Enriched acoustic environment rescales auditory sensitivity

Arnaud Jean Noreña[a] and Sylviane Chery-Croze[b]

[a]Laboratory of Integrative and Adaptative Neurobiology, University of Provence/St Charles, Pôle 3C, Marseille and [b]Neurosciences and Sensory System Laboratory, University of Claude Bernard, Lyon, France

Correspondence to Arnaud Jean Noreña, PhD, Université de Provence/CNRS, Centre St Charles, Pôle 3C-Case B, 3, Place Victor Hugo, F-13331 Marseille Cedex 03, France
Tel: + 33 0 488 57 68 63; fax: + 33 0 488 57 68 42; e-mail: arnaud.norena@univ-provence.fr

Received 11 April 2007; accepted 13 April 2007

Loudness perception may be controlled by a central gain, possibly dependent on the mean level of the acoustic environment. Owing to hearing loss, for instance, a decrease in sensory inputs could increase this central gain and cause an auditory hypersensitivity or hyperacusis. According to this model, individuals with hyperacusis, provided with an enriched acoustic environment specifically designed to compensate for the decrease in sensory inputs, should show an improvement in their hyperacusis. This study showed that such an enriched acoustic environment indeed decreased auditory hypersensitivity: stimuli initially considered as being too loud became comfortable after a few weeks of acoustic stimulation. Therefore, this original approach could provide a solution to the problem of hyperacusis. *NeuroReport* 18:1251–1255 © 2007 Lippincott Williams & Wilkins.

Keywords: central gain, hearing aid, hearing loss, inhibition, plasticity, recruitment, tinnitus

Introduction

Loudness is the attribute of auditory sensation in terms of which 'sounds can be ordered on a scale extending from quiet to loud' [1]. Loudness increases as a function of intensity level from absolute threshold to loudness discomfort level (LDL), when the loudness starts to become 'too loud' [1,2]. In the presence of hearing loss, loudness has been shown to grow at an unusually rapid rate as the LDL is not changed. This is referred to as loudness recruitment [1,3]. In some cases, the LDL is reduced and individuals tend to complain of bothersome loudness perception at moderate levels. This is referred to as auditory hypersensitivity or hyperacusis [4–6].

The prevalence of hyperacusis is around 8% in the general population [7]. Interestingly, 86% of patients with hyperacusis also report having tinnitus [6] and 40% or more of patients with tinnitus also report having hyperacusis [8–12]. The frequent co-occurrence of these two 'aberrant' perceptions suggests that these two symptoms may share common origins. Importantly, hyperacusis may severely impair quality of life and is sometimes considered as being more troublesome than tinnitus [6].

It has recently been shown that modifying sensory inputs over 2 weeks induced changes in loudness perception. Indeed, decreasing (with earplugs) or increasing (stimulation with broadband noise) sensory inputs induced an increase or a decrease in auditory sensitivity, respectively [13]. This suggests that loudness perception is highly plastic and could be controlled by a central gain, depending on the mean level of the sensory environment. In this context, hearing loss, which causes a decrease in sensory inputs to the central auditory system, could cause an abnormal increase in central gain and consequently an auditory hypersensitivity (most patients with hyperacusis present a hearing loss [6,11,14]).

According to this model, compensating for the decrease in sensory inputs owing to hearing loss could decrease the central gain and consequently reduce auditory hypersensitivity [15–18]. This study attempts to test this approach in patients reporting hyperacusis.

Methods

Participants

Eight individuals (Table 1) complaining of bilateral annoyance caused by ordinary sounds, in other words auditory hypersensitivity or hyperacusis, were involved in the study. The French Ethics Committee approved this study, conducted with the understanding and the written consent of each participant.

Psychoacoustic measures

Absolute thresholds (Table 1) were obtained between 500 and 16 kHz in half octave steps with a bracketing procedure. Loudness functions at different frequencies were estimated using a modified version of the method called loudness growth in one-half octave band (LGOB) [2,19]. The method consisted of categorizing loudness of pure tones whose intensity levels ranged from absolute thresholds to the 'too loud' level. The 'too loud' level was first obtained by increasing the intensity level in 5-dB steps from a moderate level [60 dB sound pressure level (SPL) or lower in very sensitive individuals] to the 'too loud' level. This is the level reached when the stimulus would be intolerable if

Table 1 Absolute thresholds in left (first line in each row) and right (second line in each row) ears, and demographic aspects of participants

Participant	Age (years)	Sex	Aetiology	Absolute thresholds (dB SPL) 0.5 0.7 1 1.4 2 2.8 4 5.6 8 11.3 16 (kHz)
1	38	F	Unknown	22 13 8 5 27 16 4 14 44 57 100
				18 9 13 7 26 12 2 12 39 47 99[a]
2	43	M	Noise trauma	24 19 12 7 11 9 27 47 43 51 94
				22 17 12 13 16 16 26 48 62 58 89[a]
3	30	F	Antibiotics (?) and stress (?)	25 17 11 13 10 4 16 22 24 36 84[a]
				21 15 14 11 9 7 16 16 29 19 80
4	59	F	Exposed to noise and stress (?)	18 13 17 9 18 7 2 7 40 72 100
				16 19 12 22 22 13 7 13 36 72 95[a]
5	28	M	Noise trauma	15 5 9 12 10 17 10 31 34 40 96[a]
				11 8 6 13 8 3 17 45 54 38 95
6	48	M	Noise trauma	23 13 15 10 18 26 20 42 44 67 100[a]
				22 13 13 10 22 17 18 17 38 71 100
7	31	M	Stress (?)	13 6 5 12 12 −1 1 18 21 21 48[a]
				13 6 5 9 11 −1 2 14 22 22 43
8	60	F	Unknown	12 17 18 21 12 12 6 27 59 74 100[a]
				27 20 14 12 12 12 3 13 37 58 100

F, female; LGOB, loudness growth in one-half octave band; M, male; ?, possible (but uncertain) aetiology.
[a]Ear tested in the LGOB task.

presented for a longer duration. The intensity step used in the LGOB test was then obtained by dividing the difference between the 'too loud' level and the absolute threshold by 14. As a result, 15 different intensities (three repetitions each) were then presented to the participants. The participants had to judge the loudness of the stimulus presented as being 'inaudible', 'very soft', 'soft', 'OK' (medium level), 'loud', 'very loud' or 'too loud'. Once the test is completed at a given frequency, another frequency was tested (up to six frequencies were tested).

Psychoacoustic tests were carried out at each test session, namely before the beginning of the treatment (s0), 2 weeks (s2), 5 weeks (s5), 10 weeks (s10) and 15 weeks (s15) from the beginning of the acoustic stimulation. After 15 weeks, the participants stopped listening to the acoustic stimulus. An additional test session (s + 1 m) was carried out 1 month after the end of the treatment. This test session was used as a control with acoustic stimulation removed for a month, we expect that auditory sensitivity should tend to return towards the pretreatment level (s0). The participants also answered two questionnaires aimed at estimating the auditory sensitivity [12,20]. For the multiple-activity scale of hyperacusis (MASH), participants had to provide a score (from 0 to 10) to indicate the level of annoyance caused by sounds in different activities (concert, work, driving a car, etc.). The mean MASH score was calculated by dividing the total score by the number of relevant activities [12]. In questionnaires from Khalfa *et al.* [20], participants had to answer (among four possibilities: never, sometimes, moderately, often; scores are 0, 1, 2, 3, respectively) 14 sentences or questions (example: 'Do you use earplugs to reduce your perception of noise?'). The score is simply the sum of all the answers.

Generation of the 'therapeutic signal': 'equalizing' auditory inputs towards the centres

The acoustic signal was generated individually from the audiogram of each participant. Pure tones of different frequencies (frequency step was 1/64th octave) were generated and the amplitude of each pure tone was 'weighted' [factor A in equations (1) and (2)] as a function of the audiogram: all frequencies were presented at equal level in dB SL (sensory level). Each pure tone of frequency F and as a function of time t was generated as follows:

$$\text{Signal}(F,t) = A(F) * \sin(2 * \text{pi} * F * t) * \text{env}(t), \qquad (1)$$

where $A(F)$ is a weighting factor aimed at 'equalizing' the level of pure tones and env(t) is the envelope (5-ms rise and fall linear ramp).

The weighting factor $A(F)$ was obtained as follows:

$$\text{A(F)} = 10\text{^}[((\text{AT(F)} - \text{Level\_min}) - (\text{Level\_max} - \text{Level\_min}))/20], \qquad (2)$$

where AT(F) is the absolute threshold (in dB SPL) as a function of F Level_max corresponds to the maximal hearing loss (chosen to be less than 75 dB SPL) and Level_min is the absolute threshold (in dB SPL) corresponding to the low cut-off frequency of the hearing loss in a given participant. One verifies that when AT(F)=Level_max, $A(F)$=1.

The enriched acoustic environment (EAE) was composed of pure tones of 100-ms duration with an average interstimulus interval of 100 ms (±10 ms). An entire sequence of pure tones was generated, that is frequency ranged from the cut-off frequency of the hearing loss (determined visually) to the frequency corresponding to Level_max [equation (2)], with 1/64th octave step. The EAE was then composed of pure tones at all these frequencies, weighted as a function of A(F), and presented in random order. The acoustic stimulus was generated in Matlab, saved as a .wav file and was burnt on a CD. The CD, a CD player (Panasonic, SL-SX340) and headphones (Panasonic, RP-HS55) were given to the participants with listening instructions. The participants were asked to listen daily to the EAE for a few hours and, most importantly, at a just audible level. At the end of the experiment, all participants reported having listened to the EAE at least 1–3 h every day on average.

Statistical tests

Loudness functions derived from LGOB carried out at frequencies below, equal to or above the cut-off frequency of hearing loss were grouped into three frequency bands, namely Flow, cut-off frequency of hearing loss (COF), Fhigh, respectively. A Mann–Whitney test was used to test the potential effects of the EAE (between s0 and s2, s0 and s15, and s15 and s + 1m) in each frequency band and for each loudness response category. The Wilcoxon signed-rank test was used to test the effects of the stimulation over test sessions on the scores derived from the questionnaires. For all tests, the threshold of significance (initially, P=0.05) was adjusted according to the Bonferroni correction (0.05/number of comparisons).

Results

Averaged loudness functions obtained from our participants at the pretreatment baseline session (s0) are shown in Fig. 1.

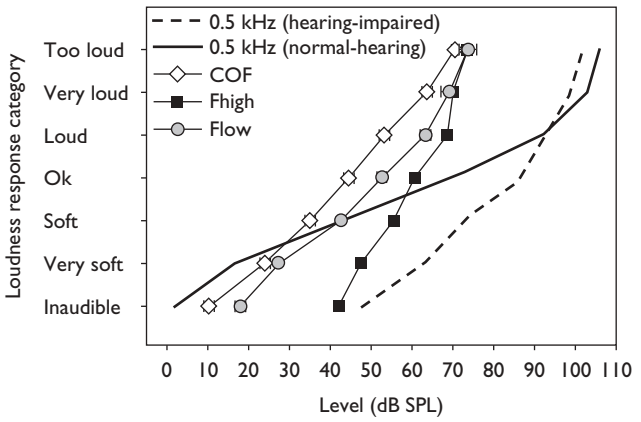

Fig. 1 Loudness functions averaged over all participants (with hyperacusis) tested in this study at the pretreatment baseline session in Flow (grey circles), COF (white diamonds) and Fhigh (black squares) frequency bands (±SEM). Loudness functions in a normal-hearing participant (thick continuous line, data adapted from Ref. [2]) and hearing-impaired participant (dashed line, data adapted from Ref. [19]) are also shown.

Averaged loudness functions from two participants without hyperacusis, a normal-hearing participant (thick continuous line, data adapted from Ref. [2]) and a hearing-impaired participant (dashed line, data adapted from Ref. [19]), obtained from the LGOB test (at 0.5 kHz), were added to provide comparative data. Our participants present, on average, a relatively low LDL (around 70 dB SPL) testifying to their high auditory sensitivity in the three frequency bands. On the other hand, the LDL in the two participants without hyperacusis is around 100 dB SPL, irrespective their absolute thresholds. Data from the hearing-impaired participant (Fig. 1, dashed line) show an example of what is called 'loudness recruitment', which could be defined as an increase in the slope of the loudness-growth curve [1,3,19].

Figure 2 shows the mean loudness judgements at s2, s15 and s+1m in separate panels for Flow, COF and Fhigh frequency bands relative to the corresponding pretreatment baseline loudness judgements (s0) (data for s5 and s10 are not shown for clarity).

The EAE induced a dramatic decrease in auditory sensitivity over the course of the treatment in all of the three frequency bands. Typically, across all loudness response categories, listeners needed more intense tones to achieve the same loudness judgements as compared with pretreatment baseline judgements. This decrease in auditory sensitivity is statistically significant as soon as 2 weeks of acoustic stimulation (s2) in the COF [category 'OK', $P<(0.05/7 \text{ categories})=0.007$] and Fhigh (categories 'OK', 'loud' and 'very loud', $P<0.007$) frequency bands. The LGOB test has been shown to give highly reliable results [2]. It is then unlikely that 'desensitization' owing to repetitive testing caused these effects on loudness perception (also see Discussion). At s15, the decrease in auditory sensitivity relative to s0 was maximal and statistically significant in all frequency bands and all loudness categories from 'soft' to 'too loud' ($P<0.007$). Finally, participants were tested 1 month after the end of the treatment. According to our model, in the absence of additional stimulation for compensating the decrease in sensory inputs owing to hearing loss, the auditory sensitivity should ultimately return to the baseline level (provided that the 'recovery' period is sufficiently long). Our results are in broad accordance with the model as the sound levels needed to achieve the same loudness judgements at s+1m, compared with judgements at s15, were significantly decreased in the COF (category 'very loud', $P<0.007$) and Fhigh (categories 'loud' and 'very loud', $P<0.007$) frequency bands. Nevertheless, the levels at s+1m are still higher when compared with the levels at s0 (all frequency bands and categories from 'soft' to 'very loud', $P<0.007$).

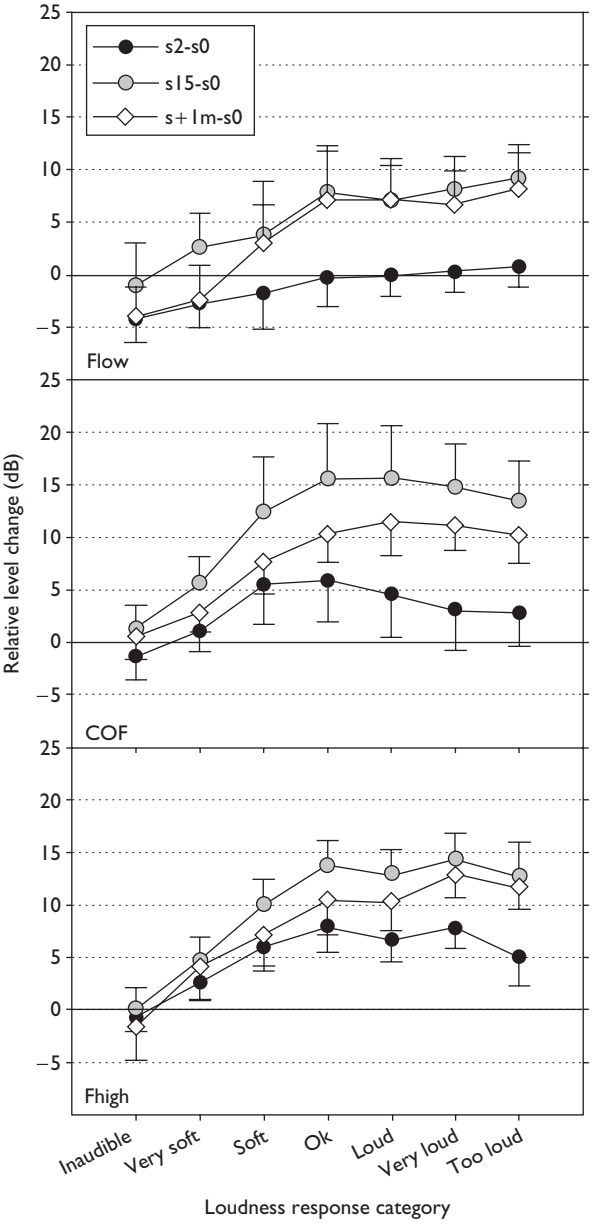

Fig. 2 Averaged loudness levels at s2 (black circles), s15 (grey circles) and s+1m (white diamonds) in Flow (upper panel), COF (middle panel) and Fhigh (lower panel) frequency bands relative to the pretreatment baseline session (±SEM), as a function of loudness response categories (data at s5 and s10 are not shown). A positive value indicates a reduced auditory sensitivity, that is, participants needed more intense tones to achieve the same loudness judgements as compared with the pretreatment baseline session (s0).

The scores derived from the questionnaires decreased in all participants over the course of the treatment. The scores

from the MASH questionnaire [12] were significantly decreased at s15 (averaged score: 4, $P<0.025$) and s+1m (average score 3.6, $P<0.025$) relative to s0 (average score: 5.23). The scores from Khalfa *et al.*'s questionnaire [20] were also significantly decreased at s+1m (average score: 26, $P<0.025$) relative to s0 (averaged score: 33.5).

Discussion

First, this study shows that participants reporting auditory hypersensitivity present low LDL over a broad frequency band. As stated in the Introduction, loudness perception could be controlled by a central gain, which is thought to be dependent on the mean level of the sensory environment. As hearing loss is confined to the high-frequency region, the auditory hypersensitivity over a broad frequency band suggests that the central gain is broadly tuned to frequencies. This is consistent with the decrease in central inhibition [21] and the increase in neural responses over a broad frequency range [17,18,22,23] induced by hearing loss. The central inhibition, known to vary with the amount of sensory inputs [21], could play a major role in controlling the central gain.

Second, this study shows that an EAE confined to the frequency range of hearing loss induced a decrease in auditory hypersensitivity (estimated from the LGOB test and questionnaires) over a broad frequency band. At first sight, these changes in loudness perception could be accounted for by a rescaling of participants' behavioural criteria (or desensitization) for judging loudness owing to repetitive testing. LGOB has, however, been proven to give high reliable results [2]. The quick (after 2 weeks of stimulation) and frequency-dependent effect of the EAE cannot then be accounted by a desensitization effect. Finally, desensitization does not account for the significant increase in auditory sensitivity between s15 and s+1m. Instead, the large decrease in auditory sensitivity induced by the EAE, is more parsimoniously explained by a decrease in the adaptive central gain [13,16,17]. Once again, the decrease in auditory sensitivity at remote frequencies from the stimulated frequency range suggests that the adaptive central gain is broadly tuned to frequencies. This is in broad agreement with a recent study in which normal-hearing participants were fitted with either earplugs (simulating the decrease in sensory inputs owing to hearing loss) or low-level noise generator for 2 weeks [13]. The earplugs and low-level noise generator respectively induced an increase and a decrease in auditory sensitivity over a broad frequency band.

Finally, chronic stimulation with broadband noise has been shown to reduce auditory hypersensitivity [9,10, 12,24]. Nevertheless, stimulation with broadband noise has been reported to be slow and has limited effects for providing a solution to the problem of hyperacusis [12]. This further emphasizes the importance of providing an EAE equalizing sensory inputs, that is compensating the reduced sensory inputs owing to hearing loss.

Recently, a passive acoustic stimulation in the frequency range of noise-induced hearing loss has been shown to prevent the central changes usually induced after a noise trauma in cats [16,17]. This suggests that in compensating the decrease in sensory inputs related to hearing loss, the EAE may have restored central inhibition and, consequently, normal neural responses. In this study, the EAE provided to participants possibly restored central inhibition and decreased the central gain, which could have decreased auditory sensitivity.

Conclusion

This study shows that an acoustic stimulation confined to the frequency range of hearing loss reduces auditory hypersensitivity over a broad frequency band. The general approach of providing adapted EAE to reduce the central gain of the auditory system could be applied in all participants experiencing auditory hypersensitivity.

Acknowledgements

This study was supported by 'La fondation pour la recherche sur le cerveau' and the 'Agence Nationale pour la Recherche'.

References

1. Moore BCJ. *An introduction to the psychology of hearing*. 3rd ed. London: Academic Press; 1995.
2. Rasmussen AN, Olsen SO, Borgkvist BV, Nielsen LH. Long-term test-retest reliability of category loudness scaling in normal-hearing subjects using pure-tone stimuli. *Scand Audiol* 1998; **27**:161–167.
3. Yost WA. *Fundamentals of hearing. An introduction*. London: Academic Press; 2006.
4. Vernon JA. Pathophysiology of tinnitus: a special case – hyperacusis – and a proposed treatment. *Am J Otol* 1987; **8**:201–202.
5. Baguley DM. Hyperacusis. *J R Soc Med* 2003; **96**:582–585.
6. Anari M, Axelsson A, Eliasson A, Magnusson L. Hypersensitivity to sound– questionnaire data, audiometry and classification. *Scand Audiol* 1999; **28**:219–230.
7. Andersson G, Lindvall N, Hursti T, Carlbring P. Hypersensitivity to sound (hyperacusis): a prevalence study conducted via the internet and post. *Int J Audiol* 2002; **41**:545–554.
8. Fabijanska A, Rogowski M, Bartnik G, Skarzynski H. Epidemiology of tinnitus and hyperacusis in Poland. In: Hazell, J, editor. Proceedings of the VIth Tinnitus Seminar. Cambridge, UK; 1999. pp. 569–571.
9. Gold SL, Frederick EA, Formby C. Shifts in dynamic range for hyperacusis patients receiving tinnitus retraining therapy (TRT). In: Hazell J, editor. Proceedings of the VIth tinnitus seminar. Cambridge, UK; 1999. pp. 297–301.
10. McKinney CJ, Hazell JWP, Graham RL. Changes in loudness discomfort level and sensitivity to environmental sound with habituation based therapy. In: Hazell J, editor. Proceedings of the VIth Tinnitus Seminar. Cambridge, UK; 1999. pp. 499–501.
11. Pilgramm M. Rychlick R, Lebisch H, Siedentop H, Goebel G, Kirchhoff D. Tinnitus in the Federal Republic of Germany: a representative epidemiological study. In: Hazell J, editor. Proceedings of the VIth Tinnitus Seminar, Cambridge, UK; 1999. pp. 64–67.
12. Dauman R, Bouscau-Faure F. Assessment and amelioration of hyperacusis in tinnitus patients. *Acta Otolaryngol* 2005; **125**:503–509.
13. Formby C, Sherlock LP, Gold SL. Adaptive plasticity of loudness induced by chronic attenuation and enhancement of the acoustic background. *J Acoust Soc Am* 2003; **114**:55–58.
14. Goldstein B, Shulman A. Tinnitus-hyperacusis and the loudness discomfort level test – A preliminary report. *Int Tinnitus J* 1996; **2**:83–89.
15. Norena A, Micheyl C, Chery-Croze S, Collet L. Psychoacoustic characterization of the tinnitus spectrum: implications for the underlying mechanisms of tinnitus. *Audiol Neurootol* 2002; **7**:358–369.
16. Norena AJ, Eggermont JJ. Enriched acoustic environment after noise trauma reduces hearing loss and prevents cortical map reorganization. *J Neurosci* 2005; **25**:699–705.
17. Norena AJ, Eggermont JJ. Enriched acoustic environment after noise trauma abolishes neural signs of tinnitus. *Neuroreport* 2006; **17**:559–563.
18. Norena AJ, Eggermont JJ. Changes in spontaneous neural activity immediately after an acoustic trauma: implications for neural correlates of tinnitus. *Hear Res* 2003; **183**:137–153.

19. Allen JB, Hall JL, Jeng PS. Loudness growth in 1/2-octave bands (LGOB)–a procedure for the assessment of loudness. *J Acoust Soc Am* 1990; **88**:745–753.
20. Khalfa S, Dubal S, Veuillet E, Perez-Diaz F, Jouvent R, Collet L. Psychometric normalization of a hyperacusis questionnaire. *ORL J Otorhinolaryngol Relat Spec* 2002; **64**:436–442.
21. Milbrandt JC, Holder TM, Wilson MC, Salvi RJ, Caspary DM. GAD levels and muscimol binding in rat inferior colliculus following acoustic trauma. *Hear Res* 2000; **147**:251–260.
22. Salvi RJ, Wang J, Ding D. Auditory plasticity and hyperactivity following cochlear damage. *Hear Res* 2000; **147**:261–274.
23. Wang J, Ding D, Salvi RJ. Functional reorganization in chinchilla inferior colliculus associated with chronic and acute cochlear damage. *Hear Res* 2002; **168**:238–249.
24. Wolk C, Seefeld B. The effects of managing hyperacusis with maskers (noise generators). In: Hazell J, editor. Proceedings of the VIth Tinnitus Seminar, Cambridge, UK; 1999. pp. 512–514.