Decreased sound tolerance: predisposing factors, triggers and outcomes after TRT

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An analysis of 187 cases of decrease sound tolerance (DST) reveals a variety of predisposing (trigger) factors. These include exposure to loud sounds, fear about the effects of sound on the ear and the hearing, and the development of more non-specific anxieties relating to environmental threats. Diagnosis by Jastreboff category, and the relative importance and pathophysiology of hyperacusis, misophonia and phonophobia are discussed. Following treatment in a tinnitus retraining therapy programme loudness discomfort levels reached normal levels in 44.8% by the 6 month visit, 51.4% by the 15 month visit and 60.4% by the 25th month visit .The mean number of life factors / activities affected by hyperacusis fell from 3.5 at the 1st visit to 1.1 at the 15 month visit. Trigger factors did not affect outcome.

Introduction

Data was collected from 187 consecutive patients with a primary diagnosis of decreased sound tolerance (DST) who attended the Tinnitus and Hyperacusis Centre, London. Patients were referred by ENT colleagues, primary health care physicians, and a number by self-referral from our website (www.tinnitus.org). The majority of patients came with the expectation that they would receive tinnitus retraining therapy (TRT) based on the Jastreboff neurophysiological model. Data was recorded on a questionnaire during a structured interview. All patients had a full otological examination including microscopy of the ears, followed by pure tone audiometry, loudness discomfort levels at octave intervals between 125 Hz and 8 kHz but including 6kHz, impedance audiometry and stapedius reflex measurement. Transient evoked otoacoustic emissions and distortion products were also measured together with an Audioscan, or Bekesy audiometry, though these results are not reported here.

All patients were categorised according to whether the symptoms were enhanced by exposure to sound for an extended period in excess of 24 hours - Jastreboff category 4, or not - Jastreboff category 3 [1]. Patients were also identified as to a whether the primary problem was one of hyperacusis, misophonia or phonophobia (see discussion). Only three patients with severe phonophobia did not, in addition, exhibit hyperacusis . All patients exhibited misophonia. Tinnitus was also present in the majority of patients, although this was never the dominant symptom. Only 3.6% had no awareness of tinnitus at anytime and 9% of patients had tinnitus awareness without any tinnitus distress. Tinnitus severity was assessed by the percentage of waking hours either awareness of tinnitus, or distress from tinnitus was present. DST and tinnitus were assessed by the number of life factors affected, and hyperacusis (as a component of DST) by loudness discomfort levels (LDL).

Data was collected at each of 4 visits: The average visit intervals were as follows 2nd visit, 6.23 months(n=124), 3rd visit 15.23 months visit (n=76) and 4th visit 25.56months (n=44). The total number of visits per patient varied from two to seven, according to need, and according to the TRT protocol. Some patients needed additional early visits, but data collected is not presented. Patients who had defaulted during the study were contacted by letter and requested to return by stamped addressed envelope a simple two-question form indicating whether the symptoms had changed, and in which direction.

All data was transferred to a computer database and analysed by SPSS.

Treatment Protocol

All patients were treated according to a strict TRT protocol appropriate for Jastreboff category 3 and 4 [2,1]. Treatment involved sessions of teaching or training, when the Jastreboff model was presented in order to explain and demystify the patient's experience. The first session when the model was presented was always recorded on cassette tape for the patient to review. Each patient was subsequently checked for knowledge and understanding of the model, and further teaching and counselling was given as necessary, until understanding was complete. All patients were given careful instruction on the gradual reduction of the use of earplugs, or avoiding sound (except in potentially cochlea-damaging situations), with a gradual return to a normal sound environment.

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	%
1. Acute sound exposure	11.23
2. Chronic sound exposure	10.16
3. Head injury	3.21
4. Injury other than head	2.14
5. Recent onset tinnitus	21.39
6. Stress	17.11
7. Longterm dislike of sound	10.70
8. Quiet environment	4.81
9. Failed ear surgery/middle ear disease	1.07
10. Other illness/surgery	3.21
11. Hydrops	3.74
12. Ear toilet	1.07
13.Highly phobic/many other phobias	4.81
14. Ear infection/ear pathology	3.74
15. Acoustic neuroma	0.53
16. None	1.07

Table 1 Predisposing or trigger factors in DST. Only the dominant predisposing cause is recorded for each patient.

All patients (except those experiencing phonophobia exclusively) were treated with binaural wearable sound generators, which in this study were post-aural instruments fitted to personal open moulds. Patients were instructed on the specific use of these instruments to reduced sensitivity to sound. Instruments were used daily for a minimum of 50% of waking hours, at a clearly audible level regardless of sound environment. The volume of the instruments was to be gradually increased in situations where normally distressing levels of environmental sound would be present, never exceeding a level of discomfort from the wearable sound generators. Volume levels were then reduced or reset in quiet surroundings. When tinnitus was present, instruments were set to avoid tinnitus suppression, if at all possible. However, first priority was given to reducing sound sensitivity, with a view to achieving habituation of tinnitus at a later date.

A technique of sound enrichment was used with constant digitally produced nature sound sources used on a 24-hour basis. The criteria for speed-of-change in the sound environment was dictated by whether or not aversive symptoms occurred during treatment. If this happened and sensitivity to sound increased, instead of decreased, then patients were instructed to begin that particular phase of sound reintroduction again, this time more slowly.

Category 4 patients were treated similarly, but with a particularly gentle and slow regime, involving very gradual increase of sound generator volume from zero, and a very cautious reintroduction of external sounds to which aversions were present, over a period of months. These patients experience worsening of their symptoms after more than 24 hours, or a good night's sleep, when exposed to ordinary levels of environmental sound. This is due to a kindling, and/or winding up effect [1], and therefore any change in sound reaching the ears must be extremely gradual to avoid worsening symptoms still further.

Three patients only, where severe phonophobia was present in the absence of any hyperacusis, were treated a) by specific counselling about the sounds feared, being harmless b) sound enrichment, and c) a very gradual reintroduction of sounds feared. Instruments were not used.

Results

187 patients took part with an average age of 45.34 years (SD 14.51). The sex difference was males 50.3%, females 49.7%.

Predisposing / trigger factors

A list of all pre-disposing factors for DST is given in Table 1. Only one possible pre-disposing cause was given for each patient. Where multiple factors were present, only the dominant one was recorded. In some cases there was a very clear and obvious event, which preceded the onset of DST. In others cases, the onset of symptoms was more gradual. Then environmental, cognitive, and behavioural factors were taken into account. Factors were then grouped in several ways to see

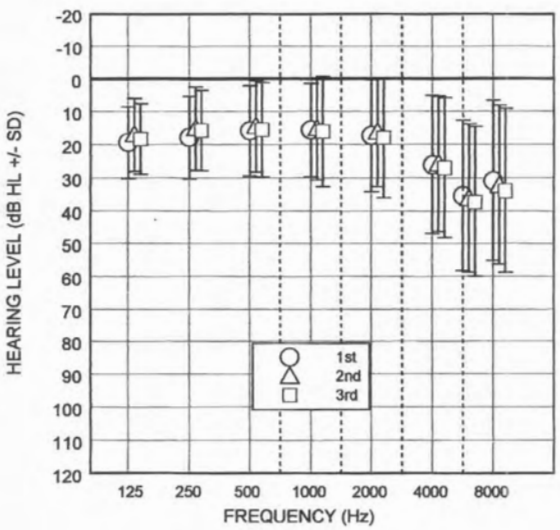

Figure 1 Audiometric thresholds for all cases averaged between ears with "1SD bars. Circle 1st visit, triangle 2nd visit, square 3rd visit

if relationships could be established with outcome measures, Musicians(14%) were also studied separately. All musicians described acute or chronic noise exposure and misophonia or phonophobia for musical instruments.

In Table 1, the frequency of predisposing or trigger factors is given. Factors 1, 2, 9, 11, 12, 14 and 15 were grouped as 'ear related factors', the remainder as 'non-ear related'.

Audiometry

The results of pure tone audiometry are shown in Figure 1 with standard deviations at each frequency. Results are given for all patients for each of the first 3 visits (circles, triangles and squares). It is clear that this population had hearing which was normal, or near normal for age. There was no statistical difference in hearing thresholds during the treatment period, or between right and left ears (paired t-tests were highly correlated; sig <0.001), so data from right and left ears were averaged. The average hearing thresholds for 500Hz ,1, 2kHz were

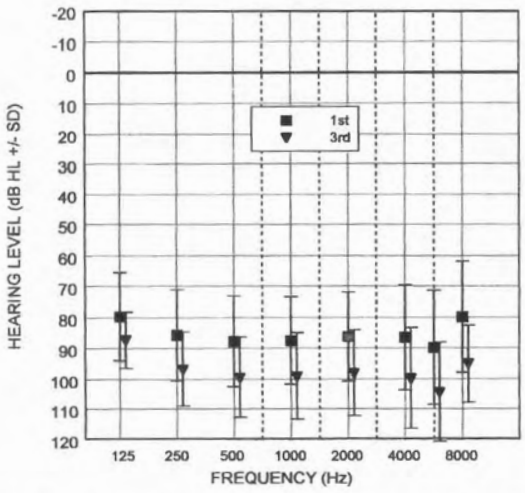

Figure 2a Average LDLs for all cases both ears. 1st visit (squares) 15 month visit (triangles) with "1SD bars.

as follows 15.86 dB (SD 13.68), 15.55 dB (SD 14.09), 17.31 dB (SD 16.94).

Loudness Discomfort Levels

LDLs at the start of treatment are shown by square symbols in Figure 2a, together with standard deviations. LDL values at the 3rd visit are indicated by inverted triangles. These measurements show the characteristic U-shaped configuration of loudness discomfort levels in hyperacusis.

The mean changes in LDL at each frequency between 1st and 2nd ,and 1st and 3rd visits are shown in Figure 2b. The average mean change between 1st and 2nd visit is 11.13 dB and between 1st and 3rd visit is 12.83 dB. Paired t-tests of LDL values measured from the 1st visit were highly significantly different (2nd visit t 9.12, df 114, $p<0.001$, 3rd visit t 8.28, df 67, $p<0.001$, 4th visit t 5.97, df 44, $p<0.001$). There was also significant improvement between 3rd and 4th visits (t 2.82, df 44, $p<0.01$). Results for 4th visit are not shown in the figure 2, as numbers were small.

Cumulative distributions of LDLs for all cases are shown in Figure 3 for each of the first 4 visits. Mean duration between first and subsequent visits in months was: 2nd visit 6.23 (SD 4.18), 3rd visit 15.23 (SD 10.54), 4th visit 25.56 (SD 14.90). Each data point is the mean loudness discomfort level averaged between ears and between 125 Hz and 8 kHz, at octave intervals, but including 6 kHz. Where loudness discomfort levels could not be measured because they exceeded machine maxima, a value of machine maximum plus 5 dB was used (125Hz – 95 dB, 250Hz-115 dB, 500Hz to 6kHz – 125 dB, 8kHz –105 dB).

Some patients who failed to attend for the second visit, attended at subsequent visits so the true number of non-attenders who were only seen on the first visit was 50.The majority of improvement in loudness discomfort levels was experienced between the first and second visit (Figure 3), although further improvement was experienced even up to the fourth visit (see above).

Previous research has indicated that the level for loudness discomfort for pure tones averaged over 125 Hz to 8 kHz is 104 dB. [3] Therefore 100 dB was taken as a conservative estimate for normal averaged loudness discomfort levels. Values below 100 dB where taken to indicate an element of hyperacusis. It is a necessary over-simplification to average LDL values over frequencies, and indeed the common configurations for hyperacusis is a U shaped curve as can be seen from Figure 2a. Many patients have symptoms of DST as a result of reduction of specific LDL frequencies only, particularly low frequencies and high frequencies.Patients with pure phonophobia did not have reduced LDLs as the auditory system was functioning normally.

From Figure 3, 15.3% of patients had normal LDLs according to our criteria at the start of treatment. In patients where phonophobia was dominant and hyperacusis less marked, LDLs were not significantly reduced. LDLs reached normal levels in 44.8% by the second visit, 51.4% by the third visit and 60.4% by the fourth visit. These changes were significant in each case ($p<.0.001$) – see above.

Questionnaire data

Hyperacusis impact was assessed by the number of life factors/activities which are interfered with. Improvement can be monitored by the reduction in life factors so

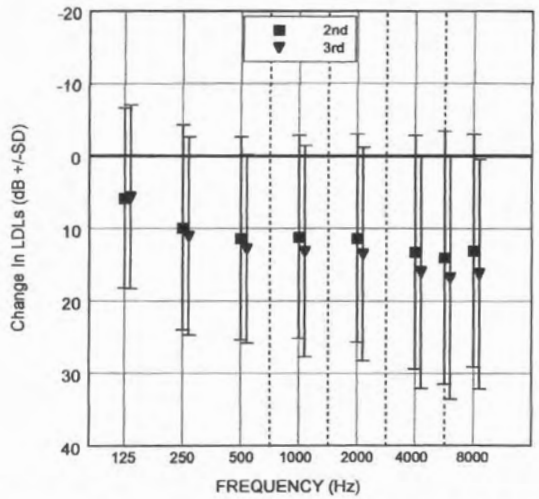

Figure 2b Change in averaged LDLs at 6month visit (squares) and 15 month visit (triangles) with "1SD bars.

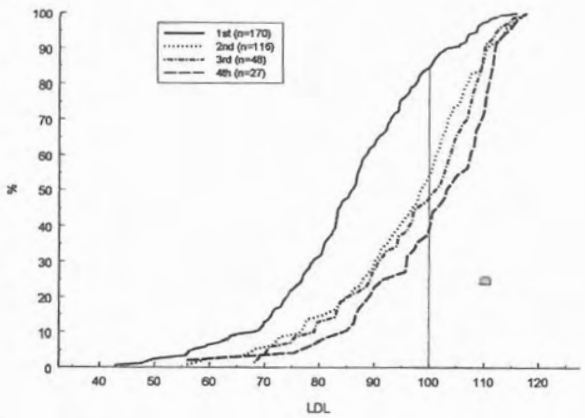

Figure 3 Cumulative distribution of all cases of LDLs (dB) averaged over all frequencies and between both ears for 1st, 2nd 3rd and 4th visits.

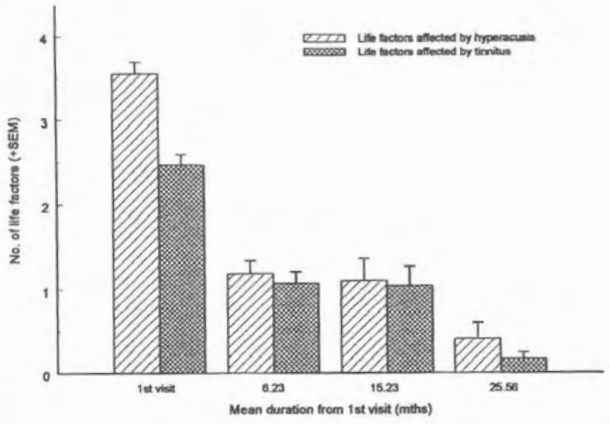

Figure 4a Mean number of life factors affected by tinnitus and hyperacusis at each visit showing SEM

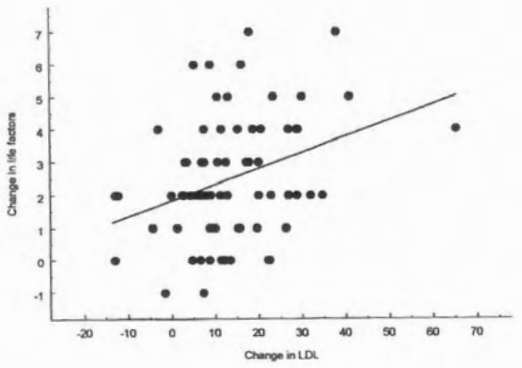

Figure 5 Change in averaged LDLs for all cases compared with change in life factors between 1st and 2nd visit. Pearson correlation = 0.33 p=0.004 n=74

affected [4]. In Figure 4a the number of life factors affected by both hypercusis and tinnitus are shown at each visit. The mean number of life factors for hyperacusis was 3.6 at the 1st visit and 1.1 at the 3rd visit (paired t-test t 10.39, df 56, p<0.001). For tinnitus fewer life factors were affected overall, as this was not the dominant symptom. However improvement was still significant between 1st and 3rd visits (paired t tests t 9.29, df 55, p<0.001).

Tinnitus was also measured by the percentage of waking time the patient is aware of tinnitus and by the percentage of time it creates distress. Both awareness and distress from tinnitus decline significantly during hyperacusis treatment (awareness 59.9%, to 28.6% (paired t-test t 6.92, df 56, p<0.001) and distress 42.3% to 15.8% (t 6.92 df 56, p<0.001), see figure 4b.

Analysis for different sub-groups

Cumulative distributions were performed for sub-groups of patients (figures not shown). There was no difference in outcome between those patients who described phonophobia against those who did not, those who had a clear history of ear pathology, against those in whom this was absent, in musicians against non-musicians and between category 3 and 4 patients as measured by the Kolmogorov-Smirnov test .

LDL data correlated well with improvement in "life factors interfered with due to hyperacusis", as can be seen in Figure 5 (Pearson correlation = 0.33 p=0.004 n=74),. However there was no relationship between LDL changes and the change in "life factors affected by tinnitus" (Pearson correlation 0.163 p=0.16 n=74); graph not shown.

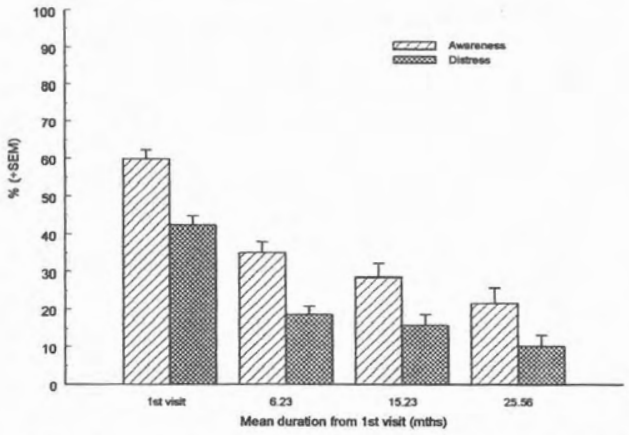

Figure 4b Percentage change in awareness and distress from tinnitus only at each visit, with SEM. (see text for method of measurement)

Discussion

Decreased Sound Tolerance (DST) involves more than one element symptomatically and pathophysiologically. Previously two terms have been used: 1) Hyperacusis describing patients experiencing discomfort from sound resulting from abnormally high activation of the auditory system, and 2) phonophobia describing patients expressing a fear of certain sounds, or all sounds, and resulting from abnormal activation of the limbic and autonomic nervous systems. In phonophobia, patients fear that specific sounds in the environment will damage their hearing or make their symptoms worse. However, in the past, many patients labeled as phonophobic were not really afraid of sound, but simply disliked it. For this reason the term misophonia has been introduced [5] which means literally a dislike or hate of sound, without necessarily any fear that it will cause damage. It is important to make this distinction, because, although in both cases there is activation of the limbic and autonomic nervous system, treatment is significantly different, particularly in counselling the patient. In practice, misophonia or phonophobia normally do not present in isolation, but are usually combined with hyperacusis as well. That is certainly true of this study. The Jastreboff model explains how increased gain in auditory pathways resulting in abnormal loudness perception can lead to a dislike of the sounds that cause this effect, and vice versa. With the exception of three cases in this series when phonophobia was treated in isolation, phonophobia or misophonia were always present with hyperacusis. Also in the majority of cases of hyperacusis some misophonia was likely to be present.

Phonophobia

In this condition patients develop an irrational, but often understandable fear that sound exposure, even normal environmental sounds, may damage the ears and hearing, or make tinnitus or hyperacusis worse. As hyperacusis develops, the perception of everyday sounds changes and they appear over loud, leading patients to believe that these sounds are actually at a harmful or damaging level. Ear protection, such as earplugs are frequently used by patients in increasingly low levels of environmental sound, in the belief that the hearing is being harmed. It is unfortunately still common for some professionals to recommend this as a treatment for DST. Such practice leads to the development of greater gain or amplification in the auditory system, which responds by adaptation to decreased sensory input. This in turn leads to greater hyperacusis and worse phonophobia. Our aim is the exact opposite, to gradually decrease hearing protection, and re-introduce normal environmental sounds.

Patients may be phobic to all sounds, or specific sounds, typically those that are sudden or impulsive, such as doors slamming. Among musicians it is common to develop the fear that the sound of your own instrument, or others with whom you are playing can damage hearing. The evidence for this may be simply that episodes of noise induced short duration tinnitus ("disco tinnitus") persists a little longer than usual. The effect is catastrophic; as the implications are that the musicians' life ambition, as well as their means of financial support, have suddenly disappeared.

Other cognitive factors may play a part, such as a director of a mobile phone company who produced powerful phobic reactions whenever he put his phone near to his ear. He had been worried by the publicity about mobile phones and the possible effects of microwaves.

Misophonia

Misophonia is a very widespread phenomena. Almost everyone has some sound that they disliked at some time, for instance the once ubiquitous scratch of chalk on blackboard, or the neighbours television set heard through the wall. A generalised dislike for environmental sound may simply indicate a response to territorial intrusion, increasingly common in a world of stress and violence. In an attempt to exclude external sounds individuals seek quieter and quieter environments. This simply makes the auditory system more sensitive to the presence of sound, and increases the perception of loudness.

Some individuals exhibit very specific dislike for individuals sounds which suggest an often long forgotten event during which the sounds were associated with some unpleasant experience. The trainee gynaecologist who became aversive to the sounds of an unborn baby's heart beat, and to the sound of newborn babies crying (common after delivery), was found to be in the wrong profession for various reasons! His initial presentation however was simply with phonophobia and hyperacusis. Examples of these sounds of which our patients complained in this study are given in table 2

Street sounds – car brakes
Snoring
Cutlery and plates
Vacuum Cleaner
Computer
Refrigerator
Waste (Garbage) disposal
Husband breathing in bed
Supermarket freezers
Sounds of eating
Own voice
Keys rattling
Toilet flushing
Other people's headphones
Swimming pool pump
Sound of singing
Mains electricity hum
Shovelling cement
Foetal heart sounds
Babies crying
School bell
Sound of drawing with felt-tipped pen.
Sounds Rarely Disliked
Bird song
Running water
Wind / rain

Table 2 Sounds reported as causing misophonia; present study.

Hazell and Sheldake were the first to described a protocol for desensitisation of hyperacusis, and at the same time show that loudness discomfort levels were significantly reduced after a treatment period (1991). The patients described in this paper were treated in a period prior to knowledge of the Jastreboff model and TRT. This is interesting in that it identifies the importance of the role of wearable sound generators, which were essentially the only aspect of treatment used during the 1980s, apart from general reassurance. There is no doubt however that using sound generators within a TRT programme and after Jastreboff categorisation, is much more effective. This approach is orientated to dealing with the additional tinnitus which is so frequently present, and commonly becomes the dominant symptom when DST is dealt with. It also avoids worsening the symptoms of category 4 patients, by improper use of sound therapy.

Mechanisms

The two principal mechanisms which are active in DST are hyperacusis, indicating an abnormal response of the auditory system, and misophonia or phonophobia indicating an abnormal response of the limbic and autonomic system in response to certain sounds. These two mechanisms are interactive according to the Jastreboff model. [1] see Figure 6. An increase in auditory sensitivity, indicated principally by decreased LDLs, produces increased awareness of environmental sound due to increased perceptual loudness and a change in attentional focus. Although the degree to which these stronger perceptions are tolerated, and the extent to which a dislike of sound is triggered by hyperacusis varies, this study shows that aversion to external sounds in terms of

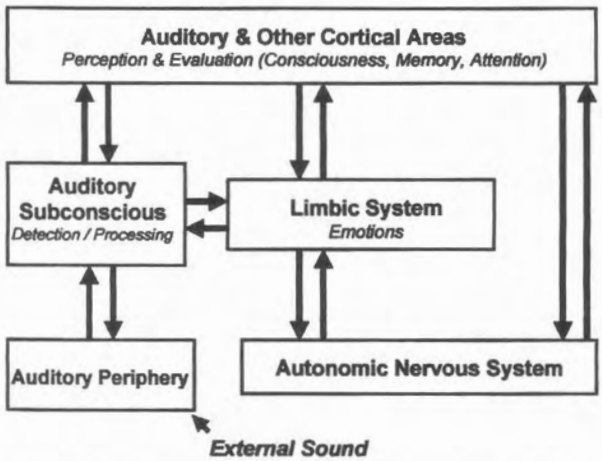

Figure 6 The Jastreboff neurophysiological model, as it relates to hyperacusis, misophonia and phonophobia. Hyperacusis is caused by changes in the auditory pathways leading to abnormal perception of environmental sounds. Misophonia and phonophobia results from abnormal activity in the limbic and autonomic nervous systems, producing an aversive / phobic reaction to certain sounds. These two systems interact, so that an aversive reaction to a sound commonly results in its increased detection, and visa-versa.

misophonia and phonophobia commonly occurs. In this study misophonia was present in each case.

In some patients, a decrease in sensory input, due to an abnormally quiet sound environment, can cause hyperacusis to develop slowly on its own. In other patients exposure to loud sounds, or the development of the belief that certain sounds are dangerous to the hearing, lead to an aversive, or sometimes phobic response to these sounds. In other situations certain sounds may evoke unpleasant memories or thoughts with which they are associated, so that the sounds themselves produce aversive conditioned responses by association. In most cases where these aversions are generated, a subsequent increase in central auditory gain occurs resulting in hyperacusis.

The mean LDL changes with treatment in this study were 12.82 dB as against 25.0 dB in a similar American study of hyperacusis cases without tinnitus [6]. Clinical data from the USA suggests a higher proportion of severe hyperacusis patients seeking treatment, or being aware that treatment exists, than in the UK, and possibly Europe as a whole. Of the patients in this study almost all had some degree of tinnitus and therefore are nearer to the LDL improvement exhibited by tinnitus-only patients in the American study (8.4 dB). In this present study 6 patients were exclusively hyperacusic (without tinnitus). They had a mean change of LDL of 20.47 dB during treatment. In all, the results in this paper are similar to those previously published by our group in 1991 for a UK population [7].

The high correlation between LDL data, and the improvement in life factors affected by hyperacusis, but not tinnitus, helps to validate the measures used in this study.

Trigger / predisposing factors

Both acute and chronic exposure to loud sound may produce either temporary or permanent changes in the cochlea or changes in central auditory processing. In this study this was the commonest trigger reliably associated with DST onset. It was the only factor seen among the musicians. Anxiety that hearing may be damaged leads to misophonia or phonophobia, and an abnormal response in the limbic and autonomic nervous systems whenever the sounds are present. Similar anxieties about ear dysfunction can arise after painful ear infection, ear syringing or perceived failure of surgery on the ear. Stress factors are often important in triggering the onset of DST, as well as tinnitus [8]. One patient had just helped to bury thousands of massacre victims in an African civil war. In many cases individuals feel threatened by circumstance, or they may retreat from environmental stimuli, including sound, resulting in adaptive hearing sensitivity. 5% described living in, or moving to, a very quiet environment. Ear plugs may have been used to improve sleep, disturbed by environmental sound. If such practices persist, hearing sensitivity increases and plugs are increasingly worn . External sounds become increasingly more intrusive as a vicious circle develops. In 11% of patients hyperacusis develops following a lifetime of dislike of sound intrusion, behavioural traits often learned from parents.

DST can follow injury and accident; a common event is being hit from behind in a motorcar shunt. Concepts of threat to life, combined with feelings of anger, generate powerful emotions linked to the aversive reactions in misophonia and phonophobia. In 5% there were severe and numerous phobias to heights, confined spaces, animals, social events etc. Generalisation to aversion to environmental sounds, in those with multiple phobic reactions, is predicted.

In 21.4% of patients there was no trigger factor other than the recent onset of tinnitus. It is likely that the similarity of mechanisms in tinnitus and hyperacusis, as shown by the Jastreboff model, makes the development of hyperacusis more likely when tinnitus is present. We have already pointed out this possible mechanism in previous studies [7]

As the treatment protocol for category 3 and 4 patients requires follow-up on at least one occasion, the patients who attended one session only could not be considered to have received appropriate treatment using 'full' TRT.

An attempt was made to contact the 50 non-attendees by mail. 18 replies were received. And of these 61.1% indicated that their hyperacusis has either disappeared or improved since their visit to the centre. Only 3 patients had got worse, reporting that they were unable to re-attend because their sound sensitivity precluded further travel.

The results in this study confirm our previous findings [7] and are in agreement with others using a TRT approach. The improvement in life factors affected by DST, reductions in LDLs, as well as the reduction in awareness and annoyance of coincident tinnitus, testify to the success of this approach.

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