candidates with a probable cause for deafness such as acoustic trauma.

Comparator regimens differed between trials. Schwab 1998 and Cavallazzi 1996 compared HBOT to a multimodal pharmacological approach, while Fattori 2001 used a vasodilator alone. Hoffmann 1995 (chronic ISSHL) compared HBOT to a sham treatment and Hoffmann 1995b (acute ISSHL) compared HBOT to no treatment. Details of comparator therapies are given in the table 'Characteristics of included studies'.

The follow-up periods varied between immediately following the treatment course (Cavallazzi 1996; Hoffmann 1995) to 10 days (Fattori 2001) and 3 months (Schwab 1998; Hoffmann 1995b). All included studies reported at least one clinical outcome of interest. Of the outcomes identified above, these trials reported data on both primary outcomes (pure tone audiometric documented change in hearing and relief of tinnitus) but none of the secondary outcomes of interest.

Other outcomes (including non-clinical) reported by Fattori 2001 included: auditory evoked potentials, videonystagmography, static posturography, neurological examination, doppler echography, magnetic resonance imagery and computed tomography. No other trials reported additional outcomes.

Methodological quality

Study quality was generally assessed as low. Two of the four included studies were assigned a score of one (Cavallazzi 1996; Schwab 1998), two further studies a score of two (Fattori 2001; Hoffmann 1995b) and the remaining study a score of three (Hoffmann 1995). The significance of this small variation is unclear and it was not used as a basis for sensitivity analysis by study quality.

- Randomisation
- Allocation concealment was not adequate in any of the studies, being inadequate in Hoffmann 1995b and unclear in the remaining studies. Randomisation procedures were not described in any of the studies, and may not have been truly random for Cavallazzi 1996, where the allocation method was not clearly described. For none of the studies is there a clear indication that the investigators were unable to predict the prospective group to which a participant would be allocated.

- Patient baseline characteristics
- All participants had suffered ISSHL and/or tinnitus. Four of the studies defined a time-based entry criteria (Fattori 2001 48 hours; Schwab 1998 and Hoffmann 1995b two weeks; Hoffmann 1995 six months). All trials required no prior specific therapy except Hoffmann 1995b where all participants had failed to respond to two weeks of pharmacological therapy in hospital. Only Schwab 1998 defined a degree of hearing loss as a requirement for entry (at least 20 dB loss in one or more frequencies). Only Fattori 2001 and Cavallazzi 1996 stratified subjects on entry for severity of hearing loss. While all trials included subjects with ISSHL, only Schwab 1998 and Cavallazzi 1996 specifically identified individuals with tinnitus in the absence of hearing loss.

- Blinding
- Only Hoffmann 1995 described sham therapy with blinding of subjects to the allocated therapy. No trial described blinding of investigators or outcome assessors.

- Subjects lost to follow-up
- Schwab 1998 did not report results for seven subjects with ISSHL and 11 with tinnitus. This trial enrolled 31 subjects with both ISSHL and tinnitus, and 43 with one diagnosis or the other. It is not clear how many of the losses were individuals with both diagnoses, making an intention to treat analysis problematic. None of the remaining studies suffered any losses to follow-up, or reported any violation of allocated treatment. As Schwab 1998 did not report any dichotomous outcomes, sensitivity analysis making best and worst case analyses to examine potentially important effects of these losses on outcome has not been performed.

- Intention-to-treat analysis
- No trial mentioned this strategy, but neither were there any losses to follow-up or violations of protocol reported except for Schwab 1998. We did not attempt an intention to treat analysis with subjects from this trial.

Results

Primary outcomes

- 1. Acute ISSHL: Pure tone audiometric change in hearing (comparison 01)

- All trials reported on this outcome, but there were a variety of reporting methods that limited the possibility of pooling those results. We requested data on percentage return of hearing from all authors where it was not reported in the original paper, but none were able to provide this.

- 1.1 Proportion of subjects with greater than 50% return of hearing at end of therapy (comparison 01, outcome 01)
- Two trials reported this outcome (Cavallazzi 1996; Fattori 2001), involving 114 subjects (45% of the total subjects in this review). Cavallazzi 1996 contributed 64 subjects and Fattori 2001 50 subjects. There was no statistically significant increase in the proportion of subjects with more than 50% improvement in PTA assessed hearing loss over four frequencies following HBOT (RR of improvement with HBOT was 1.53, 95% CI 0.85 to 2.78, P = 0.16). There was moderate heterogeneity between trials (I2 = 38.2%) and therefore a random effects model was used to calculate the pooled estimate.
- Cavallazzi 1996 gave results stratified by severity of hearing loss at enrolment. There were no statistically significant differences reported. However, there was a trend suggested toward greater treatment effect with less severe presentation (RR for improvement of 50% with HBOT in mild hearing loss 1.42, 95% CI 0.79 to 2.55, P = 0.24; moderate loss 1.2, 95% CI 0.54 to 2.67, P = 0.66; severe loss 1.07, 95% CI 0.29 to 3.88, P = 0.92).

- 1.2 Proportion of subjects with greater than 25% return of hearing at end of therapy (comparison 01, outcome 02)
- Two trials reported this outcome (Fattori 2001; Cavallazzi 1996), involving 114 subjects (45% of the total subjects in this review). Cavallazzi 1996 contributed 64 subjects and Fattori 2001 50 subjects. There was a statistically significant increase in the proportion of subjects with more than 25% improvement in PTA assessed hearing loss over four frequencies following HBOT (RR of improvement with HBOT was 1.39, 95% CI 1.05 to 1.84, P = 0.02). There was no evidence of significant heterogeneity between trials (I2 = 0%) and therefore we used a fixed effects model to calculate the pooled estimate. The absolute risk difference of 22% is statistically significant, with a NNT to achieve one extra good outcome of 5 (95% CI 3 to 20).
- Cavallazzi 1996 gave results stratified by severity of hearing loss at enrolment. There were no statistically significant differences reported (RR for improvement of 25% with HBOT in mild hearing loss 1.33, 95% CI 0.89 to 1.99, P = 0.16; moderate loss 1.33, 95% CI 0.74 to 2.41, P = 0.34; severe loss 1.28, 95% CI 0.56 to 2.91, P = 0.56).

- 1.3 Mean improvement in PTA as a percentage of baseline (comparison 01, outcome 03)
- Only one trial contributed results to this outcome (Fattori 2001) involving 50 subjects (20% of the total), 30 (60%) randomised to HBOT and 20 (40%) to control. There was a mean improvement in PTA of 61% with the application of HBOT, versus an improvement of 24% in control subjects, and this difference was statistically significant (WMD 37% in favour of HBOT, 95% CI 22% to 53%).

- 1.4 Proportion of subjects with absolute improvement in PTA more than 20 dB (comparison 01, outcome 04)
- Only one trial contributed results to this outcome (Hoffmann 1995b) involving 20 subjects (8% of the total), 10 randomised to both HBOT and control. Only one subject improved and that individual was in the HBOT arm. There was no significant increase in the proportion of subjects with more than 20 dB return of hearing following the application of HBOT (RR 3.0, 95% CI 0.14 to 65.9, P = 0.49).

- 1.5 Mean improvement in hearing over all frequencies (dB) (comparison 01, outcome 05)
- Two trials reported on this outcome (Hoffmann 1995b; Schwab 1998) involving 77 subjects (30% of the total). Schwab 1998 contributed 57 subjects, and Hoffmann 1995b 20 subjects. While both trials reported a greater mean improvement in the HBOT arm

compared to the control (4.9 and 8.2 dB respectively), neither trial reported standard deviation around those means, making a pooled analysis impossible.

2. Chronic ISSHL: Pure tone audiometric changes in hearing (comparison 02)

- 2.1 Proportion of subjects with improvement in PTA (comparison 02, outcome 01)
- Only one trial contributed results to this outcome (Hoffmann 1995) involving 44 subjects (17% of the total), 22 randomised to each arm (HBOT and control). More individuals in the control group showed some improvement in hearing (seven versus 11), but the difference was not statistically significant (RR for improvement with HBOT 0.64, 95% CI 0.30 to 1.33, P = 0.23).

3. Acute Tinnitus: Relief of tinnitus (comparison 03)

- 3.1 Mean improvement in tinnitus score (comparison 03, outcome 01)
- Two trials reported on this outcome (Hoffmann 1995b; Schwab 1998) involving 53 subjects (21% of the total). Schwab 1998 contributed 33 subjects, and Hoffmann 1995b 20 subjects. While both 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.

4. Chronic Tinnitus: Relief of tinnitus (comparison 04)

- 4.1 Proportion of subjects with improvement in tinnitus score (comparison 04, outcome 01)
- Only one trial contributed results to this outcome (Hoffmann 1995) involving 44 subjects (17% of the total), 22 randomised to each arm (HBOT and control). More individuals in the control group showed some improvement in tinnitus (four versus nine), but the difference was not statistically significant (RR for improvement with HBOT 0.44, 95% CI 0.16 to 1.23, P = 0.12).

Secondary outcomes

- 5. Activities of daily living (ADL).
- No trials reported any data on this outcome.

- 6. Subjective or objective improvements in depression or mood disturbance.
- No trials reported any data on this outcome.

- 7. Hearing handicap inventory change (and similar tool for tinnitus).
- No trials reported any data on this outcome.

- 8. Adverse events associated with hyperbaric oxygen therapy and comparators.
- No trials reported any data on this outcome.

Three of these trials had low power to detect clinically significant differences in the main outcome of interest (a 50% improvement in average pure tone hearing loss or subjective tinnitus score), and the other two (Hoffmann 1995; Schwab 1998) had > 80% power to detect a clinically significant improvement in hearing from the control group estimates. No trial reported formal power or sample size calculation. Details are given in the table 'Characteristics of included studies'.

Discussion

This review has included data from five trials and we believe these represent all randomised 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 (Cavallazzi 1996; Fattori 2001) that HBOT increases the proportion of patients gaining more than 25% improvement in hearing, while one of those trials (Fattori 2001) suggested there was a greater mean improvement in PTA as a percentage of baseline following HBOT. 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 five trials with 254 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 (Jadad scores: two trials scored one, two trials scored two and the other scored three), 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. The conclusions of this review are, therefore, to be interpreted with great caution.

These trials were published over a six year period up to 2001, 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. In particular, the Hoffmann 1995b trial, which differed significantly in that these authors admitted only subjects who had failed to respond to two weeks of intensive multiple pharmacotherapy, did not contribute to any pooled analysis. Response rates stratified by severity of hearing loss were only reported by Cavallazzi 1996 and while these suggest a trend to greater treatment effect in those less severely affected, there is no statistical significance at any reported severity grade, and we have not subjected any possible trend to formal statistical testing. Patient inclusion criteria were not standard, and were poorly reported in some trials. No standard severity scale was employed across these trials, and the time to entry varied from within 48 hours for Fattori 2001 to two weeks for Schwab 1998 and Hoffmann 1995b.

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% or 25% from baseline to the end of therapy. While the chance of a 50% improvement was not significantly increased following HBOT, the chance of a 25% improvement in hearing was statistically significant (RR 1.39, 95% CI 1.05 to 1.84, P = 0.02). Heterogeneity did not seem to be an issue (I^2 = 0%). This analysis suggests that we would need to treat five patients with HBOT in order to improve one person's hearing by 25% (NNT 5, 95% CI 3 to 20). Given the small number of subjects and generally poor quality of these trials, this result needs to be interpreted with caution. Furthermore, the clinical significance of a 25% 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 (Hoffmann 1995b; Schwab 1998). 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 (Hoffmann 1995).

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 by as many as 50% of those having a course of 30 treatments (Khan 2003). 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. Barotrauma can affect any air-filled cavity in the body (including the middle ear, lungs and respiratory sinuses) and occurs as a direct result of compression. Aural barotrauma is by far the most common as the middle ear air space is small, largely surrounded by bone and the sensitive tympanic membrane, and usually requires active effort by the patient in order to inflate the middle ear through the eustachian tube on each side. Barotrauma is thus not a consequence of HBOT directly, but rather of the physical conditions required to administer it. Most episodes of barotrauma are mild, easily treated or recover spontaneously and do not require the therapy to be abandoned. Less commonly, HBOT may be associated with acute neurological toxicity manifesting as seizure.

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.

Reviewers' conclusions

Implications for practice

There is limited evidence from methodologically poor studies 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. Moreover, this review does not give any information regarding the safety of HBOT for these patients.

Implications for research

Given the findings of improved hearing with the use of HBOT in these patients, there is a case for large randomised trials of high methodological rigour in order to define the true extent of benefit (if any) from the 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 is not known. Any future trials would need to consider in particular:

- appropriate sample sizes with power to detect expected differences
- careful definition and selection of target patients
- appropriate range of oxygen doses per treatment session (pressure and time) as well as total number of treatments
- appropriate and carefully defined comparator therapy
- use of an effective sham therapy
- effective and explicit blinding of outcome assessors
- appropriate outcome measures including all those listed in this review
- careful elucidation of any adverse effects
- the cost-utility of the therapy.

Acknowledgements

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Potential conflict of interest

None known.

Tables

Characteristics of included studies

Study	Cavallazzi 1996
Methods	Method of allocation not clear, no discussion of blinding.
Participants	64 subjects with a diagnosis of ISSHL, time course unknown.Stratified into mild, moderate, severe and 'deep'.
Interventions	Control (30): Multiple drug therapy consisting of heparin, betamethasone, nicotinic acid, flunarizine, citidinephosphocoline, dextran, vitamins, neurotropic and antiviral drugs - doses not given.HBOT (34): Pharmacotherapy as for control group plus oxygen at 2.5 ATA for 60 minutes daily for 15 sessions over three weeks.
Outcomes	PTA recovery, stratified into pecentage improvement shown at 4 strata of severity at presentation.
Notes	Rank 2 for sample size. Power to detect significant difference in proportion with 50% recovery of hearing is < 80%. Jadad score 0. Further details requested from authors but no reply to date.
Allocation concealment	B
Study	Fattori 2001
Methods	Method of randomisation not clear, no discussion of blinding.
Participants	50 subjects with ISSHL referred within 48 hours. Stratified into mild, moderate and severe.
Interventions	Control (20): Vasodilator therapy: 10 day course iv 200 mg/day buflomedil in 250 ml physiological solution. No sham treatment.HBOT (30): 10 once-daily treatments breathing 100% oxygen at 2.2 ATA for 90 minutes.
Outcomes	PTA recovery, stratified into percentage improvement shown at 3 strata of severity at presentation.Mean PTA recovery.
Notes	Rank 3 for sample size. Power to detect improvement from 25% to 50% of subjects with 50% return of hearing < 80%. Jadad score 2.
Allocation concealment	B
Study	Hoffmann 1995

Methods	Method of randomisation not clear, patients and outcome assessors blinded.
Participants	44 subjects with ISSHL for greater than six months.
Interventions	Control (22): Air breathing at 1.5 ATA for 45 minutes daily, five days each week for three weeks.HBOT (22): 100% oxygen breathing at 1.5 ATA on the same schedule as controls.
Outcomes	Improved hearing and tinnitus.
Notes	Rank 4 for sample size. Power > 80% to detect an increase in proportion of subjects with significant return of hearing. Jadad score 3.
Allocation concealment	B
Study	Hoffmann 1995b
Methods	Method of randomisation not clear, not blinded.
Participants	20 subjects with ISSHL with or without tinnitus. All subjects had no improvement after 14 days of pharmacological treatment with hydroxyethyl starch, pentoxifylline and cortisone.
Interventions	Control (10): No treatment.HBOT (10): 100% oxygen at 1.5 ATA for 45 minutes daily, five days each week for two to four weeks (10 to 20 sessions).
Outcomes	Audiometry at three months, subjective tinnitus scale.
Notes	Rank 5 for sample size. Power to detect mean hearing improvement of 20 dB more in active group than control < 80%. Jadad score 2
Allocation concealment	D
Study	Schwab 1998
Methods	Method of randomisation not clear, no evidence of blinding.
Participants	75 subjects with sudden hearing loss with at least 20 dB loss in one or more frequencies and /or tinnitus, seen within two weeks and without any prior therapy.
Interventions	Control (38): No treatment.HBOT (37): 100% oxygen at 1.5ATA for 45 minutes daily, five days each week for two to four weeks (10 to 20 sessions).
Outcomes	Audiometric hearing improvement and tinnitus.
Notes	Rank 1 for sample size. Power > 80% to detect an increase in proportion of subjects with signficant return of hearing. Jadad score 1.
Allocation concealment	B

ISSHL - Idiopathic sudden sensorineural hearing loss
PTA - Pure Tone Audiometry

Characteristics of excluded studies

Study	Reason for exclusion

Dauman 1985	Non-random allocation and many patients had sequential treatment
Dauman 1993	Two groups both received hyperbaric oxygen
Goto 1979	No indication that allocation to groups was random
Joachims 1978	Case series only
Lamm 1995	Review only, no new data
Lamm 1998	Review only, no new data
Sano 1988	Case series
Sparacia 2003	No appropriate comparator therapy to HBOT
Tisch 2000	Review, no new data
Xiao 1986	No suitable comparator to HBOT

Additional tables

Table 01 Search strategies for different databases

CENTRAL	MEDLINE (OVID)	CINAHL	EMBASE	AMED	LILACS	PUBMED
#1HYPERBARIC OXYGENATION #2oxygen* #3HBOT #4HBO #5#1 or #2 or #3 or #4 or #5 #6HEARING LOSS, SUDDEN #7HEARING LOSS, SENSORINEURAL #8sudden* #9#7 and #8 #10sshl #11snhl #12ishl #13isshl #14issnhl #15ssnhl #16(sudden near hearing) #17(sudden near deaf*) #18#6 or #9 or #10 or #11 or #12 or #13 or #14 or #15 or #16 or #17 #19TINNITUS #20tinnitus	1 Hyperbaric Oxygenation/ 2 oxygen$.tw. 3 HBOT.tw. 4 HBO.tw. 5 or/1-4 6 exp Hearing Loss, Sudden/ 7 exp hearing loss, sensorineural/ 8 sudden$.tw. 9 7 and 8 10 sshl.tw. 11 snhl.tw. 12 ishl.tw. 13 isshl.tw. 14 issnhl.tw. 15 ssnhl.tw. 16 (sudden adj3 hearing).tw. 17 (sudden adj3 deaf$).tw. 18 or/6,9-17 19 exp Tinnitus/	1 exp Hyperbaric Oxygenation/ 2 oxygen$.tw. 3 HBOT.tw. 4 HBO.tw. 5 or/1-4 6 exp hearing loss, sensorineural/ 7 sudden$.tw. 8 6 and 7 9 sshl.tw. 10 snhl.tw. 11 ishl.tw. 12 isshl.tw. 13 issnhl.tw. 14 ssnhl.tw. 15 (sudden adj3 hearing).tw. 16 (sudden adj3 deaf$).tw. 17 or/8-16 18 exp Tinnitus/ 19 tinnitus.tw.	1 exp hyperbaric oxygen/ 2 oxygen$.tw. 3 HBOT.tw. 4 HBO.tw. 5 or/1-4 6 exp sudden deafness/ 7 exp perception deafness/ 8 sudden$.tw. 9 7 and 8 10 sshl.tw. 11 snhl.tw. 12 ishl.tw. 13 isshl.tw. 14 issnhl.tw. 15 ssnhl.tw. 16 (sudden adj3 hearing).tw. 17 (sudden adj3	1 exp hyperbaric oxygen/ 2 oxygen$.tw. 3 HBOT.tw. 4 HBO.tw. 5 or/1-4 6 sshl.tw. 7 snhl.tw. 8 ishl.tw. 9 isshl.tw. 10 issnhl.tw. 11 ssnhl.tw. 12 (sudden adj3 hearing).tw. 13 (sudden adj3 deaf$).tw. 14 or/6-13 15 exp tinnitus/ 16 tinnitus.tw. 17 15 or 16 18 5 and (14 or 17)	hyperbaric [Palavras] and tinnitus OR sudden OR sshl OR snhl OR ishl OR isshl OR issnhl OR ssnhl [Palavras]	#1 Search hyperbaric oxygenation[mh] #2 Search hyperbaric 09:42:38 #3 Search hbo 09:42:46 #4 Search hbot 09:42:49 #5 Search #1 OR #2 OR #3 OR #4 09:43:05 #6 Search Field: All Fields, Limits: Randomized Controlled Trial 09:43:23 #7 Search #5 Limits: Randomized Controlled Trial 09:43:46 #8 Search tinnitus OR sudden OR ishl OR sshl OR snhl OR isshl OR issnhl OR ssnhl

#21#19 or #20 #22#18 or #21 #23#5 and #22	20 tinnitus.tw. 21 (ear adj1 (buzz$ or ring$)).tw. 22 or/19-21 23 5 and 22	20 (ear adj1 (buzz$ or ring$)).tw. 21 or/18-20 22 17 or 21 23 5 and 22	deaf$).tw. 18 or/6,9-17 19 exp tinnitus/ 20 tinnitus.tw. 21 (ear adj1 (buzz$ or ring$)).tw. 22 or/19-21 23 18 or 22 24 5 and 23			Limits: Randomized Controlled Trial 09:45:07 #9 Search #7 AND #8 Limits: Randomized Controlled Trial 09:45:32

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** indicates the major publication for the study*

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Graphs

Graphs and Tables

To view a graph or table, click on the outcome title of the summary table below.

01 Acute presentation. Recovery of hearing as measured by audiometry				
Outcome title	No. of studies	No. of participants	Statistical method	Effect size
01 Greater than 50% return of hearing			Relative Risk (Random) 95% CI	Subtotals only
02 Greater than 25% return of hearing			Relative Risk (Fixed) 95% CI	Subtotals only
03 Mean improvement in PTA (% baseline)	1	50	Weighted Mean Difference (Fixed) 95% CI	37.30 [21.75, 52.85]
04 Mean absolute improvement in PTA > 20 dB	1	20	Relative Risk (Fixed) 95% CI	3.00 [0.14, 65.91]
05 Mean hearing improvement over all frequencies (dB)	2	77	Weighted Mean Difference (Fixed) 95% CI	Not estimable
02 Chronic presentation. Recovery of hearing as measured by audiometry.				
Outcome title	No. of	No. of	Statistical method	Effect size

	studies	participants		
01 Some improvement, all grades	1	44	Relative Risk (Fixed) 95% CI	0.64 [0.30, 1.33]

03 Acute presentation. Improvement of tinnitus

Outcome title	No. of studies	No. of participants	Statistical method	Effect size
01 Mean change in tinnitus score (0 to 10 scale)	1	33	Weighted Mean Difference (Fixed) 95% CI	Not estimable

04 Chronic presentation. Improvement of tinnitus.

Outcome title	No. of studies	No. of participants	Statistical method	Effect size
01 Improvement in tinnitus	1	44	Relative Risk (Fixed) 95% CI	0.44 [0.16, 1.23]

Cover sheet

Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and tinnitus

Reviewer(s)	Bennett MH, Kertesz T, Yeung P
Contribution of Reviewer(s)	Bennett - Conceived review, primary author of all sections, handsearching, critical appraisal and statistics Kertesz - Assistance with text, content expert in otorhinolaryngology, critical appraisal of selected articles Yeung - Assistance with text, content expert in otorhinolaryngology, critical appraisal of selected articles
Issue protocol first published	2004 issue 2
Issue review first published	2005 issue 1
Date of last minor amendment	Information not supplied by reviewer
Date of last substantive amendment	06 September 2004
Most recent changes	Information not supplied by reviewer
Date new studies sought but none found	Information not supplied by reviewer
Date new studies found but not yet included/excluded	Information not supplied by reviewer

Date new studies found and included/excluded	Information not supplied by reviewer
Date reviewers' conclusions section amended	Information not supplied by reviewer
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Cochrane Library number	CD004739
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Synopsis

Hyperbaric oxygen may improve deafness after sudden hearing loss of unknown cause, but there is little evidence of benefit for tinnitus.

Idiopathic sudden sensorineural hearing loss (ISSHL) is common and often results in permanent hearing loss. Tinnitus (abnormal persistent noises) is similarly common and often accompanies the hearing loss. Although their cause is not clear, these complaints may arise from a lack of oxygen secondary to a vascular problem not yet identified. Hyperbaric oxygen therapy (HBOT) increases the supply of oxygen to the ear and brain to reduce the severity of hearing loss and tinnitus. Methodologically poor studies with a limited number of patients could not make clear the value of HBOT for ISSHL or tinnitus. Further research is needed.

Keywords

Humans; Hearing Loss, Sensorineural[*therapy]; Hearing Loss, Sudden[*therapy]; *Hyperbaric Oxygenation; Randomized Controlled Trials; Tinnitus[*therapy]