

Hyperbaric oxygen therapy in the treatment of sudden sensorineural hearing loss: a retrospective analysis of outcomes

Susannah Sherlock, Kenneth Thistlethwaite, Mohsina Khatun, Christopher Perry and Alexis Tabah

Abstract

(Sherlock S, Thistlethwaite K, Khatun M, Perry C, Tabah A. Hyperbaric oxygen therapy in the treatment of sudden sensorineural hearing loss: a retrospective analysis of outcomes. *Diving and Hyperbaric Medicine*. 2016 September;46(3):160-165.)

Objective: To analyse predictive factors affecting outcome after treatment with hyperbaric oxygen (HBOT) in patients with idiopathic sudden sensorineural hearing loss (ISSHL).

Methods: This is a retrospective audit of outcome in 77 consecutive patients referred for consideration of HBOT for ISSHL for either adjunctive treatment or after failure of steroid therapy. The hearing measured from the pre- and post-HBOT pure-tone audiogram (PTA₄) at four frequencies; 500 Hz, 1 kHz, 2 kHz and 4 kHz, was averaged and compared. The PTA₄ score was classified into three groups: complete improvement (≤ 25 dB residual hearing loss); moderate improvement (11–50 dB gain) and no improvement (≤ 11 dB gain). Data were also analysed using mean residual loss on completion as the outcome measure.

Results: Seventy-six patients underwent 1,029 HBOT sessions. Twenty five of 78 ears (33%) had complete resolution of deafness after HBOT. A further 31 (40%) had a significant improvement in PTA₄. Delay (> 28 days) and older age were associated with worse outcomes in PTA₄ improvement. Those with less severe hearing loss and short delay (< 15 days) had the best outcome (mean residual loss 28 dB). Eight of nine patients who were delayed > 28 days had no improvement in PTA₄.

Conclusions: Fifty-six of 76 (74%) patients had complete (25) or moderate (31) improvement in hearing loss after HBOT. Short delay to HBOT, a severer degree of hearing loss and younger age were the best predictive factors of improved PTA₄. Outcome was poor if treatment was delayed over 28 days. Well-designed randomised controlled trials are needed to clarify the role of HBOT and steroids.

Key words

ENT; inner ear; risk factors

Introduction

The diagnosis, incidence, pathology, treatment and natural history of idiopathic sudden sensorineural hearing loss (ISSHL) are all areas of controversy. It is defined by the National Institute on Deafness and Other Communications Disorder (NIDOC) as the “*sudden loss of hearing over three contiguous pure-tone frequencies of 30 dB or more that develops over 72 hours or less*”.¹ Due to the high number of potential causative agents, including ischaemia, it is not surprising that many therapies have been tried: among them hyperbaric oxygen treatment (HBOT), rheological agents, antiviral agents, acupuncture, vitamins and steroids.^{2–4}

The American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) recently released clinical practice guidelines suggesting HBOT could be useful up to three months after onset of symptoms; however, the hyperbaric literature suggests best outcomes if utilised within two weeks of symptoms.^{5,6} On the basis of this discrepancy we conducted a retrospective audit to identify patient factors affecting outcome.

BACKGROUND

ISSHL was first described in 1944.⁷ The reported incidence of 160 per 100,000 may be an underestimation because

up to 65% of cases resolve spontaneously usually within two weeks.^{8,9} This high rate has been disputed by others and may be as little as 25%.¹⁰ However, spontaneous resolutions are acknowledged to be rare beyond two weeks.¹¹ Recently published work suggests 72% of ISSHL cases are idiopathic.¹² Most cases are unilateral. Bilateral disease has a poorer prognosis and should raise suspicion of serious systemic aetiology.¹³ The recurrence rate is quoted as being 5%.¹⁴ ISSHL is considered to be a medical emergency by many (though not all) ear, nose and throat (ENT) surgeons and can have a profound impact on a patient’s ability to communicate, especially when it is bilateral.¹⁵ However, despite its importance, the optimum management remains unclear.

A Cochrane review of the use of anti-viral agents to treat ISSHL concluded their use was questionable.¹⁶ AAO-HNS clinical practice guidelines suggest they should not be routinely prescribed. Corticosteroids have traditionally been considered the gold standard treatment but there is considerable debate about the level of effectiveness, dosing, timing and route of administration. A Cochrane review suggests their usefulness on the basis of randomised, controlled trials (RCTs) is not proven.¹⁷ HBOT has been advocated as salvage treatment when steroids have failed.¹⁸ When HBOT is employed, there is inconsistency in previous publications on the timing, treatment depth and number

of treatments. A Cochrane review of the use of HBOT for ISSHL concluded that “for people with acute ISSHL, the application of HBOT significantly improved hearing, but the clinical significance remains unclear.”¹⁹

In a survey of the clinical management of ISSHL by otorhinolaryngologists in the UK, 96% recommend corticosteroids, 45% recommend antiviral therapy and only 4% recommend HBOT.³ Despite supportive evidence for HBOT, it is not recommended as first-line treatment but, rather, as salvage therapy when other medical therapies have failed.^{8,20}

HBOT RATIONALE

HBOT was first recommended in the treatment of ISSHL in the 1960s to improve cochlear ischaemia by reducing cochlear oedema. Auditory cells and peripheral nerve fibres have no direct vascular supply and are dependent on oxygen diffusion through the perilymph and cortilymph. This is likely to be improved by circulating arterial oxygen tensions over 1,500 mm Hg produced by HBOT at 243 kPa. Treatment at 152 kPa has been shown to be of no benefit after unsuccessful steroid therapy.²¹ Furthermore, HBOT has been demonstrated to have immunomodulatory and anti-inflammatory effects, to improve local haemodynamics and induce angiogenesis.^{22,23} HBOT is considered a relatively safe treatment. The most common reported side effect is barotrauma of the tympanic membrane, mild and self-limiting in most cases, (overall incidence often quoted as 17.8%) which can complicate therapy, especially if continued treatment requires grommet insertion.²⁴

Methods

We conducted a retrospective review of all the patients treated with HBOT for ISSHL at a quaternary referral hospital, the Royal Brisbane and Women’s Hospital, between 01 January 2012 and 30 June 2014. This study is reported in accordance with the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) guidelines.²⁵ The project was exempted from full ethics review as a low- and negligible-risk research project by the Royal Brisbane and Women’s Hospital Human Research Ethics Committee (HREC/15/QRBW/55).

Patient records were identified from the local database with the code “hearing loss”. Only patients with the strict definition of ISSHL as used by the NIDOCDC were included. The pre- and post-HBOT audiograms were extracted and general demographics collected such as date of birth, gender and previous medical history. Possible predictors of outcome recorded included age, sex, affected ear, severity of hearing loss, delay until HBO treatment and number of treatments.

HBOT consisted of 90 mins daily at 243 kPa for 10 treatments, then repeat audiogram. If the audiogram had

improved less than 11 dB, treatment was ceased. If 11 dB or more, five further HBOT were offered. The audiogram was repeated after every five HBOT until no improvement was noted, at which point treatment ceased. The hearing measured from pure-tone audiogram (PTA₄) at four frequencies (500 Hz, 1 kHz, 2 kHz and 4 kHz) was averaged and compared. All data were de-identified and extracted to an Excel™ file to be analysed by an independent statistician.

To explore potential factors associated with outcome, we defined the following categorical variables: severe (> 60 dB) and moderate (≤ 60 dB) pre-treatment hearing loss; age with ≤ 50 years and > 50 years. Number of treatments was categorised as ≤ 10 or > 10. Delay in presentation was categorized as early (within 14 days), moderately delayed (15–28 days) and late (> 28 days). In the absence of consensus in the literature, these categories were defined arbitrarily using categories published by other groups. Mean residual losses were compared by the delay in presentation for treatment accounting for severity of pre-treatment PTA₄ score using the F-test statistic.

Improvement in the PTA₄ score (before/after HBOT) was categorized into three groups; complete recovery, moderate improvement (11–50 dB gain) and no improvement (≤ 10 dB gain). Complete recovery was defined as a hearing loss of ≤ 25 dB (below predicted) at the end of treatment as defined by the NIDOCDC and the World Health Organisation.^{1,26}

STATISTICS

General linear modelling was applied to examine the unadjusted and adjusted association of patient demographic and clinical characteristics with the absolute change in hearing after treatment (dB). The change in hearing was calculated taking the difference between post- and pre-treatment PTA₄ score. The results were rounded to the nearest whole number. Selection of potential characteristics for the adjusted analysis was based on the unadjusted association with *P*-value < 0.10. A stepwise, backward selection process was applied to find the parsimonious model with all the significant covariates. The estimated marginal mean values were presented and the results were evaluated using 95% confidence intervals (CI) and the *P*-values. A *P*-value less than 0.05 was accepted as statistically significant.

Results

There were 77 patients with 79 affected ears (two bilateral). One patient with unilateral loss was excluded as they did not meet the definition of ISSHL. The remaining 76 patients underwent a total of 1,029 patient compressions (Table 1). Nine patients suffered self-limiting tympanic barotrauma and two required grommet insertion to complete therapy. There were no major complications. All patients had been prescribed oral corticosteroids at the specialist’s discretion of

Table 1

Patient demographics (*n* = 78), hyperbaric oxygen treatment (HBOT) and hearing loss pre and post HBOT; * *P* < 0.001

Patient characteristics	Mean	(95% CI)
Pre-treatment hearing loss (dB)	69	(64, 75)
Post-treatment hearing loss (dB)	47	(40, 53)
Mean gain in hearing post HBOT (dB)	23	(19, 27)*
Age (year)	52	(48, 56)
Male/Female	42/36	
Ear treated (left/right)	41/37	
Delay to HBOT (day)	13	(11, 16)
No. of HBOT	14	(12, 15)
Smoker (<i>n</i> = 50)		
Ever smoker	20	
Never smoker	19	
Unknown	11	
Heavy alcohol use (<i>n</i> = 50)		
Yes	5	
No	33	
Unknown	12	

Figure 1

Treatment outcomes relative to the delay in presentation

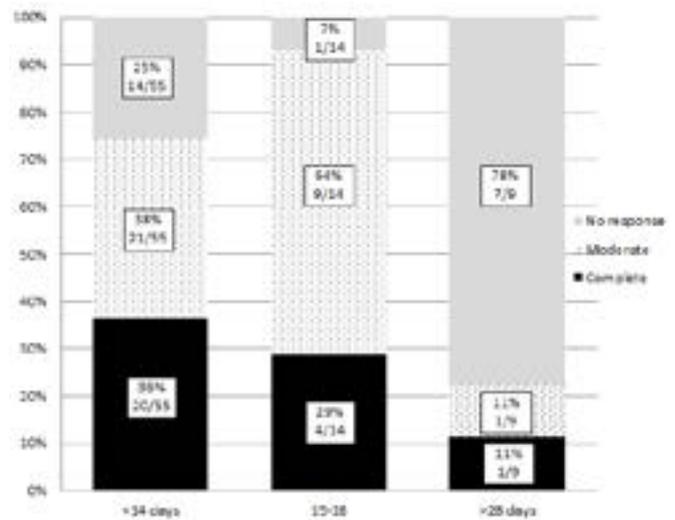


Table 2

Pure-tone audiogram PTA₄ (dB) scores after treatment for two levels of pre-treatment PTA₄ and delay at presentation; mean (95% CI)

Pre-treatment PTA ₄ Delay to presentation	(n)	Post-treatment PTA ₄ loss		P-value
		≤ 60 dB	> 60 dB	
< 15 days	55	Mean 28 (95% CI 21, 34)	Mean 59 (95% CI 47, 71)	≤ 0.001
15–28 days	14	Mean 30 (95% CI 16, 44)	Mean 55 (95% CI 35, 75)	0.151
> 28 days	9	Mean 50 (95% CI 31, 69)	Mean 63 (95% CI 30, 95)	0.280
Mean (95% CI)	76	31 (25, 36)	59 (50, 68)	
P-value		0.027	0.911	

dose, most commonly 1 mg·kg⁻¹ prednisone, prior to referral. All patients had an MRI scan to exclude retrocochlear malignancy.

Among the four continuous variables: age; mean gain in hearing and post-treatment hearing loss all followed a normal distribution. Pre-treatment hearing loss did not, and should be more correctly shown as a median score since it was moderately skewed (skewness = 0.685). Also, delay in presentation was positively skewed. However, the mean score of delay in presentation was 13 days and the median was 12 days (interquartile range, IQR 4, 17), which are not much different from each other. For consistency with other mean values in Table 1, the mean values for this variable with 95% CI are shown.

Figure 1 compares the percentage of people who benefitted from HBOT (measured by improvement in PTA₄) between groups classified by delay to HBOT. Delay over 28 days meant no improvement with HBOT for seven of the nine patients in this category. One patient had complete recovery

with delayed presentation compared to 20 patients in the < 14 day group.

Fifty-six (73%) of 78 ears with ISSHL had complete or moderate improvement in hearing loss when HBOT was added to conventional steroid therapy. Improvement was moderate in 31 patients after HBOT, whilst 24 (25 ears; 32%) were considered to have normal hearing post treatment, according to the definition of the NIDOC. Nine patients had minor worsening of PTA₄ (range -7 dB to -1 dB deterioration) which was not considered a significant change on the audiogram.

Table 2 demonstrates mean residual hearing loss after treatment among the patients with different delay times and accounting for different levels of pre-treatment PTA₄ scores. Results show a significant effect on poor outcome for delay to HBOT but only for the moderate hearing loss afflicted patients (*P* = 0.027). Delay did not show a statistically significant association in those with > 60 dB loss at presentation (*P* = 0.911). Of those with greater impairment,

Table 3

Unadjusted and adjusted means (95% CI and *P*-values) of the change in hearing after hyperbaric oxygen treatment by the patient's demographic and clinical features

Selected characteristics (<i>n</i> = 78)	Change in hearing after the treatment (dB)					
	Unadjusted		<i>P</i> -value	Adjusted		<i>P</i> -value
	Mean	(95% CI)		Mean	(95% CI)	
Pre-treatment PTA₄ (dB)						
≤ 60	34	18 (12, 24)	0.032	16	(9, 22)	0.029
60+	44	27 (21, 32)		25	(18, 31)	
Age (y)						
≤ 50	37	27 (21, 33)	0.086	24	(17, 30)	0.043
50+	41	19 (14, 25)		16	(10, 22)	
Sex						
Male	42	25 (19, 30)	0.393			
Female	36	21 (15, 27)				
Ear						
Left	41	19 (14, 25)	0.071	16	(10, 22)	0.071
Right	37	27 (21, 33)		23	(17, 30)	
Number of treatments						
≤ 10	33	21 (15, 28)	0.566			
10+	45	24 (18, 29)				
Delay in presentation						
Early presentation	55	24 (20, 29)	0.036	24	(19, 28)	0.028
Moderately delayed	14	26 (17, 36)		27	(18, 36)	
Late presentation	9	8 (-4, 20)		9	(-3, 20)	

even early intervention with HBOT was less likely to be of benefit when examined with respect to final PTA₄ rather than absolute change in PTA₄ ($P < 0.001$). Early or moderately delayed presentation showed significant benefit compared to late presentation ($P = 0.028$).

Age also became a significant prognostic indicator in the adjusted analysis ($P = 0.043$), showing younger patients derived more dB gain. The unadjusted and adjusted association with the absolute change in hearing (dB improvement) after the treatment is presented in Table 3. In the adjusted analysis, patients with severe hearing loss (> 60 dB) had a significantly greater improvement (25 dB) compared to patients with moderate hearing loss (16 dB) ($P = 0.029$).

Discussion

In this study of patients with ISSHL treated with HBOT we found that delay to treatment was strongly correlated with poor outcome, which is consistent with previous reports.²⁷ Delay in receiving HBOT of more than four weeks significantly reduced any perceived benefit of HBOT; indeed, successful treatment of ISSHL by any modality after four weeks is rare. Our data does not support the AAO-HNS guidelines that HBOT should be considered up to three months after onset of sudden deafness. We would suggest HBOT beyond four weeks has little benefit but should be considered on a case-by-case basis.

Younger age, hearing loss > 60 dB and early HBOT were significant prognostic indicators of a greater improvement in PTA₄. The best hearing outcome, when defined as mean residual hearing loss post treatment, was achieved in younger patients with a hearing loss < 60 dB who presented early. This group achieved a mean residual loss of 28 dB which approaches the adult defined limits of normal hearing at -25 dB. Severe loss and younger age have been reported to be good prognostic indicators of success in patients treated with steroids.²⁸ This may be because those with a greater hearing loss have more to gain and the young have less vascular disease. Reports on age-related effects on responses to treatment have been conflicting when comparing different therapies.^{16,29} Our results support younger age as a good prognostic indicator of response to HBOT.

However, when analysing the data using the more patient-centred outcome measure of mean residual loss after treatment as the outcome measure rather than the overall improvement in PTA₄, the results were different. Those with a loss < 60 dB achieved better outcomes ($P \leq 0.001$). And even more interestingly, delay had a statistically significant negative effect on outcome in the group with a loss < 60 dB ($P = 0.027$). The same effect from delay to treatment was not reflected in the group with an initial loss > 60dB. We hypothesise that the evolution of the injury involves an additional pathology that causes a more severe audiological impairment and resistance to treatment with HBOT. This may simply represent a

more severe ischaemic insult with secondary oedema and inflammation and is worthy of further investigation.

This highlights the importance of choice of outcome measure when assessing improvement. Residual hearing loss after treatment is arguably more functionally important than absolute change in PTA₄ from presentation. A patient with profound loss may have a marked measured improvement in PTA₄ but still have a severe hearing deficit resistant to amplification. We believe residual mean loss is the better outcome measure. Those who presented with profound loss but gained some hearing improvement may still have benefitted from HBOT. This level of gain can functionally change a non-aidable level of deafness to a level that can be improved with a hearing aid. However, the numbers in the delayed group were small, so interpretation of statistical significance should be cautious.

Definitions of what constitutes a response to treatment differ widely in the literature. Improvement is usually defined as greater than 10 dB improvement in pure-tone average (PTA) but other definitions and mathematical formulae have been used in different studies. In our analysis a change of 10 dB or less was considered no improvement. Some studies have also used speech discrimination score (SDS) as a measurement of improvement. We chose to measure PTA₄ to assess response as it is simple, repeatable and robust. This is an easier marker than SDS for the hyperbaric physician to interpret and to tailor treatment duration by. PTA₄ and SDS usually reflect each other; however, some authors consider the mean PTA₄ to be a more objective measurement of outcome than SDS.^{4,30} Future studies should include SDS and PTA₄ plus a functional assessment such as the (modified) Amsterdam Inventory for Auditory Disability and Handicap and a quality of life score such as that developed by Hawthorne.^{30,31} No trials to date have assessed the functional impact of any measured improvements in PTA₄.

In the natural history of ISSHL described for 88 patients, only five patients had a good recovery after 14 days.⁹ This study had a variety of interventions including conservative management and there was no difference shown between those who received drugs and those who did not. In our study, 13 of the 14 patients referred between 15 to 28 days after onset showed complete or moderate recovery. This is important since spontaneous improvement is reported in the ENT literature to be unlikely beyond two weeks. Interestingly, this group had the highest percentage of overall responders. This needs to be interpreted with caution as the smaller number of patients in this group decreases the power of the calculation. This may suggest that ischaemia reversal is not solely responsible for improvement. The mechanism may be due to reduction of oedema or blunting of secondary ischaemia due to reperfusion injury or may even indicate that HBOT has a better chance of success after steroid priming.

If the suggested pathology in patients who improve with HBOT is considered to be ischaemia, it would seem reasonable to institute therapy as early as possible. When delay was over 28 days, our small sample (nine patients) suggested reduced benefit from HBOT. This sample size is too small to determine the therapeutic window for HBOT. However the association with delay and worse outcome is very strong in the analysis using mean residual loss in those with ≤ 60 dB (34 patients) and reached high statistical significance ($P = 0.027$). Delay to HBOT is often due to misdiagnosis and being unable to access an ENT surgeon in a timely fashion. This should be addressed so patients get urgent referral for specialist care.

The incidence of barotrauma was similar to that reported in a French study and lower than previously reported in other series.^{24,32}

There are limitations to the present study; most importantly that it is a retrospective, single-cohort study without a control group. In addition, the lack of inclusion of SDS scores and the risk of chance associations owing to the small numbers of patients in each group may be important. Most patients were referred promptly (early presentation) whilst still taking steroids rather than after failure of primary treatment. This makes it difficult to discriminate between HBOT and steroid effectiveness or a potential synergistic interaction between the two treatments in the early presenters. A randomised controlled trial comparing the effectiveness of steroids, HBOT alone or in combination is warranted to determine the most appropriate treatment for ISSHL.

Conclusion

Fifty-six (73%) of 78 ears with ISSHL had complete or moderate improvement in hearing loss when HBOT was added to conventional steroid therapy. Shorter delay prior to commencing HBOT and being younger than 50 years old were the best predictors of good outcome. In those with < 60 dB loss on presentation, delay to treatment impacted negatively on outcome whether the outcome measure was overall improvement in dB gain or mean residual loss at the end of treatment. This study has not established clearly a role for HBOT in ISSHL but the outcomes do appear to be improved compared to the literature on currently accepted treatments. Therefore, there is an urgent need for a large randomised controlled trial to fully elucidate the best treatment of ISSHL, a highly debilitating condition.

References

- 1 National Institute on Deafness and Other Communication Disorders. *Sudden deafness*. [cited 2016 May 25]. Available from: <http://www.nidcd.nih.gov/health/Pages/sudden.aspx>.
- 2 Kaya H, Koc AK, Sayin I, Gunes S, Altintas A, Yegin Y, et al. Vitamins A, C, and E and selenium in the treatment

- of idiopathic sudden sensorineural hearing loss. *Euro Arch Otorhinolaryngol.* 2015;272:1119-25.
- 3 Stobbs N, Goswamy J, Ramamurthy L. How are we managing sudden sensorineural hearing loss in the United Kingdom?: our experience. *Clin Otolaryngol.* 2014;39:385-8.
 - 4 Seggas I, Koltsidopoulos P, Bibas A, Tzonou A, Sismanis A. Intratympanic steroid therapy for sudden hearing loss: a review of the literature. *Otol Neurotol.* 2011;32:29-35.
 - 5 Stachler RJ, Chandrasekhar SS, Archer SM, Rosenfeld RM, Schwartz SR, Barrs DM, et al. Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg.* 2012;146:S1-35.
 - 6 Murphy-Lavoie H, Piper S, Moon RE, Legros T. Hyperbaric oxygen therapy for idiopathic sudden sensorineural hearing loss. *Undersea Hyperb Med.* 2012;39:777-92.
 - 7 De Kleyen A. Sudden complete or partial loss of function of the octavus system in apparently normal persons. *Acta Otolaryngol.* 1944 32:407-29.
 - 8 Stew BT, Fishpool SJ, Williams H. Sudden sensorineural hearing loss. *Br J Hosp Med (London).* 2012;73:86-9.
 - 9 Mattox DE, Simmons FB. Natural history of sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol.* 1977;86:463-80.
 - 10 Schuknecht HF, editor. *Pathology of the ear.* Philadelphia; Lea and Febiger; 1993. p. 524-9.
 - 11 Kumar A, Sinha A, Al-Waa AM. Resolution of sudden sensorineural hearing loss following a roller coaster ride. *Indian J Otolaryngol Head Neck Surg.* 2011;63:104-6.
 - 12 Chau JK, Cho JJ, Fritz DK. Evidence-based practice: management of adult sensorineural hearing loss. *Otolaryngol Clin North Am.* 2012;45:941-58.
 - 13 Sara SA, Teh BM, Friedland P. Bilateral sudden sensorineural hearing loss: review. *J Laryngol Otol.* 2014;128(Suppl 1):S8-15.
 - 14 Wu CM, Lee KJ, Chang SL, Weng SF, Lin YS. Recurrence of idiopathic sudden sensorineural hearing loss: a retrospective cohort study. *Otol Neurotol.* 2014;35:1736-41.
 - 15 Huy PT, Sauvaget E. Idiopathic sudden sensorineural hearing loss is not an otologic emergency. *Otol Neurotol.* 2005;26:896-902.
 - 16 Awad Z, Huins C, Pothier DD. Antivirals for idiopathic sudden sensorineural hearing loss. *Cochrane Database of Systematic Reviews.* 2012;8:CD006987. doi: 10.1002/14651858.CD006987.pub2.
 - 17 Wei BP, Stathopoulos D, O'Leary S. Steroids for idiopathic sudden sensorineural hearing loss. *Cochrane Database of Systematic Reviews.* 2013;7:CD003998. doi: 10.1002/14651858.CD003998.pub3.
 - 18 Muzzi E, Zennaro B, Visentin R, Soldano F, Sacilotto C. Hyperbaric oxygen therapy as salvage treatment for sudden sensorineural hearing loss: review of rationale and preliminary report. *J Laryngol Otol.* 2010;124:e2.
 - 19 Bennett MH, Kertesz T, Perleth M, Yeung P, Lehm JP. Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and tinnitus. *Cochrane Database of Systematic Reviews.* 2012;10:CD004739. doi: 10.1002/14651858.CD004739.pub4.
 - 20 Cekin E, Cincik H, Ulubil SA, Gungor A. Effectiveness of hyperbaric oxygen therapy in management of sudden hearing loss. *J Laryngol Otol.* 2009;123:609-12.
 - 21 Kau RJ, Sendtner-Gress K, Ganzer U, Arnold W. Effectiveness of hyperbaric oxygen therapy in patients with acute and chronic cochlear disorders. *ORL J Otorhinolaryngol Relat Spec.* 1997;59:79-83.
 - 22 Drenjancevic I, Kibel A. Restoring vascular function with hyperbaric oxygen treatment: recovery mechanisms. *J VascRes.* 2014;51:1-13.
 - 23 Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. *Plast Reconstr Surg.* 2011;127(Suppl 1):131S-41S.
 - 24 Plafki C, Peters P, Almeling M, Welslau W, Busch R. Complications and side effects of hyperbaric oxygen therapy. *Aviat Space Environ Med.* 2000;71:119-24.
 - 25 von Elm E, Altman DG, Egger M, Pocock SJ, Gotsche PC, Vandenbroucke JP, et al. The Strengthening of Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet.* 2007;370(9596):1453-7.
 - 26 Clark JG. Uses and abuses of hearing loss classification. *ASHA.* 1981;23:493-500.
 - 27 Caers D, Lafère P, Vanhoutte D, Germonpré P. Retrospective analysis of 101 deafness cases treated with hyperbaric oxygen therapy. In: Kot J, editor. *37th Annual Meeting of the European Underwater and Baromedical Society (EUBS);* 2010 24th-27th August, 2011. Gdansk: EUBS; 2011.
 - 28 Fetterman BL, Saunders JE, Luxford WM. Prognosis and treatment of sudden sensorineural hearing loss. *Am J Otol.* 1996;17:529-36.
 - 29 Topuz E, Yigit O, Cinar U, Seven H. Should hyperbaric oxygen be added to treatment in idiopathic sudden sensorineural hearing loss? *Eur Arch Otorhinolaryngol.* 2004;261:393-6.
 - 30 Meijer AG, Wit HP, TenVergert EM, Albers FW, Muller Kobold JE. Reliability and validity of the (modified) Amsterdam Inventory for Auditory Disability and Handicap. *Int J Audiol.* 2003;42:220-6.
 - 31 Hawthorne G, Hogan A. Measuring disability-specific patient benefit in cochlear implant programs: developing a short form of the Glasgow Health Status Inventory, the Hearing Participation Scale. *Int J Audiol.* 2002;41:535-44.
 - 32 Bessereau J, Tabah A, Genotelle N, Francais A, Coulange M, Annane D. Middle-ear barotrauma after hyperbaric oxygen therapy. *Undersea Hyperb Med.* 2010;37:203-8.
- Conflicts of interest:** nil
- Submitted:** 20 February 2016; revised 02 June and 26 July 2016
Accepted: 07 August 2016
- Susannah Sherlock^{1,4}, Kenneth Thistlethwaite¹, Mohsina Khatun², Christopher Perry³, Alexis Tabah^{1,4}
- ¹ Hyperbaric Medicine Unit, Royal Brisbane and Women's Hospital (RBWH), Brisbane, Queensland, Australia
² School of Public Health, Faculty of Medicine and Biomedical Sciences, The University of Queensland, Brisbane, Australia
³ Queensland Institute of Medical Research, Brisbane
⁴ Burns, Trauma, and Critical Care Research Centre, RBWH and University of Queensland
- Address for correspondence:**
 Dr Susannah Sherlock
 Royal Brisbane and Women's Hospital
 Hyperbaric Medicine Unit
 Butterfield Street
 Herston QLD 4026
 Australia
 susannah.sherlock@health.qld.gov.au