Structured Counseling for Auditory Dynamic Range Expansion

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ABSTRACT

A structured counseling protocol is described that, when combined with low-level broadband sound therapy from bilateral sound generators, offers audiologists a new tool for facilitating the expansion of the auditory dynamic range (DR) for loudness. The protocol and its content are specifically designed to address and treat problems that impact hearing-impaired persons who, due to their reduced DRs, may be limited in the use and benefit of amplified sound from hearing aids. The reduced DRs may result from elevated audiometric thresholds and/or reduced sound tolerance as documented by lower-than-normal loudness discomfort levels (LDLs). Accordingly, the counseling protocol is appropriate for challenging and difficult-to-fit persons with sensorineural hearing losses who experience loudness recruitment or hyperacusis. Positive treatment outcomes for individuals with the former and latter conditions are highlighted in this issue by incremental shifts (improvements) in LDL and/or categorical loudness judgments, associated reduced complaints of sound intolerance, and functional improvements in daily communication, speech understanding, and quality of life leading to improved hearing aid benefit, satisfaction, and aided sound quality, posttreatment.

KEYWORDS: Structured counseling, dynamic-range expansion, sensorineural hearing loss, sound therapy.

Learning Outcomes: As a result of this activity, the participant will be able to: (1) describe the relationship between the presence of reduced auditory dynamic range and unsuccessful efforts in using amplified sound; and (2) describe the efficacy of counseling and sound therapy to facilitate the expansion of the auditory dynamic range.

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Counseling, as described here, was integral to the successful sound therapy treatment for expansion of the auditory dynamic range (DR) for loudness among persons with elevated hearing thresholds and slightly lower-than-normal to borderline-normal loudness discomfort levels (LDLs).[1,2] The primary goals of the structured counseling protocol were to promote expansion of each participant's DR, corresponding improvement in his or her sound tolerance and, ultimately, successful use of amplified sound from hearing aids. The latter was not possible before treatment because of his or her limited DRs and reduced tolerance for amplified sound. The purpose of this report is to describe in greater detail the structured counseling approach used in Formby et al so that the principles can be applied by other investigators and clinicians in sound therapy-based treatments and related research aimed at facilitating DR expansion.[1,2]

The principles, content, and format of our counseling protocol were devised and shaped by the senior author (S.G.). S.G. was experienced in providing counseling for sound therapy-based treatments, mostly in applications of Tinnitus Retraining Therapy (TRT) at the University of Maryland Tinnitus and Hyperacusis Center.[3] Historically, TRT was designed as a protocol for habituation of intrusive tinnitus.[4] Subsequently, TRT evolved to include alleviation of sound tolerance problems, including hyperacusis (decreased sound tolerance), misophonia (dislike of sounds perceived as loud), and phonophobia (fear of sounds). Patients with normal/near-normal hearing sensitivity and those with all degrees of hearing loss were evaluated and treated with TRT. The TRT protocol combined counseling, as advocated by Jastreboff and Jastreboff,[5] and low-level sound therapy, initially promoted by Hazell and advanced by Hazell and Sheldrake.[6,7] As part of the TRT initial audiological evaluation, LDL judgments were routinely measured for every patient. Early on, we noticed a recurrent and surprising finding among many of our hearing-impaired patients. Namely, they often presented with reduced LDL values and associated sound tolerance complaints at the start of TRT.[8–10] However, when tested at TRT follow-up visits, their LDLs were typically increased over the course of treatment, even among patients with initially normal LDLs (i.e., $\geq$100 dB hearing level [HL]).[8–10]

Among the hearing-impaired patients treated by TRT were those who previously had attempted to use hearing aids with limited success. They often reported their initial efforts to use amplified sound pretreatment were unsuccessful because of their reduced DRs and associated aided intolerance and discomfort. However, posttreatment, after DR expansion, many of these patients were then able to make a comfortable transition to appropriate amplification. We previously reported some of these successful treatment effects, which were often accompanied by subjective reports of improvements in sound tolerance during and subsequent to treatment.[8,10,11] The basic counseling principles described in those reports were adapted by S.G. for our application in the treatment of relatively typical hearing-impaired persons with reduced DRs.[2] Most of the participants described in Formby et al were not bothered by tinnitus, nor by distress associated with primary hyperacusis[2]; rather, their main sound tolerance issues were related to their elevated hearing thresholds and, on average, lower to borderline-normal LDLs, which together restricted comfortable aided listening and hearing aid benefit. Our target patient group in Formby et al therefore differs from primary tinnitus and hyperacusis patients, who were routinely managed by S.G. in the University of Maryland Tinnitus and Hyperacusis Center.[2,3] Here we consider the specifics of the structured counseling protocol as implemented by S.G. in Formby et al.[2]

COMPONENT 1: OVERVIEW OF COUNSELING APPOINTMENT AND AUDIOMETRIC RESULTS

The counseling protocol was conducted in a single session, usually lasting ~1 to 1.5 hours. S.G. counseled each participant individually, following the format of the scripted checklist shown in Table 1. S.G. began by providing a general overview of the counseling and purposes of the research. She then explained to each participant that during the counseling session, he/she would discuss the participant's results for

Table 1 Counseling Checklist for Dynamic Range Expansion

Component 1: Overview of Counseling Appointment and Audiometric Results
Present general overview of counseling for reduced sound tolerance.
Review participant's pretreatment audiometric results.
Review participant's pretreatment loudness discomfort levels (LDLs) and reduced dynamic range (DR) for pure tones.
Present treatment goals of improved sound tolerance, an expanded DR for loudness (illustrated with a case example), and an enhanced ability to use appropriate amplification.
Component 2: Overview of Auditory Anatomy and Neurophysiology
Explain anatomy and physiology of auditory system.
Describe outer, middle, and inner ear components (relate to sensorineural hearing loss).
Explain that people hear at the brain (auditory system acts as a transformer).
Describe structure and function of inner and outer hair cells with reference to sound tolerance.
Explain afferent/efferent neural control of peripheral auditory gain.
Explain that if auditory input is reduced, then the brain will "turn up the gain" in an attempt to enhance the input.
Component 3: Overview of Central Auditory Gain Control Mechanisms
Explain how the brain handles the input from the peripheral auditory system.
Describe subcortical areas: monitoring and filtering roles.
Discuss cortical areas and perception.
Explain central auditory gain and its role in modulation of loudness perception.
Discuss the Jastreboff model and the misophonic response.
Component 4: Role of Sound Therapy
Explain the use of low-level sound therapy as a tool to "turn down the gain" within the auditory pathways.
Show again examples of positive treatment effects achieved with treatment approach.
Emphasize the importance of a sound-enriched environment in regulating the auditory gain mechanism.
Avoid silence. Keep a low level of neutral sound on at all times, day and night. Examples are a fan, computer, sound machine, nature tapes, humidifier, or fountain set at low volume.
Use appropriate noise protection, but not overprotection.

several hearing tests and address why the findings qualify the participant for this study. S.G. related the treatment goal of the investigation was for each study participant to use appropriate amplification successfully to communicate effectively and comfortably in an often noisy world. She explained the counseling session would help the participant to understand why she or he may have had difficulty using hearing aids to date, what the treatment protocol is, and why this treatment may help him or her achieve the treatment goal of aided benefit and satisfaction. S.G. noted this is the first step in the treatment protocol and encouraged each participant to ask questions at any time during the counseling session to clarify issues or concepts that were unclear.

The framework of the counseling session included coverage of the following topics: results of the audiological evaluation (specifically, her or his audiometric thresholds and LDLs), the associated DR, and the hearing-impaired participant's reduced DR in relation to the normal DR; the anatomy and physiology of the normal auditory system and the changes that occur as a consequence of sensorineural hearing loss; the processes by which the central auditory system and higher cortical processes interpret an auditory signal; an introduction to the concepts of adaptive plasticity (of loudness) and auditory gain control processes in the context of Jastreboff's neurophysiological model (as it relates to decreased sound tolerance, i.e., hyperacusis, misophonia, phonophobia)[12]; the sound therapy treatment rationale; and the intervention in terms of the goal of "turning down the gain" of the auditory system while also stressing the importance of maintaining healthy

environmental sound exposures in this process. S.G. systematically presented the information to help the participant understand and facilitate change (if necessary) in the way that he or she viewed his or her ill-defined intolerance problem, addressed each participant's concerns, and discussed the long-term goal for the participant to use appropriate amplification, without discomfort or distress, after completion of treatment.

Next, S.G. reviewed and explained to the participant the results of his or her pretreatment audiological evaluation. A primary objective of this review was to help the participant understand and separate the contributions of her or his hearing issues and associated audibility problems from sound tolerance complaints as factors limiting the auditory DR and her or his potential use and benefit from amplified sounds. A copy of the participant's audiogram was enlarged for each ear separately and generated for S.G. to discuss the participant's audiological results. Shown on the audiogram were the audiometric thresholds, the LDLs, and speech test measures. S.G. reviewed the audiometric results for each ear and explained to the participant how to interpret the pure tone thresholds and speech test data. S.G. compared the participant's results to those expected for a normal-hearing person, noting any audiometric differences between the participant's ears and the participant's audiometric configuration in relation to her or his history of noise trauma, ototoxicity, or ear-related disease.

The concept of loudness and the measurement of LDLs were reviewed next. S.G. explained the LDL measurement protocol, its purpose, and showed the participant his or her results. These results were then compared with those for someone with normal sound tolerance (i.e., LDLs ~100-dB HL) and also compared with those for someone with reduced sound tolerance, either because of elevated hearing thresholds, reduced LDLs, or both together.[13] These differences were used to explain the normal and reduced DR, respectively. S.G. noted that persons with sensorineural hearing losses have a reduced DR for at least some frequencies because of their elevated audiometric thresholds. When the DR is limited to less than 60 dB, then typical hearing-impaired listeners may experience loudness-related problems,[13] especially when hearing aids are fitted.[14,15] She related that she was evaluating the efficacy of low-level sound therapy as an intervention to expand the DR and enhance sound tolerance.[2] S.G. explained that a similar intervention strategy was implemented clinically in a gentle, noninvasive protocol for almost 20 years in related treatment applications. S.G. emphasized that many patients who successfully completed treatment with sound therapy achieved an expanded DR. These intervention effects result from incremental shifts in LDL values over the course of treatment, which often are sustained in whole or part posttreatment, allowing for comfortable use of appropriate amplification subsequent to DR expansion. S.G. then personalized the purpose of this study for the participant, explaining the intent was to evaluate the extension of these previously successful sound therapy principles in a protocol format appropriate for application to persons with sensorineural hearing losses and reduced DRs.

Finally, S.G. briefly reviewed the speech tests and the corresponding results, explaining that these tests are designed to demonstrate the ability of the participant to detect and understand speech at conversational levels. The results of the speech reception threshold measurements typically agree closely with the pure tone thresholds measured for the primary frequencies that are critical in understanding speech (i.e., 500, 1,000, and 2,000 Hz). The speech discrimination scores measure the percentage of single syllable words, understood correctly in each ear, when the words are presented at a normal conversational level. This information laid the groundwork for later discussion of the benefits of amplification.

COMPONENT 2: OVERVIEW OF AUDITORY ANATOMY AND NEUROPHYSIOLOGY

The next component of the counseling protocol was comprised of a review and simplified explanation of the anatomy and physiology of the auditory system, with visual presentation materials designed to illustrate the key points being discussed. A three-dimensional model of

the ear also was used for this part of the counseling (ADV, Replicas Associates Inc., Jacksonville, FL).[14]

The visual aid presentation began with a diagram of the ear (Zenetron, Inc., Chicago, IL), including a description of the gross anatomy of the components of the ear. S.G. simultaneously used both the illustrations in the visual aid material and the three-dimensional model of the ear to describe the anatomy and physiology of the outer, middle, and inner ear and to discuss their relations to possible origins of the participant's associated hearing loss. S.G. spent the majority of this phase of counseling educating the participant about the inner ear and associated damage and/or disease that might explain or contribute to his or her reduced tolerance condition.

Points covered about the outer ear included that the pinnae capture and direct sound waves to the eardrums and that sound waves cause mechanical vibration of the eardrum.

Points covered about the middle ear were that the middle ear is air filled; transmission of sound through the middle ear takes place via a chain of three small bones interconnected within the middle ear (malleus, incus, and stapes); and the mechanical transmission of sound between the eardrum and the round-window entrance to the inner ear (i.e., cochlea) occurs when these three bones are set into vibration by sound impinging on the eardrum.

S.G. then used the three-dimensional model of the ear to describe the anatomy of the inner ear, including the cochlea and semicircular canals. S.G. focused primarily on the cochlea, which is the important sensory organ for hearing. She demonstrated the relation between the bones of the middle ear and the cochlea. She then explained that the outer and middle ear structures facilitate sound transmission to the cochlea, which transforms mechanical transmission of the sound to electrical signals for neural conduction. The cochlea is best described as a hollow spiral tube filled with fluid that bathes sensory cells contained within this tube. These sensory cells are called inner hair cells (IHCs) and outer hair cells (OHCs). S.G. stressed that the transmission of a sound signal within the cochlea is due to the wave motion of the fluid within the cochlea, which selectively activates hair cells at the place of primary stimulation along the cochlea corresponding to the frequency of vibration.

S.G. used the next set of diagrams to show the IHCs and OHCs, the basilar membrane, and the neural fibers that extend from the bottom of the hair cells to become the auditory nerve bundle.[16,17] S.G. pointed out the hair cell bodies, their cilia, and the nerve fibers that form the auditory nerve. She explained the hair cells are key sensory structures that transduce and amplify the mechanical sound signal from the eardrum. The hair cells act as a transformer changing mechanical energy into an electrochemical signal. At the IHCs, the sound signal is converted to an electrical signal that is ultimately transmitted through auditory neuronal pathways to the brain where the signal is recognized as sound. S.G. emphasized that people *hear* when higher centers within the brain are activated. The ear is the peripheral sensory processor of the sound stimulation.

S.G. then explained the separate functions of the IHCs and OHCs using electron microscopy images of the basilar membrane and hair cells.[16,17] The IHCs are the primary sensory transducers of sound and send information through central neural auditory pathways to the auditory cortex. The neural activity is in the form of a series of electrical impulses that go primarily in one direction (i.e., sending information from the IHCs via neural pathways to the brain). The IHCs receive little or no input back from the brain. Thus, the IHCs are the sensory cells that convey sound information to the brain. The IHCs are sheltered and protected from damage, more so than the OHCs.

In contrast, the OHCs boost the signal provided to the IHCs and fine-tune the frequency specificity of the auditory system.[18] The OHCs also amplify weak signals and attenuate or compress moderately intense signals, thereby extending the auditory DR. Thus, OHCs are important in the overall gain adjustment to sound.[18] OHCs send signals or impulses along the nerve fibers going up to the brain (afferent fibers), but, unlike the IHCs, they also have large numbers of nerve fibers (efferent fibers) coming from the brain back to the OHCs.[17] This unique innervation characteristic suggests the OHCs may be important in regulating

instantaneous auditory system gain and amplifying and modifying the signal-to-noise levels reaching the brain.[18] Thus, in effect, an *automatic gain control process*, mediated through the OHCs, instantaneously monitors and adjusts for ongoing changes in our sound environment at both peripheral and central levels. A soft signal may be amplified by the OHCs by up to 60 dB.[19–22] This signal may be further amplified in the central auditory pathways.[23–26] If the mechanisms controlling this gain process produce higher levels of amplification, then sounds may be perceived as abnormally loud.[27]

S.G. related that the OHCs are not as protected as are the IHCs. Consequently, the OHCs are susceptible to greater wear and tear from noise, ototoxic drugs, viral infections, and aging. People tend to lose ~0.5% of our OHCs each year from the time they are very young. Fortunately, people have a built-in redundancy and an abundance of OHCs. S.G. informed the participant that a typical listener could lose up to ~30% of her or his OHCs, spread evenly throughout the cochlea, and still have audiometrically normal-hearing sensitivity as measured by audiometric pure tone thresholds.[28,29] Finally, S.G. returned to the discussion of the cochlea and explained the cochlea is a coiled structure that is frequency specific. This frequency specificity means that, in response to stimulation by sounds of different wavelengths, the cochlea vibrates at different frequencies; high-frequency sounds stimulate mechanical activity at the entrance to the cochlea and low-frequency sounds activate the far end of the cochlea. The hair cells that respond to the higher frequencies, at the entrance to the cochlea, are more vulnerable to wear and tear much like the carpet at the front door wears out first. This vulnerability of the basal hair cells at the entrance to the cochlea accounts for the greater susceptibility of the higher audiometric frequencies to hearing loss. To illustrate this point, S.G. showed the participant a visual aid with familiar sounds represented schematically on an audiogram across frequency from low to high.[30] S.G. also explained hearing loss typical of aging-related deficits, acoustic trauma, and ototoxicity within the context of the participant's damaged cochlear anatomy. S.G. described the expected impact of frequency specificity on hearing sensitivity and related possible difficulty understanding sounds at the higher frequencies in the face of this damage.

COMPONENT 3: OVERVIEW OF CENTRAL AUDITORY GAIN CONTROL MECHANISMS

S.G. used a new series of visual aids to address how the brain and the central auditory pathways process sound inputs from the peripheral auditory system.[31] S.G. pointed out the auditory fibers as they arise from the cochlea and travel through intricate auditory pathways and structures to the auditory cortex. Points made by S.G. included that the nerve fibers in the auditory pathways cross over so that fibers from one ear stimulate both sides of the brain. This neural crossover facilitates localization of sound. The neural pathways that lead from the cochlea to the cortex are conveyed through and by brainstem and midbrain structures that consist of a series of complex and specialized interconnected nerve cells called neural networks. S.G. reiterated the auditory nerve has both afferent and efferent fibers, meaning that signals go from the IHCs (and, to a lesser extent, from OHCs) to the brain via afferent fibers and from the brain back to the OHCs via efferent fibers.

S.G. explained auditory signals are interpreted within higher cortical areas of the brain, which are responsible for cognition (i.e., our thoughts, perceptions, and understanding). S.G. then explained the brain has other subcortical, nonauditory areas that operate at a subconscious level. There are at least five different subconscious levels (structures) that are responsible for monitoring, filtering, and enhancing information from the peripheral auditory inputs (as well as our other senses) to the brain.[27] Each person weighs and processes this information differently at these lower subcortical levels.

Primary loudness-based hyperacusis is assumed to be solely an auditory pathway problem in Jastreboff's neurophysiological model (described later).[27] The problem is thought to arise from abnormally elevated auditory activity or gain within the auditory

pathways, which is manifested by abnormally lower LDLs and reduced sound tolerance.[11] Treatment is based on desensitization (or recalibration) of central gain processes by systematic exposure to nonannoying, low-level sound, which typically results in improved LDLs.[12,32,33] The concepts of misophonia and phonophobia were explained to the extent that the participant reported distress or annoyance and/or was fearful of experiencing discomfort from or dislike of certain sounds.[12] (These latter issues were screened and were not typically reported as primary problems among the treatment groups in Formby et al.[2]) S.G. also noted that, due to the plasticity of the brain, these undesirable associations can be altered or relearned, enabling the listener to function comfortably in noisy situations without fear or avoidance of sound.[27]

At this point in the counseling session, S.G. introduced the next visual aid, which depicted a block diagram of the Jastreboff model as it relates to decreased sound tolerance.[34] S.G. went through the block diagram, identifying the various components of the model and discussing the contribution of each of these components to hyperacusis, misophonia (if indicated), and phonophobia (if indicated). The order of presentation of the model components was described in terms of the processing and associated anatomical mechanisms. The essential points S. G. covered with respect to the model were:

Source

In the case of decreased sound tolerance, an external sound (the source) can be significantly amplified by instantaneously activated gain control processes of the peripheral auditory system mediated through the OHCs.

Detection Mechanisms

Subcortical and subconscious structures (the detection mechanisms) filter random neural activity (coming from the peripheral source). Moderate or even soft sounds can be amplified by the OHC system and further augmented and enhanced by centrally mediated gain control processes within the higher auditory pathways.[35] If the mechanisms that control auditory system gain inordinately amplify or give rise to higher levels of stimulation within the auditory pathways, then the result is the perception of abnormally loud sound or even a painful response to sound. This experience is the phenomenon of primary loudness-based hyperacusis.[36,37]

Emotional Associations

Hyperacusis in the model is a problem occurring exclusively within the auditory pathways.[27] Hyperacusis can, over time, also increase activation of emotional associations to sound (through the limbic system).[33,35,37] When sounds are perceived to be abnormally loud, the neural networks can become activated and highly tuned to monitor conscious and subconscious signals resulting in distress, annoyance, and anxiety. The limbic system plays an important role in controlling our emotions, with direct connections between the limbic and auditory systems. If a connection between loud sounds and the limbic system is made, then associated distress in the form of misophonia and/or phonophobia may also occur based on conditioned reflexes.

Annoyance

Persistence of the activation of the limbic system by sound can result in subconscious annoyance mediated by the autonomic nervous system (ANS).[27] Activation of the ANS, in turn, can result in increased muscle tension and anxiety, elevated levels of stress hormones, enhanced blood flow, fight-or-flight reactions, and/or panic attacks. If the activation of the ANS is excessive and prolonged, then an unhealthy stress response may arise as evidenced by insomnia or ongoing exhaustion. The ANS then may become negatively conditioned to the adverse perception of loud sounds, reacting strongly, and placing those sounds at the top of the list of stimuli for the brain to monitor. These augmented annoyance and distress responses also can result in even greater awareness, activating both limbic and ANS reactions to sounds in the environment. S.G. emphasized in this phase of the counseling that undue annoyance to loud sounds is not the

consequence of a system that is working poorly. Rather, it is an indication of a system that is working too well and overcompensating to a change in the normal gain control setting of the auditory system.

Perception and Evaluation

Disproportionate activation can take place at a subconscious level (subconscious loop of the model) without any involvement of conscious thoughts. There also can be a higher-level conscious loop (involving the auditory cortex and other cortical structures) activated by perceptually loud sound.[27] This additional activation can contribute to and may evoke conscious worries and concerns about increasing hearing loss, an inability to cope with or to use amplification successfully, lack of available treatments, increased isolation, and so on. The latter concerns and negative thoughts may contribute to even greater activation of limbic and ANS processes.

COMPONENT 4: ROLE OF SOUND THERAPY

Most clinically significant sound tolerance problems consist of a combination of loudness-based hyperacusis and some element of distress or misophonia.[12,33,38] If a participant has physical discomfort when exposed to certain sounds, or discomfort when presented with sound levels that typical listeners would not ordinarily report as problematic, then aversion and emotional responses will become involved. The brain is constantly monitoring sounds around people through adaptive and plastic processes that can modify the gain of the central auditory pathways. Sometimes the gain mechanisms amplify too much. Sounds may then become uncomfortable, if not painful, even though these sounds are comfortable for most people. To attack complaints of abnormally reduced sound tolerance or hyperacusis, clinicians have used controlled low-level sound therapy successfully for the past two decades to treat these problems.[3] S.G. reiterated the same basic sound therapy principles also may be beneficial for resolving the sound tolerance conditions that limit the participant's use of amplified sound from hearing aids. These are the principles evaluated in this study.[2] The sound therapy treatment in this study was presented at soft levels slightly above the subject's hearing threshold. Sound therapy is usually best delivered with sound generators used in both ears, which emit a soft "seashell-like" sound that can be used to treat the subject's condition. S.G. emphasized that, in addition to using the sound generators as prescribed, it is important to avoid silence at all times, day and night; additional low-level sound therapy can be achieved by the use of healthy environmental sound from sound machines, sound pillows, fans, air conditioners, tabletop fountains, nature tapes, aquariums, or similar devices. Improvements in sound tolerance and expansion of the DR can generally be seen within a few weeks or months of initiating sound therapy. These changes are shown by increases in the LDL judgments across most or all frequencies in both ears at follow-up visits and by subjective reports of enhanced sound tolerance to everyday sound.[11]

The participant was informed that he or she does not appear to suffer a primary problem related to either phonophobia, an inordinate fear of certain sounds, or misophonia, a strong dislike of certain sounds.[27] These issues may manifest as concern that specific sounds or sound categories may damage the auditory system, cause undue discomfort, or exacerbate hyperacusis. When these problems are present, many persons have an increased tendency to overprotect the ears.[6] This overprotection against otherwise healthy sound can evoke an undesirable compensatory increase in auditory pathway gain, making the auditory system even more sensitive to suprathreshold sound. Appropriate noise protection is important for minimizing the damaging effects of high-intensity sound exposure; however, the use of sound-attenuating plugs or muffs inappropriately for prolonged periods can have unintended detrimental effects. When the brain is deprived of healthy sound input, as a consequence of the prolonged use of earplugs, this reduced stimulation can produce a compensatory response that further increases sensitivity within the auditory pathways. This increased auditory gain response

may further exacerbate the ongoing sound tolerance problem, making the condition even more difficult to treat.[7,39,40]

S.G. explained that the participant's reduced DR problem is likely treatable and may be improved with the gentle and noninvasive sound therapy intervention to be evaluated in this study.[2] The goal of the protocol is for the participant to live comfortably in a busy, noisy world and for her or him to use appropriate amplification posttreatment to hear and communicate better with greater ease. The use of low-level sound therapy from sound generators, environmental sound, and other complex sound sources is designed to turn down the gain of the auditory system over time.[27,41] It is important for the participant to maintain neutral, low-level environmental sound exposure at all times, day and night, so that the auditory system can begin the process of resetting the elevated system gain to normal levels. The sound therapy does not have to be loud to be effective and may, in fact, be very soft. Our clinical experience with sound therapy indicates that after the gain mechanism is reset to restore normal loudness perception and sound tolerance, typical gain settings tend to be sustained.

S.G. then explained the use of appropriate and healthy noise protection, such as when using power tools, lawn mowers, leaf blowers, or firing weapons, is encouraged, but chronic overprotection of the ears is actively discouraged. Additionally, she emphasized that it is imperative to avoid silence for long periods of time during the treatment period. If environmental sounds are perceived to be too soft or are not being heard adequately (as may occur when wearing hearing protection inappropriately or unnecessarily for prolonged periods), then the compensatory auditory gain response may be enhanced in undesirable ways that can be counterproductive, further exacerbating the tolerance problem.[27]

S.G. stressed the sound tolerance problem is not predictive of future changes in hearing sensitivity as measured by the audiogram. She also noted that the hearing loss and reduced measures of sound tolerance (lower-than-normal LDLs) are largely separate issues, but together these problems may be detrimental in their combined perceptual effects and contribution to the participant's reduced DR. S.G. shared that the participant's hearing thresholds and loudness measures of sound tolerance would be tested at follow-up appointments to document treatment outcome.

At the end of the counseling session, after the treatment objectives and goals were set, S. G. asked each participant if he or she had questions or concerns about the material covered. Any questions the participant had at this point were answered fully. S.G. concluded by reviewing and determining that the participant assimilated the counseling information.

DISCUSSION

We successfully used the counseling protocol described in this article, in combination with low-level sound therapy, to promote and enhance DR expansion and sound tolerance among persons with hearing loss with limited DRs and reduced sound tolerance prior to treatment.[2] The ultimate goal was to facilitate their hearing aid use and benefit. Pretreatment, these individuals were difficult-to-fit, often frustrated, hearing aid candidates for whom amplification was either uncomfortably loud at their prescribed gain settings or afforded no appreciable aided benefit for communication when gains were reduced to achieve comfort and tolerance. Other similarly affected treatment groups, including those treated with sound therapy alone, counseling in combination with an ineffectual placebo sound therapy, and the placebo sound therapy alone, did not generally benefit from their interventions.[2]

The structured counseling protocol, as described here, was based on S.G.'s personal experience, over a period of ~20 years, with principles that have proven successful in counseling hearing-impaired TRT patients.[3] These patients typically used sound generators (alone or in combination units with a hearing aid) together with continuous carefully prescribed environmental sound for their sound therapy to treat debilitating tinnitus and/or hyperacusis. Many of these patients, posttreatment, achieved clinically significant increases in their DRs and improvements in sound tolerance. This improvement enabled them

to begin or resume the use of amplification from hearing aids and to achieve improved aided benefit.[11]

The hearing-impaired participants in Formby et al were not usually bothered by tinnitus as their primary problem.[2] They commonly had borderline-normal sound tolerance or were only mildly hyperacusic as revealed by their pretreatment LDL values. Our participants, therefore, were reasonably typical of challenging sound-sensitive hearing aid candidates whom one might encounter in a traditional audiological/hearing aid practice, rather than in a specialty tinnitus/hyperacusis center. Our structured counseling was adapted to teach these participants about: their diminished DRs, owing to elevated hearing thresholds and/or decreased sound tolerance as shown by their reduced or borderline-normal loudness measures (i.e., LDL and uncomfortable categorical loudness judgments); the auditory system and hearing impairment; elevated auditory gain control processes and the plasticity of these processes in relation to their sound tolerance problem; and, in turn, the treatment goal to expand the DR and enhance sound tolerance using low-level sound therapy to "turn down" the elevated auditory system gain, thereby improving the participants' abilities to use and benefit from amplified sound from hearing aids posttreatment.

At the onset of the interventional trial in 2002,[2] we were uncertain of the nature of the participants' problems and how much information, or even the kinds of information, to include in the counseling protocol. This uncertainty included the relevance of Jastreboff's neurophysiological model,[27,34] which we and others have reported to be a primary component of a successful TRT treatment protocol for improving sound tolerance for tinnitus and hyperacusis patients (see Formby et al[42]). The counseling protocol implemented by S.G. in Formby et al included a simplified presentation and discussion of the key principles of the Jastreboff model as described here.[2] This presentation of the model was limited to loudness-based hyperacusis and to those properties that distinguish this primary problem from related problems of misophonia and phonophobia. We did not use nor convey the interpretation of the model in terms of TRT,[27] which was never mentioned nor described per se.

Initially, we questioned whether the model contributed substantively to the intervention outcomes. The treatment groups in Formby et al were mostly subclinical for primary hyperacusis and seldom in the screening process reported specific complaints or described sound-induced symptoms of distress, anxiety, or associated problems typical of misophonia.[2] However, subsequent analyses of participant responses to a sound tolerance questionnaire,[43] which was in development during the sound therapy trial, indicate issues of sound-induced distress and annoyance (and to a lesser degree tinnitus) may have been a greater problem for some participants than they related during the screening process for inclusion in the trial. Also, at the time of planning for the trial and at the onset of this study the term misophonia as a component of decreased sound tolerance was just being defined and introduced.[12,38] If these or related symptoms were present and unrecognized by either the study participants or our clinicians, then the presentation of the model may have been beneficial as a contextual and conceptual tool that contributed to improved sound tolerance. This inclusion of the Jastreboff model in our counseling protocol appears to have been warranted and perhaps more relevant than we anticipated, at least for some participants.

It is not obvious that any of the other counseling components shown in the checklist in Table 1 were dispensable. S.G. systemically addressed each and every one of the checklist topics in a single counseling session. The depth and emphasis given the different counseling topics were dependent on each individual's specific questions, concerns, and needs. The language used in the counseling also was participant dependent and was presented at an appropriate level, or simplified as required, to be effective for the needs and capabilities of each participant.

It is also not obvious how or what the mechanistic contribution of the counseling effect was in the successful full-treatment protocol reported by Formby et al,[2] nor why counseling in virtual isolation (in combination with a placebo sound therapy) was ineffectual

and no better (or even worse) than the efficacy for the placebo sound therapy control in isolation. The latter finding for counseling seems inexplicable and is especially troublesome for understanding the beneficial and essential effects of counseling to the success of the full treatment in combination with the low-level sound therapy. Specifically, how was it possible for counseling in the full-treatment protocol to have effectively doubled the treatment effect of sound therapy alone in the full-treatment protocol when counseling in virtual isolation had little or no measurable treatment effect on average for the participants assigned to counseling in combination with placebo sound therapy? The counseling session was initiated after the baseline test measurements and preceded the fitting of their conventional sound generators (CSGs) or placebo sound generators (PSGs) to begin the sound therapy. Counseling followed a structured protocol that was administered by a single TRT-experienced clinician, S.G., in a checklist format. S.G. was blinded to whether the study participant would be fitted with CSGs or PSGs.[2]

In Formby et al,[2] we considered this quandary, along with several possible mechanisms by which counseling might have acted as a synergistic force multiplier to augment the full-treatment effect. We speculated the synergistic contribution from the counseling might have been mediated by fostering the participant's partnership and belief in the intervention and goals of treatment. The participants were told at the beginning of the counseling session that we would cover the checklist topics in detail. An understanding of this information may have contributed to: the promotion of participant treatment compliance and motivation; top-down modulation and/or reinforcement of the sound therapy effects for the CSGs; reduced participant stress and anxiety (i.e., decreased activation of the limbic system and/or ANS); or in some other way less apparent. For example, when the participants used their CSGs, they may have recognized that, in the course of treatment, the improvements in sound tolerance that S.G. said would happen did, in fact, happen. Conversely, for the group that received counseling and PSGs, we suppose the continued use of placebo devices in the absence of a positive treatment effect could have negated or counteracted the participant's expected improvements that S.G. described during counseling.

Finally, in Formby et al,[2] we noted that hyperacusis (i.e., physical discomfort when exposed to sounds above a certain loudness) in its purest form is assumed in the neurophysiological model to represent abnormal enhancement of neuronal activity within the subconscious auditory pathways.[34] In misophonia (i.e., an emotional response to sound characterized by dislike or annoyance to specific sounds), activity within the auditory system is normal, but involvement of key nonauditory brain structures (i.e., the limbic system and the ANS) is enhanced. We suppose misophonia (or a low-grade form of distress to sound), which we now know is a common comorbidity with hyperacusis and deceased sound tolerance,[11,38,44] might have been present but was unrecognized or underreported by our mildly affected participants. If these conditions were more pervasive in the participants in Formby et al than they related at baseline, then our counseling may have addressed these associated problems indirectly.[2]

Another possible mechanism affecting the results of this study is that the clinical protocol implemented in TRT is designed to neutralize negative emotional associations using both counseling and sound therapy.[27] In Formby et al,[2] we applied some of the principles from TRT counseling to participants with reduced DRs to compare treatment effects with and without counseling in combination with and without conventional sound therapy. In discussing the mechanisms underlying the treatment effects of TRT, Jastreboff and Hazell proposed that counseling alone affects the cortical areas and may decrease the strength of the connections to the limbic system and the ANS,[27] but their activation persists. Sound therapy alone for hyperacusis attempts to desensitize the auditory system by systematic exposure to a variety of sounds to decrease auditory pathway gain and to reduce activation of the limbic system and ANS. Through the combined implementation of both counseling and sound therapy, cortical and subcortical connections will weaken. Moreover, activation of all centers will lessen, promoting disruption of connections between the auditory system, the limbic

system, and ANS. This mechanistic account may explain the difference in the results we reported in Formby et al between our full-treatment group 1, given counseling and using CSGs, and our partial-treatment group 2, given counseling and using PSGs.[2] Because group 2 was offered limited sound therapy from the PSGs, the sound therapy was inadequate when combined with the otherwise effective counseling. Consequently, the combined protocol for group 2, counseling in combination with PSGs, failed to achieve its intended purpose of decreasing auditory gain to promote DR expansion. In TRT theory, counseling sets the stage for demystifying the problem for the participant, neutralizing negative emotional associations. Sound therapy does not initiate this process, but it is expected to facilitate the process of decreasing auditory gain, reinforcing the basic tenets of TRT that both counseling and sound therapy, in some form, are essential components.[3]

Whatever the mechanisms of the counseling effect, we concluded in Formby et al, and believe it important to reemphasize now, that the counseling approach delineated in this protocol is beneficial, and apparently essential, for maximizing the efficacy of sound therapy.[2] Together, these combined treatment elements offer a promising intervention to expand the auditory DR, thereby enhancing sound tolerance for a large segment of the hearing-impaired population.

As Palmer suggests,[45] audiologists are ideally positioned to offer our counseling approach as a component of a sound therapy-based protocol to enhance hearing aid benefit for patients who may now be problematic to fit successfully with conventional prescriptive methods because of their limited DRs and associated reduced sound tolerance. Thus, the counseling approach and principles we described, albeit reported for application in a research study, also should be applicable clinically. This approach offers audiologists, who are uniquely qualified to deliver and share the information, a new tool to enhance aided satisfaction and benefit for many DR-challenged hearing-impaired patients. These are often among the most problematic and unsuccessful hearing aid candidates encountered in clinical practice. They may be inadequately aided (i.e., underfitted relative to target gains) or resist amplification because of their ill-defined sound intolerance associated with their reduced DRs.

We have not yet attempted to use our counseling approach with forms of sound therapy other than low-level broadband sound. We would expect our basic counseling protocol and principles to facilitate the treatment efficacy of other kinds of sound treatments that might also promote DR expansion (e.g., customized sound stimulation restricted to the range of hearing loss).[46] Sensible modifications or updated content should be considered and encouraged if these might reasonably enhance our counseling strategy for DR-limited persons with sensorineural hearing loss or augment its application with alternative sound treatments. At this time, we are not aware of substantially new knowledge or theory to apply in counseling approaches for promoting DR expansion in the context of hearing loss. What has evolved since 2002, when S.G. devised our approach, is keen interest in and experimental study of auditory adaptive plasticity and gain processes,[39,40,47–50] and related efforts to place the resulting knowledge in theoretical frameworks and models to explain suprathreshold sound sensitivity problems, including hyperacusis.[35,41,51–53] Some of this information, in simplified form, might find utility for updating our counseling approach, enhancing the content, and providing a plausible conceptual basis to help the participant better understand his or her problem and the mechanisms of treatment. We look forward to seeing this and related counseling approaches, incorporating the underlying principles described in this article, applied in future research and, more importantly, in clinical practice to enhance hearing health care.

CONCLUSIONS

Formby et al described a successful intervention to expand the audiometric DR for loudness among individuals with sensorineural hearing losses and nominal sound tolerance complaints consistent with their lower to borderline-normal loudness discomfort judgments.[1,2]

These individuals were reasonably typical of hearing aid candidates whom one might routinely encounter in a traditional audiological/hearing aid practice. They represent one of the most challenging patient groups for audiologists to fit successfully with appropriate amplification because of their limited DRs and associated discomfort to amplified sounds. The intervention, based on sound therapy principles originally described by Hazell and Sheldrake,[7] and further described by Jastreboff et al,[4] Jastreboff and Jastreboff,[5] and Jastreboff and Hazell[27] for treating hyperacusis in tinnitus patients, included a focused counseling component. The structured counseling protocol targeted the adaptive plasticity of the auditory system as a key process mediating expansion of the hearing-impaired individual's limited DR. Portions of the structure and content of the counseling protocol were borrowed from counseling principles of TRT.[4,5,27] The first author (S.G.) used this protocol as it evolved over almost two decades at the University of Maryland Tinnitus and Hyperacusis Center to promote improved sound tolerance among tinnitus and hyperacusis patients with and without hearing loss.[3]

The resulting counseling protocol, as implemented by S.G. in Formby et al,[2] addressed the DR problem by parsing the nature and extent of each participant's sound tolerance issues. These issues were often associated with slightly lower-than-normal to borderline-normal LDLs, and his or her elevated hearing thresholds. Each participant was counseled individually in a single session lasting ~60 to 90 minutes; the length of each counseling session was dependent on the extent of the participant's involvement and interaction with S.G. Language and vocabulary were simplified, if necessary, for the individual, and visual aids specific to the structured counseling were used in counseling every participant. The following information was addressed during counseling in a checklist format: first, the purposes of the counseling and treatment were overviewed; then the audiological results were explained, including the hearing thresholds, results of speech testing, and the LDLs, including the adverse effects of a decreased DR on the comfortable use and benefits of amplification; the simplified anatomy and physiology of the auditory system were then presented; processing of sound at the level of the brain was described; the concepts of adaptive plasticity and regulation/modification of auditory pathway gain with low-level sound therapy were introduced; and, finally, treatment and outcome goals were discussed with each participant. Each individual's treatment and study-related questions were addressed and answered throughout the counseling session and at the end.

This simple, but promising, counseling approach, together with low-level broadband sound therapy, offers audiologists a structured protocol and clinical tool for treating often challenging and difficult-to-fit hearing aid candidates with reduced DRs and associated sound tolerance concerns. When properly used, this novel approach promoted DR expansion, leading to enhanced aided benefit and satisfaction, with potentially less reliance on signal-distorting compression processing of the speech signal and, thus, improved aided-sound quality.

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