# The Association Between Tinnitus and Posttraumatic Stress Disorder

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**Purpose:** Posttraumatic stress disorder (PTSD) affects nearly 10% of the population, a prevalence comparable with that of tinnitus. Similarities between the way PTSD and tinnitus influence auditory behaviors include exaggerated startle responses and decreased loudness tolerance. Tinnitus loudness is often exacerbated by sounds that trigger PTSD-related anxiety. This report addresses physical and psychological relations between PTSD and tinnitus.
**Method:** A chart review of veterans seen over a 4-year period for tinnitus services was conducted. Case history and self-assessments of tinnitus handicap were examined in all patients. A review of the literature related to triggers and effects of PTSD was conducted to explore potential consequences related to the presence of PTSD in the Veterans Affairs Medical Center (VAMC) tinnitus population.
**Results:** Chart review confirmed that 34% of the first 300 patients enrolled in the VAMC Tinnitus Clinic also carried a diagnosis of PTSD. Patient reports citing tinnitus severity, suddenness of tinnitus onset, sound-tolerance problems, and sound-triggered exacerbation of tinnitus were more common for patients with a PTSD diagnosis than patients with tinnitus only.
**Conclusions:** Several neural mechanisms linked to both tinnitus and PTSD affect auditory behaviors. Audiologists should be aware that patients with tinnitus and PTSD will require test protocols and referrals that address these powerful responses.

**Key Words:** posttraumatic stress disorder, hypervigilance, tinnitus, hyperacusis, limbic system, startle response

A pronounced need for tinnitus services prompted the opening of a specialized clinic in the Audiology Clinic at the James H. Quillen Veterans Affairs Medical Center (VAMC) in 2001. Activity in the clinic focused on the management of tinnitus-related complaints by considering self-assessments of tinnitus handicap and case history information. Patients received directed and interactive counseling pertaining to hearing loss, tinnitus generation, and coping strategies, as well as tinnitus masking devices with referrals to other services (e.g., psychology) when appropriate. The literature on tinnitus co-occurring with psychological disorders is substantial (for reviews, see Andersson, Baguley, McKenna, & McFerran, 2005; Erlandsson, 2000; J. L. Henry & Wilson, 2001). In addition to the more ubiquitous reports of depression, anxiety, and suicide ideation found in the tinnitus population, patients with posttraumatic stress disorder (PTSD) from a nonveteran population also presented a variety of tinnitus-related complaints (Hinton, Chhean, Pich, Hofmann, & Barlow, 2006). Ultimately, the need to address the presence of PTSD in the VAMC Tinnitus Clinic population was motivated by consistent patient reports that suggested links between tinnitus loudness and distress to PTSD-related anxiety.

### *Psychological Disorder and Tinnitus*

As reviewed by Stephens (2000), it was clear to medical historians of the Greco–Roman era through the Middle Ages, to Itard in 1821, and to the Fowlers in the 1940s and 1950s that several psychological factors co-occurred with tinnitus. Historically, the psychological conditions present with tinnitus most often included chronic depression, anxiety disorders, and suicide ideation. More recently, Folmer, Griest, and Martin (2002) compared self-assessed tinnitus severity using the Tinnitus Severity Index (TSI; Meikle, 1992); anxiety, as measured using the State-Trait Anxiety Inventory (Spielberger, 1998); and depression, as measured using the abbreviated Beck Depression Inventory (Beck & Steer, 1987). Their findings confirmed strong correlations among patients' perceived tinnitus severity, the perceived loudness

of the tinnitus, and the patients' levels of anxiety and effects of depression.

Psychological disorders and tinnitus when present concurrently have the potential to exacerbate one another (Andersson et al., 2005; Folmer et al., 2002; McKenna, 1998). To explain this unique situation, J. L. Henry and Wilson (2001) applied the cognitive theory of depression first advanced by Beck (1976) to the common emotional attributes displayed by the tinnitus population. Their review of the psychological literature illustrated that a depressed individual might develop and employ "inaccurate, illogical, negative appraisals of events and situations" that produced "cognitive distortions or misinterpretations of events" (p. 29). Such misinterpretations could generate attention and memory strategies favoring recall and awareness of negative elements of the environment. As a consequence, the individual suffering from tinnitus could develop an emotional disorder (i.e., feelings of depression or anxiety) triggered or heightened by an inappropriate interpretation of a sensory (i.e., tinnitus) event. To convey the deep disturbance experienced by some tinnitus patients, the authors suggested that the amalgam of emotional components, when accompanying tinnitus, produced *tinnitus distress*. Andersson et al.'s (2005) and J. L. Henry and Wilson's (2001) analyses supported a speculation made by Halford and Anderson (1991) that the mutual negative effects of a patient's psychological state and tinnitus were *bidirectional*. Additional evidence for tinnitus behaving as an arousal-reactive symptom, or as a symptom that is exacerbated by anxiety or stress as it, in turn, increases the patient's levels of stress and anxiety, was provided by Hinton et al. (2006) in a population of individuals with PTSD. Bidirectionality also was evident in studies comparing tinnitus severity with environmental-physical stressors such as chronic pain (Moller, 2000; Tonndorf, 1987), allergy (McFadden, 1982), insomnia (McKenna, 2000), and events that triggered strong autonomic nervous system or survival-type responses (Hazell, 1995a, 1995b).

Patients with tinnitus also have reported that aversive sounds increased distress caused by tinnitus, particularly when the presence or anticipation of such sounds increased a patient's stress or anxiety levels (Coles, 1995; Hazell, 1995a; Jastreboff & Jastreboff, 2000). Examples of abnormal responses to sound would include exaggerated startle responses, fear of sound (phonophobia), aversion to specific sounds (misophonia), and the experience of excessive loudness in the presence of sounds that would not be considered loud by normal hearing individuals (hyperacusis). In extreme cases, intense sound-related reactions could produce a cycle of sound avoidance compelling the patient to withdraw from social situations and day-to-day activities.

J. L. Henry and Wilson (2001) described the influences of memory and experience as additional potential contributors to a patient's powerful emotional and physical responses to tinnitus. They suggested that the tinnitus exacerbation reported by some patients, when in the presence of certain environmental sounds, depended at least in part on the sound-triggered recollection of past events that produced disturbing emotional states. Such associations would be particularly acute in those individuals whose tinnitus onset could be traced to a specific episode of exposure to a sound or traumatizing event, such as one that caused PTSD.

The possibility that traumatic episode recall could be linked to, or provoked by, tinnitus was described by Hinton et al. (2006). The investigators reported that 50% of the patients from a refugee population with a history that included traumatic stress suffered from disturbing tinnitus. The study's PTSD patients with tinnitus were rated as having more severe PTSD than those patients who did not experience tinnitus. In the affected patients, tinnitus was thought to trigger memories of specific trauma or to provoke thoughts of fear related to loss of individuality or "soul." The analysis demonstrated that patients' PTSD severity was affected more by these recollections and thoughts than by the patients' ratings of their tinnitus severity. Additionally, the investigators reported that flashbacks and intrusive memories contributed to tinnitus exacerbation, consistent with the concept of bidirectionality advanced by Halford and Anderson (1991).

PTSD is a psychiatric condition that affects members of civilian and military populations who have experienced, perpetrated, or witnessed life-threatening events (National Center for PTSD [NCPTSD], 2006). PTSD was first recognized as a unique psychological disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III*; American Psychiatric Association, 1980). Earlier editions of the *DSM* identified disorders, such as gross stress reaction, to describe the symptoms that affected individuals following or during exposure to extreme stress. The label attached by the public to PTSD also has changed over the years. For example, in the World War I era, this disorder was known as *shell shock,* whereas during World War II, the symptoms were identified as *combat fatigue* (Herman, 1997). One obvious manifestation of the cluster of symptoms comprising PTSD was observed as the emotional breakdown of otherwise normal people following combat exposure (Bremner, 2002). The previous diagnosis of gross stress reaction carried the assumption that a normal personality existed prior to the stressful event(s) and that the condition should resolve over time. Following the Vietnam War, this classification scheme was modified to account for more enduring changes in psychological state, physical arousal, and the integrity of neural structures.

A variety of stressful or traumatic events—such as motor vehicle accidents and physical, emotional, and/or sexual abuse—may be factors that can trigger the development of PTSD. Recent reports indicate that nearly 8% of Americans will experience an event that can cause PTSD at some point in their lives, with women (10.4%) twice as likely as men (5%) to develop the disorder (NCPTSD, 2006). Although PTSD is currently 10 times more common than cancer, it receives only approximately 10% of the funding devoted to cancer research (Bremner, 2002). The data set and discussion that follows are intended to assist audiologists with the identification, referral, and management of patients with PTSD who seek audiologic and tinnitus services.

The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) identified the potential causes and evaluation criteria for the diagnosis of PTSD. Specifically, the *DSM–IV*

indicated that two of the following five "symptoms of increased arousal" (p. 424) must be present for the diagnosis of PTSD:

1. difficulty falling or staying asleep,
2. irritability or outbursts of anger,
3. difficulty concentrating,
4. hypervigilance,
5. exaggerated startle response.

The symptoms listed by the *DSM–IV* also affect many patients with tinnitus regardless of PTSD complications. For example, Symptoms 1 (sleep disorder) and 3 (difficulty concentrating) are addressed specifically in several tinnitus intake forms, including one used for tinnitus retraining therapy (J. A. Henry, Jastreboff, Jastreboff, Schechter, & Fausti, 2002), the Tinnitus Handicap Inventory (THI; Newman, Sandridge, & Jacobson, 1998), the Iowa Tinnitus Questionnaire (Stouffer & Tyler, 1990), and the TSI (Meikle, 1992). Tinnitus case history typically would question patients regarding Symptoms 4 and 5 (hypervigilance and exaggerated startle) in relation to sound-tolerance problems and tinnitus exacerbation caused by environmental sound. Similarly, patients with tinnitus assessed in many studies indicated irritation and anger associated with or provoked by the tinnitus sensation (Andersson et al., 2005; Erlandsson, 2000; J. L. Henry & Wilson, 2001).

Another link between PTSD and tinnitus relates to the medications prescribed for the two disorders. For PTSD, the "front-line" treatment is selective serotonin reuptake inhibitors (SSRIs), which are a class of drugs also used with some success for patients with tinnitus (Andersson et al., 2005). If the two disorders share similar biochemical underpinnings, it is logical that SSRIs would affect tinnitus and PTSD in comparable ways. In addition to SSRIs, many medications used in the past for PTSD—such as benzodiazepines and tri-cyclic antidepressants—have been applied to the tinnitus population with varying degrees of success (U.S. Department of Veterans Affairs, 2002).

Because a connection between tinnitus and PTSD was suggested by anecdotal evidence obtained from a large number of patients in our clinic over several years of activity, a quantitative study was indicated to validate and confirm these observations. The possibility that PTSD could exacerbate tinnitus distress was investigated in the following two ways: (a) by conducting a chart review comparing case history information and data from self-assessment questionnaires and (b) with a review of the PTSD literature.

The chart review examined group differences between individuals who suffered from tinnitus without PTSD (T group) and those who reported tinnitus in addition to a preexisting diagnosis of PTSD (PTSD+T group). Data analysis considered items from case history related to tinnitus onset, exacerbation, loudness, and complaints related to sound-provoked discomfort. The effect of tinnitus on patient lifestyle was measured using self-assessments of tinnitus handicap and compared across the two groups. Audiologic (pure-tone thresholds) and demographic information (age, gender) also was compared.

## Method

A chart review considering case history and self-assessments of handicap from the first 300 veterans who were treated over a 4-year period for tinnitus services at the James H. Quillen VAMC was conducted to determine the presence and effects of PTSD. All appropriate institutional review board requirements of the VAMC and East Tennessee State University were met prior to initiation of the chart review.

Charted information included demographic information, audiologic case history, pure-tone thresholds, tinnitus case history, and information contained in self-assessment inventories, including the THI (Newman et al., 1998) and the TSI (Meikle, 1992). Each patient rated aspects of several auditory behaviors that were believed to be related to tinnitus. The patients' ratings of tinnitus loudness and reports of sound-tolerance problems were considered in the data analysis. When possible, loudness discomfort levels (LDLs) were obtained using a magnitude estimation procedure; however, not all the patients were able to perform the test because either they were uncomfortable sitting in the sound booth or they declined the test when it was described. The degree to which sound tolerance affected a patient was derived from case history and subjective ratings of the problem, and the criteria used to establish the severity of the tolerance problem are listed in Table 1.

At the James H. Quillen VAMC, the PTSD diagnosis was established through screening and intake examinations conducted by medical staff in the psychiatry and psychology sections. Patient-reported symptoms were evaluated using the *DSM–IV* (American Psychiatric Association, 1994). The Mississippi Scale for Combat-Related PTSD (Keane, Caddell, & Taylor, 1988) was used as an intake screening form to facilitate diagnosis. All diagnoses of PTSD were made by Veterans Affairs physicians and psychologists using at least these two instruments. Approximately 40% of the patients with PTSD were prescribed SSRIs for the disorder; however,

**Table 1. Degree of sound-tolerance problems.**

| Sound-related discomfort | Criteria for rating sound intolerance |
|---|---|
| None | Patient denies sound-tolerance problem. |
| Mild | Patient reports problem and rates discomfort at <5. |
| Moderate | Patient reports problem and rates discomfort at 5–7.5. |
| Severe | Patient reports problem and rates discomfort at >8. |

*Note.* The criteria for rating a patient's degree of sound-related discomfort were based on patient report and a subjective rating of the problem on a scale ranging from 1 to 10.

no patients indicated that the medication they took for PTSD ameliorated in a meaningful way their tinnitus. Rather, patients indicated that some functional aspects of their lives, such as sleep and irritability, were reduced without a concurrent reduction in the tinnitus distress.

For the purpose of analyzing the effect of PTSD on tinnitus, the patients were divided into two groups—those with tinnitus and PTSD (PTSD+T group) and those with tinnitus only (T group). The patient information was divided into the four categories that are listed in Table 2. The four categories include (a) demographic information; (b) audiologic information consisting of pure-tone averages (PTAs, dB HL; American National Standards Institute, 1996) at 500, 1000, and 2000 Hz; (c) prevalence of tinnitus-related patient complaints; and (d) patient self-assessments of tinnitus. Comparisons between groups were conducted using *t* tests for demographic, audiologic, and patient ratings of tinnitus loudness. Chi-square analyses were conducted on the data related to prevalence of specific complaints made by patients in the two groups. Because multiple *t* tests and chi-square analyses were run on the same data set, the alpha levels required for significance were set at $p < .001$ for all tests. One general observation that emerged from these data was the finding that tinnitus and PTSD conspired to produce disruption and handicap that influenced a broad spectrum of emotional, functional, and sound-related domains.

As detailed later, the patients affected by both tinnitus and PTSD presented with more severe symptoms and reported a greater self-assessed tinnitus handicap than did the patients who had tinnitus without PTSD. It was apparent that the patients with both tinnitus and PTSD required management and counseling that considered the exacerbating effects of PTSD on auditory behaviors in general and tinnitus in particular as well as the tendency of tinnitus to trigger PTSD-related anxiety. This article presents audiologic and tinnitus self-assessment data that distinguish patients with PTSD and tinnitus from patients who experienced tinnitus without PTSD.

## Results

Case history reports indicated that veterans associated tinnitus-related disruptions with recall of traumatic events,

**Table 2. Audiologic and tinnitus case history information.**

| Patient information | Tinnitus and PTSD | Tinnitus only |
|---|---|---|
| Demographic for 300 patients: *n* (%) | 103 (34) | 197 (66) |
| Gender | | |
| Men (*n*) | 100 | 194 |
| Women (*n*) | 3 | 3 |
| Age (years) | | |
| *M* | 57.1 | 59.8 |
| *SD* | 7.4 | 10.9 |
| Range | 30–83 | 23–85 |
| Audiologic: Pure-tone average (500, 1000, 2000 Hz) | | |
| Right ear (dB HL) | | |
| *M* | 28.7 | 28.7 |
| *SD* | 13.0 | 15.6 |
| Left ear (dB HL) | | |
| *M* | 31.8 | 31.0 |
| *SD* | 15.9 | 15.3 |
| Tinnitus-related patient complaints | | |
| Persistent sound-tolerance problems[a] (%) | 66.3 | 20.4 |
| Exacerbation of tinnitus following noise exposure[a] (%) | 68.0 | 40.6 |
| Discomfort provoked by impulse/unexpected sounds[a] (%) | 64.4 | 31.0 |
| Sleep affected by tinnitus[a] (%) | 93.2 | 80.7 |
| Concentration affected by tinnitus[a] (%) | 93.2 | 78.4 |
| Quiet activities affected by tinnitus (%) | 91.3 | 82.6 |
| Patient self-assessments | | |
| Sudden onset[a] (%) | 51.5 | 24.3 |
| Rating of tinnitus loudness (from 1 to 10)[a] | | |
| *M* | 7.6 | 6.4 |
| *SD* | 2.0 | 2.6 |
| Rating of sound-tolerance problems (from 1 to 10)[a] | | |
| *M* | 7.1 | 4.4 |
| *SD* | 2.7 | 3.4 |
| Tinnitus Handicap Inventory total score[a] | | |
| *M* | 62.9 | 45.5 |
| *SD* | 21.1 | 23.4 |
| Tinnitus Severity Index total score[a] | | |
| *M* | 42.1 | 34.9 |
| *SD* | 8.5 | 10.2 |

*Note.* PTSD = posttraumatic stress disorder.

[a]Significant at $p < .01$.

nightmares, and exaggerated startle responses to unexpected sounds that interfered with patients' daily activities. These patients also reported that tinnitus loudness was exacerbated during such periods of stress. The condition linking these symptoms, and eventually many others, was evident in previous diagnoses and service connections for PTSD. Approximately 34% of the 300 patients studied carried a diagnosis of PTSD (the PTSD+T group) prior to their enrollment in the VAMC Tinnitus Clinic. The remaining 66% of the patients composed the tinnitus-only group (T group).

Table 2 shows the comparisons between the responses of patients in both groups. The group characteristics reported on Table 2 were obtained in response to a variety of intake questions drawn from tinnitus case history (J. A. Henry et al., 2002) and self-assessments of tinnitus handicap, including the THI (Newman et al., 1998) and the TSI (Meikle, 1992). For several items, the groups were compared using two-tailed *t* tests. There were a total of five such tests, a situation that was addressed by adjusting the alpha level to .002 in each test, consistent with a level for all tests of .01 (Rao, 1998).

### *Patient Information: Demographics*

Although it was anticipated that patients enrolled in the VAMC Tinnitus Clinic would suffer to some extent from psychological disorder, the prevalence of patients with PTSD was unexpected. All but 6 of the patients were male. Table 2 lists the mean age, standard deviations, and age ranges, which were similar for the PTSD+T and T groups.

### *Patient Information: Pure-Tone Sensitivity*

The mean PTAs (and standard deviations) at 500, 1000, and 2000 Hz indicated there were no differences between groups or between ears in either group. In some cases, asymmetric hearing loss was found in patients with unilateral tinnitus or tinnitus that was lateralized to the ear with the poorer sensitivity; these cases were extremely rare in our patient groups.

### *Tinnitus-Related Patient Complaints*

The patients reported problems related to tinnitus by completing self-assessments of tinnitus handicap and case history. These data are summarized in Table 2. Specific disturbances reported by patients with PTSD were associated with a variety of sounds—in particular, sudden, unexpected, impulse sounds (such as fireworks, plates or silverware being dropped, and cars backfiring). Patients from both groups also reported strong aversions to warning signals, such as sirens. Among the most personal complaints, patients with both PTSD and tinnitus described extreme physical discomfort when in the presence of young children (often grandchildren) who talked or yelled at one another at a level that was loud but not above the level typically produced by children playing. The patients with PTSD indicated that powerful startle responses produced by unexpected sounds precluded their attendance at social gatherings and functions. They also acknowledged that an aversion to groups of people was a general consequence of PTSD.

When the patients rated their ability to tolerate environmental sounds, members of both groups indicated that they experienced discomfort when exposed to moderate levels of sound or to sounds that did not appear to bother friends and relatives. Table 2 contains the prevalence of these problems among the patient groups. The patients in the PTSD+T group were more than three times as likely as the patients in the T group (66.3% vs. 20.4%; $p < .01$) to report persistent and frequent sound-tolerance problems. Similarly, patients with tinnitus and PTSD were more likely (68.0% vs. 40.6%; $p < .01$) to indicate that tinnitus was exacerbated in the presence of sounds that produced physical discomfort.

Although reduced loudness tolerance affected both groups, the prevalence of tinnitus exacerbation in the presence of loud sounds was reported more frequently by patients with tinnitus and PTSD than by patients with only tinnitus (see Table 2). More than 67% of the PTSD+T group reported an increase in their tinnitus annoyance when startled, whereas 40% of the T group had the same complaint. Unexpected impulse sounds were noted specifically by 64.4% of the PTSD+T group as most likely to exacerbate tinnitus loudness. The same observation was made by only 31% of the T group. The most likely patients in the PTSD+T group to indicate that unexpected impact sounds were difficult to tolerate were the same patients who reported that such sounds exacerbated tinnitus annoyance.

Patients with tinnitus PTSD were more likely to indicate sleep disruption, concentration problems, and difficulty relaxing and enjoying quiet activities than the patients with tinnitus only. Of these activities, sleep (93.2% vs. 80.7%; $p < .01$) and concentration (93.2% vs. 78.4%; $p < .01$) reached significance.

### *Patient Self-Assessments*

The patients in the PTSD+T group reported their tinnitus as more disturbing than did the members of the T group when examined using self-assessment of handicap questionnaires (Meikle, 1992; Newman et al., 1998) and case history items. The patient interviews and questionnaires were compared using a chi-square analysis. The results are indicated by superscript letters in Table 2 that signify statistically significant differences between group responses to questionnaire items. The analysis confirmed that the PTSD+T group patients perceived their tinnitus as more severe and more disruptive to daily activities than did the patients in the T group.

Sudden-onset tinnitus was reported twice as often by patients in the PTSD+T group than by those patients in the T group (51.3% vs. 24.5%; $p < .01$). The severity of tinnitus in most sudden-onset cases reportedly did not change over time. The patients indicated the tinnitus and the disruption caused by the tinnitus decades after onset were as powerful as when the tinnitus was first observed. Almost all of the patients with tinnitus and PTSD, regardless of tinnitus onset, reported that hearing tinnitus reminded them of traumatic experiences whose memory produced anxiety and seemed to increase the perceived tinnitus loudness.

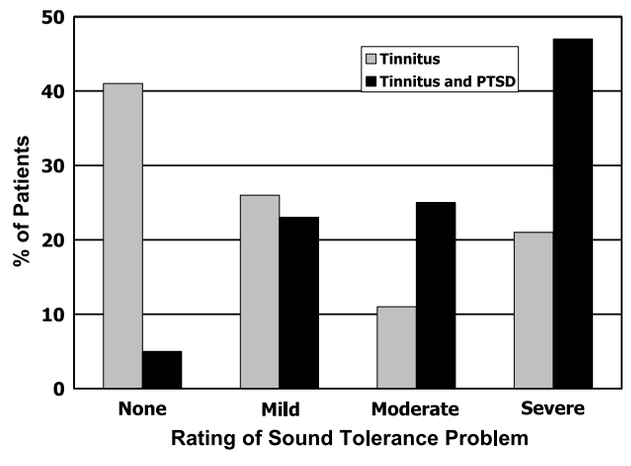
Similarly, when the patients rated the perceived loudness of tinnitus on a 1 – 10 scale, a Student's *t* test indicated that

the patient ratings of tinnitus loudness were higher in the PTSD+T group than in the T group (7.6 vs. 6.4; $t = 4.05$; $p < .01$). The loudness estimates, when considered with the suddenness of tinnitus onset, revealed that the patients with tinnitus and PTSD experienced tinnitus of greater loudness that was less likely to decrease over time than those tinnitus patients without PTSD.

The patients with PTSD were also more likely to rate sound-tolerance problems as more severe when asked to rate the discomfort produced by common sounds on a scale ranging from 1 (*no discomfort*) to 10 (*as uncomfortable as any sound can make you feel*). When scaling their loudness discomfort, the PTSD+T group members rated their experience as more physically disturbing than the T group members. A *t* test comparing the mean ratings that appear in Table 2 (7.1 vs. 4.4 of 10; $t = 8.74$; $p < .01$) indicated the PTSD+T group was more likely to rate themselves as disturbed by problems tolerating sound than the T group. The patients' ratings of disturbance then were combined with patient reports of sound-provoked discomfort in response to a variety of environmental sounds. Table 1 summarizes the criteria used to determine the degree of the patients' difficulties tolerating loud sounds. The two components specified in Table 1, the numerical ranking and the patient reports of a problem, yielded a rating of sound-tolerance severity. Figure 1 details the results of this analysis in terms of the degree to which sounds provoked discomfort in patients. These subjective data showed that the patients in the PTSD+T group rated their sound-tolerance problems as more severe than patients in the T group.

The impact tinnitus exerted on physical, emotional, and functional aspects of a person's life was assessed on the THI (Newman et al., 1998), TSI (Meikle, 1992), and tinnitus case history forms. Table 2 contains means and standard deviations on the THI and the TSI at intake for all 300 patients. A Student's *t* test indicated that self-assessed tinnitus handicap on both indices was significantly greater among the tinnitus patients with PTSD than the patients with only tinnitus (THI: $t = 6.27$; TSI: $t = 5.89$, $p < .01$). The THI scores in particular suggested perceived difficulties in patients' emotional responses to the tinnitus sensation and corroborated patient complaints related to problems with sleep, relaxation, concentration, and communication. Table 3 details this finding, as the averages for all 25 items on the THI were higher in the PTSD+T group than the T group. It is interesting that the two items yielding the smallest group differences were the items that were rated as either most handicapping (Item 19: Do you feel that you have no control over your tinnitus?) or least handicapping (Item 11: Because of your tinnitus, do you feel that you have a terrible disease?).

**Figure 1. Severity of sound-tolerance problems as reported by patients on case history and by subjective rating (see Table 1 for criteria). PTSD = posttraumatic stress disorder.**




The four largest group differences on the individual THI items were recorded for the Emotional subscale, and seven of the nine largest group differences were recorded for items associated with the emotional response category. The data in Table 3 show that these four group differences related to, in descending order of importance, tinnitus that produced feelings in patients of depression (Item 21), being upset (Item 16), anger (Item 3), and anxiety (Item 22). The patient groups also were distinguished by the level that tinnitus reportedly affected concentration (Item 1).

Additionally, the item analysis revealed that four of the five smallest group differences were recorded for items that were considered by patient rankings to be among the most handicapping aspects of tinnitus. That is, regardless of PTSD involvement, the following four aspects of tinnitus handicap affected patients in both groups to the same degree: the sense that they could not control tinnitus (Item 19), the sense that they could not escape tinnitus (Item 8), the belief that tinnitus affected hearing (Item 2), and the belief that the presence of tinnitus affected sleep (Item 7). One other item produced a similar response for the two groups; most patient responses contraindicated a belief that tinnitus signified terrible disease (Item 11). Additionally, the three smallest group differences were found for items from the Catastrophic subscale. Although Baguley and Andersson (2003) demonstrated that the three subscales of the THI (Emotional, Functional, and Catastrophic) were not statistically independent of one another, the group differences present in the current data set indicated that the organization of subscales might be appropriate for investigating tinnitus handicap reported by specific tinnitus patient groups, such as those with PTSD.

The comparison between patient pure-tone thresholds, which were not statistically different between groups, and the THI scores, which were different, suggested that the effect of PTSD on patients' assessment of tinnitus handicap and case history reports was not attributable solely to differences in hearing sensitivity. Figure 2 is a bivariate plot of the patient PTAs (ordinate) and the THI scores (abscissa) at intake. The large filled symbols depict the mean data for the PTSD+T group (square) and the T group (circle). Both the PTAs and THI scores displayed substantial variability, and the comparison of means demonstrated that self-assessed handicap between the two groups was independent of sensitivity. Perhaps the most informative statement regarding Figure 2 was that only 4 patients in the PTSD+T group rated themselves

**Table 3. Group mean scores from the Tinnitus Handicap Inventory (THI) broken down by item.**

| THI item | PTSD+T group | Tinnitus group | Group difference |
|---|---|---|---|
| F1–Concentration | 3.3 | 2.4 | 0.9 |
| F2–Hearing | 3.2 | 2.8 | 0.4 |
| E3–Angry | 2.6 | 1.6 | 1.0 |
| F4–Confused | 2.0 | 1.4 | 0.6 |
| C5–Desperate | 1.8 | 1.1 | 0.7 |
| E6–Complain | 2.1 | 1.2 | 0.9 |
| F7–Sleep | 2.8 | 2.5 | 0.3 |
| C8–Escape | 3.5 | 3.1 | 0.4 |
| F9–Social | 2.7 | 1.8 | 0.9 |
| E10–Frustrated | 3.3 | 2.4 | 0.9 |
| C11–Disease | 0.8 | 0.6 | 0.2 |
| F12–Enjoy life | 2.6 | 1.7 | 0.9 |
| F13–Household | 1.8 | 1.2 | 0.6 |
| E14–Irritable | 2.8 | 2.1 | 0.7 |
| F15–Reading | 2.3 | 1.6 | 0.7 |
| E16–Upset | 2.9 | 1.9 | 1.0 |
| E17–Relationships | 2.5 | 1.7 | 0.8 |
| F18–Focus attention | 2.8 | 2.1 | 0.7 |
| C19–Control | 3.6 | 3.3 | 0.3 |
| F20–Tired | 2.2 | 1.7 | 0.5 |
| E21–Depressed | 2.8 | 1.6 | 1.2 |
| E22–Anxious | 2.5 | 1.5 | 1.0 |
| C23–Cope | 1.4 | 1.0 | 0.4 |
| F24–Stress | 2.6 | 2.1 | 0.5 |
| E25–Insecure | 1.9 | 1.0 | 0.9 |
| F = Functional Subscale (11 items) | 28.4 | 21.4 | 7.0 |
| E = Emotional Subscale (9 items) | 23.3 | 14.9 | 8.4 |
| C = Catastrophic Subscale (5 items) | 11.2 | 9.1 | 2.1 |
| Total THI | 62.9 | 45.4 | 17.5 |

*Note.* The item numbers are preceded by a letter (either F, E, or C) corresponding to the Functional, Emotional, and Catastrophic subscales. PTSD+T group = individuals who reported tinnitus in addition to a preexisting diagnosis of posttraumatic stress disorder.

at lower than a 36/100 on the THI, whereas nearly 40% of the T group yielded a score on the THI below 36.

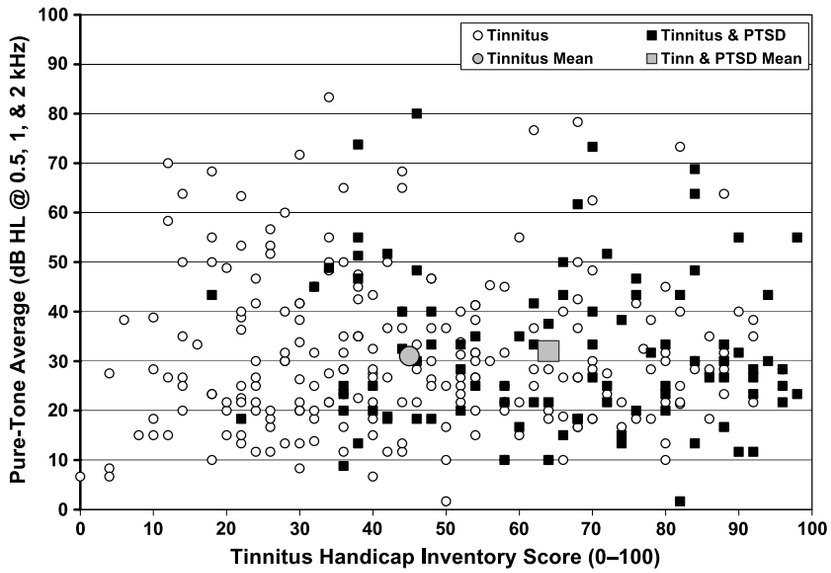
## Discussion

The frequency of PTSD co-occurring with tinnitus was observed through consistent patient reports obtained through years of clinical activity. The patients with tinnitus and PTSD (PTSD+T group) were more likely to present with the most severe tinnitus symptoms and sound-tolerance problems than were patients with only tinnitus (T group). That tinnitus and PTSD could be linked to one another or could interact in ways that exacerbated both disorders was an idea expressed by many patients, not just in the current chart review but also in a population of refugees interviewed by Hinton et al. (2006). Approximately two thirds of the patients in the PTSD+T group reported tinnitus exacerbation in the presence of sounds that also triggered PTSD-related symptoms, such as feelings of stress and anxiety. The PTSD+T group patients were also more likely to report sudden tinnitus onset, and they responded to self-assessment inventories with significantly greater perceived handicap than tinnitus patients who did not suffer from PTSD.

A search of the PTSD literature was initiated in the attempt to identify a physiological connection between tinnitus and PTSD, and this review provided substantial evidence for neural mechanisms common to both. Specifically, limbic system structures were cited as essential contributors to the generation and perpetuation of these two disorders. Further, the intrusive nature of both disorders could be augmented by a patient's misconceptions regarding the potential threat posed by sound in general and the tinnitus sound in particular. Aspects of the tinnitus may be misinterpreted, thereby provoking in patients the sense that they cannot control the function of their auditory system or, in the words of many patients, that they lose control over their own "mind." For patients in the PTSD+T group in particular, the lack of control was associated with sound-related sources of physical discomfort and annoyance (including the tinnitus sound). Patient complaints were consistent with powerful arousal responses caused not only by startling sounds but by the sensation that the tinnitus was an inescapable entity that required constant monitoring.

The discussion that follows provides information relevant to audiologists who test and treat patients suffering from PTSD because such patients may require modifications to audiologic diagnostic strategies. A review of neural mechanisms associated with both tinnitus and PTSD follows the brief discussion of clinical implications for audiologists treating these patients. This review supports the notion that tinnitus and PTSD interact by affecting similar central nervous system (CNS) mechanisms and auditory behaviors

**Figure 2. Scatterplot comparing pure-tone averages at 500, 1000, and 2000 Hz with intake scores on the Tinnitus Handicap Inventory.**




and that ultimately the understanding of one disorder may facilitate treatment and management strategies for the other. One rationale for this brief review is the hope that clinicians and researchers who work with tinnitus patients can tailor their management strategies by incorporating interventions and counseling elements previously shown to reduce the intrusiveness of PTSD symptoms.

## *Audiology Clinic Implications*

The audiologic test suite is among the most aversive environmental settings for individuals with tinnitus and PTSD. Patients with tinnitus generally do not like the quiet environment of the booth, and many PTSD patients do not like being closed into small areas from which there is no easy escape. The sounds that comprise basic audiologic evaluation may provoke discomfort because both T and PTSD+T group patients tended to dislike tonal signals, especially high-pitched tones. Clinicians should be sensitive, therefore, to the stress associated with threshold assessment experienced by patients with tinnitus. Consider the basic evaluation, during which silence is interspersed at random intervals with annoying signals and an occasional human voice presented at a relatively loud conversational level, usually with no warning, as a difficult undertaking for a substantial proportion of patients.

Highlighting the need for flexibility during a hearing evaluation, a demographic study and review by Nelting (2002) reported that the prevalence of potentially debilitating sound-tolerance problems ranged from 2% in the general population to more than 85% when assessed among patients enrolled in tinnitus clinics. Patients with PTSD often report exaggerated startle responses, related by patients to loudness tolerance issues. The *DSM–IV* (American Psychiatric Association, 1994) diagnostic criteria for PTSD and prevailing CNS-based models of tinnitus (Cacace, 2003; Jastreboff, 1999; Kaltenbach, Zhang, & Finlayson, 2005) have attributed disturbances related to each disorder at least in part to enduring changes in CNS arousal and behavior. Associated hyperarousal would have the potential to affect several components of the test battery, including suprathreshold measures of speech recognition, the aural–acoustic immittance battery, and high-level electrophysiological testing.

Patients' sound-tolerance problems also may limit assessment of amplification options and contribute to exaggerated startle responses during real-ear measurement and evaluation of LDLs. All patients with PTSD indicated that they would not tolerate the use of random stimulus levels during LDL measurement; therefore, some established loudness measuring techniques (e.g., Hawkins, Walden, & Montgomery, 1987) either could not be used or must be modified for this population. In our experience, establishing a loudness standard to conversational speech and then employing magnitude estimation using stimuli raised by small increments in level was the most acceptable method for determining loudness growth in the patients with tinnitus and PTSD. However, even with an agreeable test strategy, uncomfortable loudness experienced by patients with PTSD occasionally precluded completion of LDL testing.

Loudness tolerance issues also demand care when assessing acoustic reflexes and when administering word-recognition tests at high presentation levels. It is recommended that acoustic reflex testing be conducted at the end of the evaluation to avoid strong negative reactions to the stimuli and minimize the potential for sound-provoked tinnitus exacerbation that could subsequently influence the measurement of pure-tone thresholds or word-recognition ability.

Negative reactions to moderately loud or sudden sounds may increase the difficulty of adjusting to personal amplification for patients with PTSD and tinnitus. Loud or large social gatherings compel some patients to shut off their hearing aids, thereby enabling the patients to use the hearing aids as earplugs. Hearing aid output must be controlled carefully for such patients, and it is probable that the most appropriate electroacoustic characteristics for hearing loss may not be the optimum setting for a patient with tinnitus or a patient with PTSD (Searchfield, 2006). In cases with patients who complain of exaggerated startle responses, a device with multiple memories should include a program for use in environmental settings conducive to unexpected sounds. Ideally a program that assured the patient of a maximum output that would not trigger an aversive response would raise the probability that the patient would accept and use the hearing aid.

It is imperative for the audiologist to be aware of the signs that a patients suffers from PTSD, suicide ideation, depression, or other psychological disorders. Case history or previously charted patient information must also be considered when planning test strategies for patients thus affected. These disorders should alert the audiologist to communicate with other professionals and implement an interdisciplinary treatment approach when planning a management strategy (J. L. Henry & Wilson, 2001).

### *Neural Mechanisms Associated With PTSD and Tinnitus*

Reviews of imaging studies conducted on patients with either tinnitus or PTSD revealed that both conditions were associated with, or produced, physical changes to the thalamus, the hippocampus, and the amygdala (Bremner, 1999, 2002, 2005; Cacace, 2003; Herman, 1997; Jastreboff & Jastreboff, 2000; Lockwood et al., 1998; Shulman et al., 1995). Several studies by Bremner and colleagues demonstrated reduced blood flow and subsequent reduced hippocampal volume in abuse and trauma victims. Because the hippocampus is implicated in functions ranging from simple memory tasks to the development, learning, and retention of complex emotional coping strategies, traumatic stress could exert long-term effects on the ability to cope with novel sources of stress. As Folmer, Griest, Meikle, and Martin (1999); Fowler (1948); J. L. Henry and Wilson (2001); McKenna (1998); Sweetow (1986); and others have asserted, tinnitus could be construed as such a stress-provoking sensory disturbance.

In addition to the hippocampus, the amygdala's activity is most often implicated in patients with tinnitus (Jastreboff & Jastreboff, 2000; Lockwood et al., 1998) and PTSD (Bremner, 2002, 2005; Herman, 1997). Bremner, Herman, and others (e.g., Kandel & Schwartz, 1981) have reported the amygdala's influence on a patient's reactions to stress and trauma produced by sensory events that triggered fear. In general, the more pronounced the fear response, or the more threatening the stimulus, the greater the activity found in the amygdala. However, the situation is complicated by the fact that activity in the amygdala may also be implicated in the extinction of fear responses when activated by the medial prefrontal cortex (Sotres-Bayon, Bush, & LeDoux, 2004). Therefore, terms—such as *limbic system*—when used to describe the location of the mechanisms responsible for the fear response are likely an oversimplification of a set of complicated and subtle pathways. For the purposes of our discussion, we intend the use of *limbic system* to facilitate consideration of the current results in the context of reports from previous studies and reviews.

Although it was not always the case that a single traumatic event would generate overwhelming and persistent neural activity, ongoing exposure to disturbing stressful and traumatic stimuli increased the likelihood of the permanent CNS changes associated with PTSD (Bremner, 2002). Negative emotional responses resulting from disordered or modified CNS behavior was attenuated in some, but not all, individuals if contextual information from the environment was inconsistent with the sensory disturbance. Bremner (2002) asserted the importance of cortical mediation in response to stress-provoking environmental stimuli. He asserted that a patient's inability to consciously mediate a negative emotional response could cause an individual to misinterpret environmental events and experience distress when encountering sensory events that more accurately should be perceived as neutral or nonthreatening.

For example, a scene of violence in a movie should produce a different emotional response from that exhibited following exposure to the same visual and auditory stimulus experienced as an event in real life. Conscious awareness that the graphic violence was being shown on a screen should inhibit the emotional response otherwise appropriate for the sensory input associated with violent or troubling visual and auditory images integrated by the thalamus and sensory mechanisms. Such attenuation of the response produced by disturbing images was less likely to occur in a PTSD patient for whom the images might trigger overwhelming sensations of fear or memories of traumatic events.

Bremner (2002) reviewed several neural imaging studies in an attempt to identify the source of such misinterpretations characteristic of PTSD patients. He reported that combat veterans with PTSD exhibited abnormally low levels of activity in the prefrontal cortex when presented with images reminiscent of trauma experienced during combat. In one study, veterans were shown photographs and presented with audio recordings of combat episodes while they were seated in a laboratory environment. When scanned using positron emission tomography, the veterans' limbic systems displayed significantly stronger activity than a control group consisting of combat veterans without PTSD. The group differences were consistent with a stronger fear response in the PTSD group, even though the individuals in both groups were consciously aware they were in a laboratory environment. It was also noted that the prefrontal cortex region of the PTSD patients was not as active as in control participants viewing the same material. Bremner described the difference as one in which the "higher" cortical areas failed in PTSD patients to "inhibit the more primitive lower brain areas" (p. 133). The participants with PTSD, therefore, experienced powerful and overwhelming emotional responses generated by the "lower brain" structures of the thalamus and limbic system even when consciously aware that they were in an environment that posed no overt danger.

Another aspect of long-term changes to the CNS resulting from exposure to traumatizing stimuli or events was revealed in measurements of hippocampus function. The hippocampus, implicated in the learning and memorizing of environmentally sensitive coping strategies, should contribute substantially to the generation and maintenance of appropriate responses to stressful stimuli. The actions that allowed an individual to cope successfully with difficult or threatening situations should be learned and stored for recall, particularly those that allow the individual to withstand the most demanding survival situations. Bremner (2002) reported that the consequences of trauma-related hippocampus damage included impairment of information recall immediately following an event or an unwanted recall of similar events that took place in the past. Additionally, the impaired individual may be unable to cope with situations that demand accurate recall of events for the planning and execution of appropriate motor and emotional responses. In other words, individuals with hippocampal damage will have a difficult time learning new coping strategies or adapting familiar coping strategies to deal with new and difficult situations or sensations, particularly when such situations are reminiscent of negative events stored in memory.

The ability to cope with stressful situations relies at least in part on an accurate analysis of the sensory event provoking the stress. Poor coping abilities of patients with tinnitus and PTSD may be produced by an inaccurate assessment of the patient's surroundings. As indicated by J. L. Henry and Wilson (2001), much of the distress experienced by a patient with tinnitus may be generated by a misinterpretation of the event—the patient's inaccurate evaluation of the signal as a symptom of serious illness, psychological disease, or impending deafness. Patients with tinnitus and PTSD tend to perceive sensory inputs as provocative and threatening, and likewise they experience a stress response to the perception of the tinnitus sound. The effects of stress could then impair coping strategies, thereby making the individual more susceptible over time to continued disturbance.

All combat veterans are at risk for developing noise-induced (temporary or permanent) hearing loss and tinnitus from exposure to high sound pressure levels under extremely stressful, perhaps life-threatening, conditions. The exposure to traumatic events could produce a variety of psychological sequelae (Kulka et al., 1990). If the tinnitus onset were associated with a dire event or a survival moment, then the tinnitus sound might trigger memories associated with that specific event. Hinton et al. (2006) reported such a finding in the majority of the PTSD patients with comorbid tinnitus. Additionally, stress, anxiety, and memories associated with changes in tinnitus loudness were reported by many of our patients, and upon specific questioning, almost invariably were identified in patients who carried a diagnosis of PTSD. Sounds that provoked such responses were often identified as sudden, unexpected, impulse noises (such as dropped silverware or a car backfiring) that did not reach damaging sound pressure levels (see Table 2).

Because PTSD can produce blatant sound-related disruptions, it would follow that tinnitus patients would experience substantial distress when a specific event or sequence of events triggered PTSD concurrent with the initial detection of a tinnitus signal. These patients could reexperience traumatic memories associated with the initial appearance of tinnitus or an exacerbation of its loudness. The tinnitus sound experienced by a patient would be linked by memories to the triggering event(s), and a patient thus affected would display markedly different tinnitus-related disturbances from those patients whose tinnitus was not directly related to memories of trauma. Hinton et al. (2006) also demonstrated that PTSD complaints were influenced more by the degree to which tinnitus affects a patient's self-image and "soul" than by the patient's estimate of the tinnitus loudness.

In conclusion, although tinnitus and PTSD involve distinct perceptual events, they may share many CNS mechanisms, particularly those comprising the limbic system and auditory subcortical pathways. When present concurrently in an individual, their effects can be staggering, reducing dynamic range, exacerbating startle responses, and producing aversive and uncontrollable physical and emotional responses to sound. Audiologists should be aware of the *DSM–IV* criteria for evaluation and diagnosis of PTSD and should be prepared to refer to psychologists, psychiatrists, and/or mental health providers when PTSD is suspected in a patient seeking audiologic service.

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