One-Year Audiologic Monitoring of Individuals Exposed to the 1995 Oklahoma City Bombing

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Abstract

This longitudinal study evaluated subjective, behavioral, and objective auditory function in 83 explosion survivors. Subjects were evaluated quarterly for 1 year with conventional pure-tone and extended high-frequencies audiometry, otoscopic inspections, immittance and speech audiometry, and questionnaires. There was no obvious relationship between subject location and symptoms or test results. Tinnitus, distorted hearing, loudness sensitivity, and otalgia were common symptoms. On average, 76 percent of subjects had predominantly sensorineural hearing loss at one or more frequencies. Twenty-four percent of subjects required amplification. Extended high frequencies showed evidence of acoustic trauma even when conventional frequencies fell within the normal range. Males had significantly poorer responses than females across frequencies. Auditory status of the group was significantly compromised and unchanged at the end of 1-year postblast.

Key Words: Acoustic trauma, auditory blast injury, extended high frequencies, noise-induced hearing loss, tinnitus

Abbreviations: ANOVA = analysis of variance, CF = conventional frequencies (0.25–8 kHz), EHF = extended high frequencies (10–20 kHz), LSD = least significant difference, psi = air pressure measured in pounds per square inch, dB pSPL = decibel re: peak sound pressure level, Q = quarterly evaluations, TNT = trinitrotoluene

Classic blast overpressure resulting from an explosion or firing of a large caliber weapon is characterized by a sharp rise in atmospheric pressure followed by a supersonic positive pressure wave and then by a subatmospheric phase (Kerr and Byrne, 1975; Phillips, 1986; Yelverton et al, 1996). Subsequent internal injuries, termed “primary blast injury,” predominantly are localized to hollow organs of the ear, lung, and gastrointestinal tract. Medical experience with this type of event mainly has been in the military purview; however, in an era with terrorist activity, civilians also confront the risk of blast-related trauma. Consequently, civilian medical personnel can face challenges in the assessment and treatment of blast survivors, for which they may not be prepared adequately. The topic of aural blast injury merits forethought since proper understanding of the pathology will determine quality of patient care.

A blast wave is particularly damaging to the ear since pressure variations exceed the physical limits of auditory structures (Hamit, 1973; Ziv et al, 1973; Phillips, 1986; Frykberg and Tepas, 1988; Mellor, 1988). Although this is well documented, comprehensive and conclusive statements regarding the functional nature of the injury are difficult to make due to a scarcity of systematic audiology investigation. Previous reports are exceedingly disparate in
methodology and terminology, both within and between studies, or focus primarily on otologic sequelae. Additionally, group data often are derived from subjects exposed to diverse explosive sources, including gunfire. Of seven papers that report findings from a single explosion, four delineate mostly retrospective reviews and anecdotal data (McReynolds et al., 1949; Kerr and Byrne, 1975; Pahor, 1981; Bruins and Cawood, 1991). Only three studied audiologic deficits in a planned manner (Singh et al., 1983; Shupak et al., 1993; Chandler and Edmond, 1997).

While these three investigations make important contributions to the literature, sample sizes were small (5–14) and, except for Chandler and Edmond (1997), audiometric protocols and/or follow-up were not defined clearly. No study has employed a comprehensive audiologic test battery, including extended high frequencies (EHFs), nor accounted for aging effects in a civilian population, nor inferentially analyzed change over time, gender variations, or case history effects.

The April 19, 1995 bombing of the Oklahoma City Federal Building provided an opportunity to methodically evaluate auditory sequelae of blast exposure in a large, diverse, civilian population. The 4800-pound ammonium nitrate truck bomb was one of the most powerful civilian explosions to date, resulting in 592 documented injuries and 167 deaths (Malonee et al., 1996). The purpose of this multisite, longitudinal investigation was to assess systematically behavioral, subjective, and objective auditory function in a large sample of blast survivors in order to document and analyze patterns of recovery or deficit.

**METHOD**

Experimental studies of blast injury are done with controlled detonation of a known substance within discrete distances and physical environments. With unexpected civilian explosions, systematic postblast study has inherent limitations due to time constraints, unknown exposures, and nonexistent preblast data. Methodology was designed to describe sequelae as accurately as possible and to archive results for reference in the event of similar future blasts.

**Subjects**

To reduce bias toward inflated deficit figures, two aspects of subject recruitment were implemented: (1) survivors from the downtown area that sustained 90 percent of the structural damage (roughly 0.9 mile east to west by 1.3 miles north to south) were asked to participate, whether or not they had auditory complaints; (2) subject recruitment included community announcements as well as referrals from physicians and personal assistance programs.

Eighty-six subjects signed a written informed consent to participate; however, three were eliminated due to nonorganic signs (described later). Ages ranged from 3 to 70 years (mean = 43 years). Forty-five percent of subjects were female and 55 percent were male. The average ages were 40.7 years for females and 45.6 years for males, which were not significantly different. Subject distance from the blast was equivalent between genders.

A representative sample of 10 normal-hearing subjects was included to document hearing threshold stability in nonblast-exposed individuals over a 6-month period—the period in which blast-related changes were expected (Korkis, 1952; Bruins and Cawood, 1991). All had normal auditory sensitivity (i.e., ≤ 20 dB HL from 0.25–8 kHz, including 3 and 6 kHz); negative history for otologic pathology, neurologic pathology, or noise exposure; and were not within the designated area on April 19, 1995. The group had equal gender distribution and ranged in age from 23 to 29 years (mean = 26.1 years).

**Stimulus**

Individual sound and air pressure exposure levels were unascertained and not feasible to estimate due to unknown explosive specifications and production of complex waves as the blast interacted with reflective surfaces or entered enclosed areas. To provide a reference, however, air pressure and peak sound pressure levels (dB pSPL) were calculated for a classic blast wave generated by a trinitrotoluene (TNT)-equivalent charge for distances from 5 to 1500 feet from ground zero (Wilfred Baker Engineering, Inc.). Figure 1 depicts side-on and reflected sound pressure levels. Side-on incident blast overpressure ranged from 3000 pounds per square inch (psi) at 10 feet to 25 psi at 100 feet. Corresponding decibel levels were approximately 235 dB pSPL and 199 dB pSPL, respectively. Reflected estimates are higher. Overpressure duration was approximately 2.8 msec at 10 feet and 27 msec at 100 feet. Estimates were based on previously published blast overpressure data (Baker, 1973; U.S. Army, 1990).
Figure 1 Estimated peak sound pressure levels (dB pSPL) for a range of distance from ground zero. Calculations were based on a 4000-lb TNT-equivalent charge representing 4800 lbs of ammonium nitrate. Side-on estimates assume a freefield detonation with no surface reflections and that the long axis of a subject's body is perpendicular to the blast wave. Reflected estimates are based on ground reflection. Shielding or reflections from surrounding structures were not included due to infinite possibilities. (Source: Wilfred Baker Engineering, Inc., San Antonio, TX)

Procedure

Subjects were seen quarterly (Q) at one of three collaborating clinics throughout 1 year: April to June 1995 (Q1), July to September (Q2), October to December (Q3), and April to June 1996 (Q4). The first visit fell within Q1 or Q2. Appointment reminder letters were mailed each quarter. Twenty-one subjects (25%) came for all four evaluations. The sample sizes (n) were as follows: Q1 = 42, Q2 = 64, Q3 = 62, and Q4 = 56. Nonexposed subjects were seen twice over a 6-month period.

The test battery included conventional pure-tone and EHF audiometry, otoscopic inspection, immittance and speech audiometry, and a questionnaire. Pediatric subjects were evaluated with the same battery using age- or state-appropriate methods. Nonexposed subjects were evaluated with conventional pure-tone audiometry, EHF audiometry, and tympanometry.

Audiometric testing was done in sound-treated booths with Teledynamics headphones (TDH-39P or TDH-59P) and commercial equipment conforming to ANSI (1991) standards. Conventional frequencies (CFs) included 0.25, 0.5, 1, 2, 3, 4, 6, and 8 kHz with bone-conduction testing at 0.25, 0.5, 1, 2, and 4 kHz. EHF thresholds were obtained with Koss Pro 4K or HD 250 Linear circumaural headphones and commercial equipment calibrated in sound pressure level. Frequencies included 10, 11.2, 12.5, 14, 16, 18, and 20 kHz.

Since preblast audiometric data were unknown, individual thresholds were adjusted with gender-specific age-correction factors developed by the National Institute of Occupational Safety and Health (NIOSH) for 1, 2, 3, 4, and 6 kHz (OSHA, 1993). Because age-corrected thresholds were not in 5-dB increments, "normal" threshold was defined as <22.5 dB HL and "abnormal" threshold defined as ≥22.5 dB HL.

Individual acoustic trauma severity was categorized according to a method proposed by Man et al (1981). This procedure divides the audiogram into segments and grades severity by the number of divisions encompassed by the audiometric profile. Grade I includes area A, Grade II A + B or A + C, Grade III A + B + C, and Grade IV A + B + D or A + B + C + D in Figure 2.

To reflect the high-frequency nature of noise-induced hearing loss, a 1-, 2-, 4-kHz pure-tone average (PTA_{124}) was adopted. However, nonorganicity was defined as poor (>10 dB) agreement between conventional, nonage-corrected PTA (0.5, 1, 2 kHz or two best frequencies) and speech reception threshold (SRT), and/or a positive speech Stenger.

Otoscopic inspection was done to note the appearance of the ear canal and tympanic membrane. Immittance audiometry included tympanometry; equivalent volume measures to quantify patency to the middle ear; 0.5-, 1-, and 2-kHz ipsilateral and contralateral acoustic reflexes; and contralateral acoustic reflex decay at 0.5 and 1 kHz.

Figure 2 Conventional-frequencies audiogram with categorical divisions proposed by Man et al (1981) for acoustic trauma severity classification. See text for full description.
Word recognition was performed at 40 dB SL and 95 dB HL to calculate performance-intensity function (PI-PB) rollover. To evaluate the appropriateness of word recognition ability with the degree of hearing loss, results were compared to 95 percent confidence limits for maximum word recognition scores established by Dubno et al (1995). With this method, one can parametrically state whether the maximum word recognition score (PB_{max}) is "disproportionately" poor relative to the degree of hearing loss.

A 15-item, written, closed-set questionnaire (Appendix) was completed during the first visit, addressing location at the time of the blast, initial and persistent symptoms, previous ear/hearing history, previous noise exposure, previous and current use of medications, and family history of hearing loss. During subsequent visits, subjects answered a written, closed-set questionnaire regarding persistence of symptoms (Question 4 of Appendix).

For all tests, "no response" was recorded as 5 dB higher than output limits of the equipment. While, in some cases, this may have created artifactual level effects, it was deemed more appropriate than entering "missing" data points, since they were, in fact, tested. Data were analyzed with descriptive and inferential statistics that included analysis of variance (ANOVA) and mixed linear models, using Fisher's least significant difference (LSD) procedure for post hoc testing. Since there was a positive correlation between ears for CF and EHF thresholds, individual right and left ear data were averaged together for hypothesis testing. A priori probability of significance was p < .05.

RESULTS

Pure-Tone Audiometry (CF and EHF)

Test–Retest Variability

PTA_{40} did not differ significantly between Q1, 2, 3, or 4, nor were there quarter differences by gender (n = 83). Nonexposed subjects had no significant PTA_{40} differences between tests 1 and 2 as a group or by gender (n = 10).

EHF thresholds were not significantly different between quarters as a group or by gender for subjects with no missing data (n = 7). Results were identical for those with complete data sets for Q2, 3, and 4 (n = 31). Nonexposed subjects also showed no changes between test sessions as a group or by gender (n = 10).

Due to lack of change over the year, some descriptive statistics were averaged across quarters to provide an overall summary of the event. Further inferential statistics were performed on Q4 data only (n = 56 subjects) to delineate results at 1-year postblast. For nonexposed subjects, test session 2 data were used (n = 9).

Although group data did not differ significantly between quarters, there were individual cases of air-conduction threshold shifts (±15 dB). Improvement ranged from 15 to 41 dB for any given frequency/quarter for 32 subjects (39%). Worsening ranged from 15 to 40 dB for any given frequency/quarter for 34 subjects (41%).

Audiometric Profiles

Table 1 displays audiometric profiles for blast subjects at their first visit (Q1 or Q2) with gender, laterality, and case history breakdowns. Averaged across quarters, three-quarters (76%) of subjects had abnormal hearing sensitivity at one or more frequency(s). Sixty-three percent were male. Bilateral involvement (74%) and sensorineural deficits predominated (97% of ears). One ear was conductive and four were mixed. Three ears with mixed loss had preexisting conditions, and the fourth was medically diagnosed with a possible ossicular fracture. For PTA_{40} data averaged across quarters, 21 percent of subjects were abnormal for one or both ears. Again, the majority of these were male (85%). Unilateral and bilateral involvement were fairly equally distributed (58% and 42%, respectively).

Most CF audiogram configurations were sloping toward high frequencies. Figure 3 depicts the mean Q4 audiogram for nonexposed subjects, blast subjects with bilaterally normal thresholds ("normal subjects"), and blast subjects with one or more abnormal threshold(s) ("abnormal subjects"). Using the Man et al (1981) classification system on Q4 data, 46 ears (41%) were normal, 28 ears (25%) had Grade I hearing loss, 19 (17%) had Grade II, 10 (9%) had Grade III, and 9 (8%) had Grade IV hearing loss.

For EHF, most subjects demonstrated the expected increase in threshold with increasing frequency. Figure 4 depicts the mean Q4 EHF audiogram for nonexposed subjects, normal subjects, and abnormal subjects. Blast subjects with abnormal CF thresholds had significantly poorer thresholds at 10 and 11.2 kHz than blast subjects with normal CF thresholds (covariate age; n = 65). Both blast groups had significantly poorer thresholds than the nonexposed group, except at 10 and 11.2, where the normal blast group and nonexposed group were similar.
Table 1  Audiometric Profile Tallies and Percentages for Bombing Subjects at their First Visit (Q1 or Q2)

<table>
<thead>
<tr>
<th>Individual Thresholds (0.25-8 kHz; n = 83)</th>
<th>Pure-Tone Average (1, 2, 4 kHz; n = 83)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Gender</td>
<td>24</td>
</tr>
<tr>
<td>Male</td>
<td>11</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
</tr>
<tr>
<td>Side</td>
<td></td>
</tr>
<tr>
<td>Bilateral</td>
<td>24</td>
</tr>
<tr>
<td>Unilateral</td>
<td>0</td>
</tr>
<tr>
<td>Previous hearing disorder</td>
<td>2</td>
</tr>
<tr>
<td>Previous tinnitus</td>
<td>3</td>
</tr>
<tr>
<td>Previous noise exposure</td>
<td>7</td>
</tr>
</tbody>
</table>

Subcategory percentages (i.e., gender, side, prior history of hearing disorders, tinnitus, and noise exposure) are derived from sample sizes in "normal" and "abnormal" categories. Data are age corrected. Normal was dB HL < 22.5 bilaterally; abnormal was dB HL ≥ 22.5 for one or both ears.

Gender Differences

In the CF range, male abnormal subjects had poorer thresholds than female abnormal subjects at 2, 3, 4, 6, and 8 kHz. There were no significant gender differences for normal subjects or nonexposed subjects (n = 65). For PTA134, there were no gender effects (n = 65). Results for blast subjects were unchanged with noise exposure and hearing loss history covariates (n = 56).

In the EHF range, there were no gender effects for nonexposed subjects, but blast-exposed

![Figure 3](image-url)  
**Figure 3** Mean age-corrected conventional frequencies thresholds (dB HL) for Quarter 4 (n = 65 subjects). Circles represent nonblast-exposed subjects (n = 9). Squares represent blast-exposed subjects with thresholds < 22.5 dB HL bilaterally (n = 12). Diamonds represent blast-exposed subjects with one or more thresholds ≥ 22.5 dB HL (n = 44).

![Figure 4](image-url)  
**Figure 4** Mean extended high frequencies thresholds (dB SPL) for Quarter 4 (n = 65 subjects). Circles represent nonblast-exposed subjects (n = 9). Squares represent blast-exposed subjects with age-corrected conventional frequencies thresholds < 22.5 dB HL bilaterally (n = 12). Diamonds represent blast-exposed subjects with one or more age-corrected conventional frequencies thresholds ≥ 22.5 dB HL (n = 44).
males were significantly worse than blast-exposed females (covariate age; n = 65) (Fig. 6). For normal CF subjects, males had poorer thresholds than females at 12.5, 14, 16, 18, and 20 kHz. For abnormal CF subjects, males were worse at 10, 11.2, 12.5, 14, and 20 kHz. Results for blast subjects were unchanged with noise exposure and hearing loss history covariates (n = 56).

**Case History**

At their first visit, 71 percent (n = 59) of subjects had one or more abnormal CF threshold(s) and 36 percent of these (n = 21) reported a history of hearing disorders (see Table 1). Therefore, a conservative estimate of the occurrence of hearing loss related to the bombing was 46 percent. However, there were no significant differences in Q4 CF or EHF thresholds between subjects reporting a positive history of hearing disorders and those with a negative history. There also were no effects of previous noise exposure on CF or EHF thresholds (covariate age; n = 56).

**Otoscopic Inspection and Immittance Audiometry**

Two subjects had unilateral and one had bilateral tympanic membrane perforations. All but one healed by Q2. Two subjects were located in the Federal Building and the other was inside a building about 0.3 mile east.

Impittance results clinically supported individual audiometric findings of normal auditory sensitivity; conductive, mixed, or sensorineural hearing loss; and quantified patency between the ear canal and middle ear. There were no individual data that related to the blast in an atypical manner; therefore, group analyses were not undertaken.

**Speech Audiometry**

Three of 86 subjects were eliminated from the database due to poor SRT/PTA agreement. This is a 3 percent occurrence rate for nonorganincity.

Word recognition scores at 40 dB SL varied from 20 percent to 100 percent. Averaged across quarters, 80 percent of ears had scores of 90 percent or better, and 12 percent of ears had maximum word recognition scores that fell outside of 95 percent confidence limits (Dubno et al, 1995). Five ears that fell outside of confidence limits had PI-PB rollover of 25 percent or greater (Bess et al, 1979). Across quarters, 4 percent of ears had positive PI-PB rollover.
preblast tinnitus. Other immediate symptoms included 55 subjects (67%) with distorted hearing (95 ears), 34 (41%) with loudness sensitivity (57 ears), and 36 (44%) with otalgia (53 ears). Thirty-six subjects (44%) reported dizziness/vertigo, which was addressed in a separate study. Nine subjects (11%) had no immediate auditory symptoms.

By their first visit (Q1 or Q2), 48 people (59%) still noted tinnitus. Of those with preblast tinnitus, 8 described changes in the loudness, frequency of occurrence, or quality of their tinnitus after the explosion. One subject noted unilateral tinnitus before and bilateral tinnitus after the event. Other first-visit symptoms included 57 percent of subjects with distorted hearing, 38 percent with loudness sensitivity, and 32 percent with otalgia. Only one person reported a symptom (distorted hearing) during their first visit that was not noted within seconds to days of the blast. Eleven subjects (13%) had no auditory complaints at their first visit. Later reports only were considered valid if the symptom was noted by the first visit.

At 1 year, 76 percent of subjects still were reporting tinnitus, 58 percent distorted hearing, 64 percent loudness sensitivity, and 57 percent otalgia. Averaging across quarters to summarize the year, 68 percent of subjects reported postblast tinnitus, 60 percent had distorted hearing, 60 percent had loudness sensitivity, and 45 percent had otalgia.

Due to inconsistent attendance records and variable compliance with questionnaires, patterns of symptom recovery were evaluated best for subjects who answered all four questionnaires (n = 9). Data (by ear) are displayed graphically in Figure 7 and were not adjusted for case history or reporting consistency. This subgroup showed the same pattern of immediate symptoms as the entire study sample. Subsequently, symptoms showed a decline from initial reports during Q1 or 2, and then stayed fairly constant throughout the remaining year.

Location

Sixty-seven subjects were located in a building during the explosion. Twenty-five
Table 2  Tallies and Percentages of Acoustic Trauma Classifications Referenced to Location at the Time of the Blast

<table>
<thead>
<tr>
<th>Location at Time of Blast</th>
<th>Acoustic Trauma Classifications (Man et al, 1981)</th>
<th>First Visit (Q1 or Q2)</th>
<th>Last Visit (Q4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Grade I</td>
<td>Grade II</td>
</tr>
<tr>
<td>Federal Building</td>
<td>25 30</td>
<td>10 40</td>
<td>8 32</td>
</tr>
<tr>
<td>Buildings within one block (0.10 mile) of Federal Building</td>
<td>31 37</td>
<td>11 35</td>
<td>12 39</td>
</tr>
<tr>
<td>Other buildings in vicinity</td>
<td>11 13</td>
<td>3 27</td>
<td>2 18</td>
</tr>
<tr>
<td>Outside pedestrian</td>
<td>7 9</td>
<td>2 29</td>
<td>3 43</td>
</tr>
<tr>
<td>Inside of vehicle</td>
<td>9 11</td>
<td>4 44</td>
<td>1 11</td>
</tr>
<tr>
<td>Total</td>
<td>83 100</td>
<td>30 36</td>
<td>26 31</td>
</tr>
</tbody>
</table>

Classifications were based on first visit (Q1 or Q2) and last visit (Q4) age-corrected thresholds for the subject’s worst ear. See text for explanations of acoustic trauma classification system. “Normal” as defined by this method is dB HL ≤ 25.

individuals were from the Federal Building. Seven subjects were outside and nine were in vehicles.

There was no clear relationship between location and symptoms or test results. Of subjects with no immediate symptoms, 44 percent were from the Federal Building. The remaining were in two buildings across the street to the north. Of subjects with no first-visit (Q1 or Q2) symptoms, 45 percent were from the Federal Building. The remaining were either in two buildings across the street or in cars at close-by intersections. Table 2 shows the dispersion of acoustic trauma severity grades by location for first-visit and Q4 data. In general, hearing loss predominated in eastern to northeastern locations from ground zero (Fig. 8), which corresponded to areas most damaged structurally. Ten of 25 subjects from the Federal Building had bilaterally normal (re: Man et al, 1981) age-corrected CF thresholds at their first visit. Eight had Grade I acoustic trauma in their worst ear. Nine of these 18 subjects were on the first or second floor and directly in line with the explosion. The furthest distance from the blast that resulted in hearing loss was a pedestrian approximately 0.5 mile north.

Amplification

Amplification was recommended for 20 (24%) subjects. None of these individuals reported preblast use of hearing aids or perceived need for such. Seventeen subjects were fitted. Eleven fittings were binaural and six were monaural.

DISCUSSION

Human exposure to unplanned explosions presents many assessment, treatment, and interpretative challenges for clinician and scientist. Blast trauma is determined by myriad conditions such as device and explosion specifications, subject distance from and positioning to the blast wave, and the physical environment (i.e. outdoors or indoors, structural characteristics of building or vehicle, proximity to reflective surfaces, windows, doors, and hallways). For the ear, individual noise susceptibility and auditory history complicate these factors. Nevertheless, if the present data typify injuries subsequent to a large-magnitude explosion, then the delineated patterns should provide a valuable
Figure 8  Aerial view of geographic area that sustained 90% of building damage and where 83 subjects were located during the explosion. The large white outline is the city block of the Federal Building complex; the small white outline is the structure itself. The blue truck represents the truck bomb. Person icons designate pedestrians, bullseye designates subjects inside a vehicle, and houses designate subjects inside a building. House icons may represent more than one subject. Yellow denotes bilaterally normal (<22.5 dB HL) age-corrected pure-tone thresholds at the first evaluation. Red denotes at least one abnormal ear (≥22.5 dB HL age-corrected threshold[s]) at the first evaluation. Orange denotes locations with both normal and abnormal auditory findings.
knowledge base for similar future events. In light of renewed awareness that blast trauma is not limited to military or industrial settings, professional preparedness is prudent, if not necessary.

Since preblast audiometric data were unavoidably absent, several data management methods were employed to facilitate meaningful interpretations: (1) inferential statistical methods were used to determine significance; (2) CF pure-tone data were age corrected, and EHF were analyzed with age as a covariate; (3) test–retest reliability and gender differences were explored with nonblast-exposed subjects; (4) subjects were queried with closed-set questionnaires regarding symptomatology and auditory history; (5) histories of hearing disorder, noise exposure, or tinnitus were considered both inferentially and/or descriptively; and (6) the occurrence of hearing loss and tinnitus will be discussed in relation to national census figures. None of these methods has been employed in former civilian blast studies. Findings will be discussed in relation to previous blast data; however, comparisons between reports are limited due to disparate explosion characteristics, sample sizes, terminology, and subject recruitment and assessment methodology.

In order of reporting magnitude, symptoms presenting within seconds to days of the blast included tinnitus, distorted hearing, loudness sensitivity, otalgia, and dizziness/vertigo. These were consistent with other single-explosion papers reporting immediate symptoms, except that our sample additionally noted loudness sensitivity (Kerr and Byrne, 1975; Bruins and Cawood, 1991; Chandler and Edmond, 1997). The occurrence of immediate symptoms varies by report. Only Chandler and Edmond (1997) discussed immediate tinnitus, which was present in 100 percent of five subjects, compared to an occurrence rate of 67 percent of subjects for the present sample. Previous reports of immediate hearing disturbance range from 90 percent to 100 percent, compared to our 67 percent (Kerr and Byrne, 1975; Bruins and Cawood, 1991; Chandler and Edmond, 1997). Only Chandler and Edmond (1997) reported immediate otalgia (100%) and vertigo (100%). Again, our figures are lower with both symptoms noted in 44 percent of subjects.

There were no apparent relationships between subject location and immediate or persistent symptoms or test results. In general, the dispersion of hearing loss corresponded with areas that sustained the most structural damage. Subjects located inside the Federal Building fared better than expected. Sixteen percent had no immediate auditory symptoms and 20 percent had no first-visit symptoms. Seventy-two percent had either bilaterally normal results or Grade I acoustic trauma in the poorer ear during the first visit. These findings may be explained by the fact that the structure itself absorbed a significant amount of blast energy, which, in effect, shielded the occupants.

This study did not demonstrate notable auditory recovery that reportedly may occur within 6 months following blast exposure. Korkis (1952) and Bruins and Cawood (1991) indicated that sensorineural deficits may improve within this period, and Kerr (1980), Bruins and Cawood (1991), and Yetiser and Ustun (1993) reported that tinnitus diminishes or becomes intermittent with time or as hearing improves. As demonstrated with the subgroup that answered all four questionnaires, symptoms improved slightly within 6 months but, overall, auditory function was compromised throughout 1 year postblast.

Group data are more supportive of Segal et al.'s (1988) findings that thresholds are stable by 1-year postacute acoustic trauma, provided that there is no further exposure to loud noise. In our sample, there were no statistically significant changes in CF or EHF pure-tone thresholds during the year. On the average, three-quarters of subjects (76%) had abnormal hearing sensitivity at one or more frequencies and 21 percent had abnormal averaged thresholds in speech frequencies (PTA₁₂). Additionally, perception of distorted hearing did not change during the year, so that, on average, 60 percent of subjects reported the symptom, and at 1-year postblast, it persisted for 58 percent of subjects. For tinnitus, 68 percent of subjects reported the symptom on the average and 76 percent had it at 1 year.

Cause and effect are impossible to determine without preblast information; however, tinnitus figures represent subjects reporting no preblast tinnitus or those with a postblast perceptual change, and first-visit hearing loss figures were adjusted to exclude those with a history of hearing disorders. For the latter, 46 percent of subjects presumably incurred new hearing loss from the blast. Lending credence to the blast's impact are the strikingly lower reported figures for tinnitus and hearing impairment from the general U.S. population, which were 2.7 percent and 8 percent, respectively, in 1994 (U.S. Bureau of the Census, 1996).
Individual cases without symptoms or documented abnormalities and cases with clinically relevant threshold shifts reflect complexities of blast exposure, as well as individual susceptibility to acute acoustic trauma. The latter also reinforces the need for serial testing. Physiologically, acoustic overstimulation threshold changes are related to excitotoxic and plastic mechanisms and controlled degeneration within organ of Corti and primary auditory neurons (Forge, 1996; Puel et al, 1996). After a period of initial repair by supporting cells, the mammalian epithelium progressively "dedifferentiates," such that neural elements degenerate and are replaced by nonspecialized, squamous-like epithelial cells over a prolonged period of time. Based on this, it seems plausible that ears with widespread injury to organ of Corti might evidence instability and worsening of thresholds, while those with minor damage remain stable or improve.

Another explanation for threshold degradation could be "post-traumatic endolymphatic hydrops," which has been documented in guinea pig, chinchilla, and human acoustic trauma (Clark and Rees, 1977; Lim et al, 1982; Paparella and Mancini, 1983; Ylikoski, 1988; Kumagami, 1992). In humans, threshold fluctuations can begin months to years after exposure and may be due to traumatic damage to cells that produce or absorb endolymph or from cellular debris that chemically or mechanically disrupt endolymphatic absorption (Paparella and Mancini, 1983).

Loudness sensitivity and otalgia have been explored less well than hearing loss and tinnitus. Postblast loudness sensitivity (i.e., recruitment or hyperacusis) has been described as notable by its absence (Singh and Ahluwalia, 1968; Teter et al, 1970). In contrast in the present study, it was reportable during the year for an average 60 percent of subjects and for 57 percent of subjects at 1 year. One explanation for the disparity is that reporting can be confounded by difficulty differentiating between recruitment and a sensitive startle response, often associated with post-traumatic stress. In the future, this issue would be better addressed with loudness growth functions. Otalgia has not been followed for longer than 3 weeks postblast in previous studies (Singh et al, 1983; Shupak et al, 1993; Chandler and Edmond, 1997), but for that period occurrences ranged from 0 percent to 20 percent. Our reports were markedly higher, with 45 percent of subjects experiencing the symptom on the average and 57 percent with otalgia at 1 year.

Attempts to understand injuries must be illuminated with comprehension of the characteristics of an explosion. Blast waves are a unique category of hazardous noise due to their spectra and intense pressure. A blast frequency spectrum contains high levels of infrasound (i.e., frequencies < 20 Hz) (Leventhall and Kyriakides, 1976). While this frequency range is not perceived by man, the body absorbs the energy and organ dysfunction can result. Manifestations of high-intensity (≥ 145 dB SPL) infrasound on human audition are not well defined (von Gierke and Nixon, 1976), but severe damage to chinchilla middle and inner ears has been documented (Lim et al, 1982).

The pressure wave of a blast probably is responsible for initial damage to the labyrinth since it arrives prior to the associated sound wave. Collins (1948) observed that the likelihood of inner ear damage increases with increasing blast pressure. The literature also indicates that higher pressures increase the chances of tympanic membrane perforations, such that 50 percent of eardrums will rupture at 15 psi (Hirsch, 1968). Based upon Oklahoma City overpressure estimates ranging from 3000 psi at 10 feet to 0.7 psi at 1000 feet, and upon previous single-explosion perforation rates of 26.9 percent to 100 percent (McReynolds et al, 1949; Kerr and Byrne, 1975; Shupak et al, 1993; Chandler and Edmond, 1997), high numbers of perforations were expected. In contrast, however, our sample had a 3.6 percent occurrence. Initially, sampling error might be suspected, but this figure is corroborated by Malonee et al's (1996) epidemiologic account of the Oklahoma City bombing, which stated that 22 of 592 (3.7%) survivors had reported or documented perforations.

The Oklahoma City blast overpressure duration combined with its pressure and shielding by buildings or vehicles (Yelverton et al., 1996) may explain the low number of tympanic membrane ruptures, as well as the relatively mild manifestation of cochlear damage. Only 24 percent of subjects warranted intervention with amplification and only 23 percent of subjects had acoustic trauma severity grades of III or IV at 1-year postblast. This is lower than Man et al's (1981) report that 55 percent of explosion-exposed subjects had Grade III or IV acoustic trauma. In fact, the present study's Grade IV figures likely are overestimated due to a shortcoming in the grading system. With this method,
a mild low-frequency impairment does not have its own classification. When thresholds fell within audiogram division "D," the deficit defaulted into Grade IV trauma.

The severity of CF acoustic trauma extended into the EHF range in a predictable manner; with the more severe CF deficits having worse EHF thresholds. The exception was Grade IV, which is attributed to the limitations of Man et al's (1981) classification system. Although there are no previous EHF blast data, Borchgrevink et al (1996) obtained similar results with subjects exposed to steady-state and impulse noise.

Due to time constraints after the blast, this study did not use age-matched control groups for EHF evaluation, as recommended by Osterhammel (1980), Stelmachowicz et al (1989), and Buren et al (1992). Nevertheless, it seemed valid to compare blast-exposed subjects with young, nonexposed subjects since CF normalcy is derived from young, healthy ears (re: 0 dB HL). Additionally, age was taken into consideration as a covariate for hypothesis testing. Occupational noise data indicate that EHF threshold elevation is present for all grades of CF acoustic trauma and all age groups, relative to age-matched controls (Hallmo et al, 1995). The fact that normal (CF) blast subjects had significantly poorer EHF thresholds than nonexposed subjects implies that blast overpressure can inflict discrete damage to the cochlear partition. In some instances, basal regions apparently were injured while mid-range and apical areas were spared. Studies of steady-state and impulse noise exposure indicate that EHF threshold shifts occur prior to CF threshold degradation (Fausti et al, 1981; Dieroff, 1982). Fausti et al (1981) assert that it is impossible to describe accurately the effects of noise on cochlear function without EHF threshold mapping.

The effect of EHF pathology on auditory processing can only be postulated since little is known about functional use of the 9- to 20-kHz frequency range in humans. This study's subjective reports indicate that perceptual changes are noted with sudden loss of sensitivity in this region. A number of subjects with vague complaints of distorted hearing and normal CF thresholds had abnormal EHF thresholds. This may explain the frequent discrepancy between normal audiograms and "subjective deafness" reported by Bruins and Cawood (1991). Routine clinical evaluations of speech perception (e.g., word recognition lists) likely are too rudimentary to assess discrete changes in auditory processing; however, 12 percent of ears did have disproportionately low word recognition scores relative to CF thresholds (re: Dubno et al, 1995) and 4 percent of ears had positive PI-PB rollover.

Significant gender differences for EHF and CF thresholds revisit an old issue of male susceptibility to noise damage. Older data from industrial studies support male susceptibility to auditory noise damage (Gallo and Glorig, 1964; Berger et al, 1978), while more recent papers question its significance (Henderson et al, 1993; Ward, 1995). Although previous blast papers do not report on gender, several factors suggest that males are more sensitive to auditory blast injury than females. First, the majority (61%) of those with first-visit hearing loss was male. Second, there were no gender differences for nonexposed subjects for either frequency range, while there was a significant CF gender effect for abnormal blast subjects and significant EHF gender effects for all blast subjects. Third, males and females were equivalently dispersed around the explosion.

Matthews et al (1997) explained significant EHF male-female differences by relating results to CF PTAs, where males performed more poorly than females. We cannot use this explanation since EHF gender differences were present in subjects with normal CF thresholds. Another possibility is that males had preexisting EHF loss since most subjects with a history of hearing disorders or noise exposure were male (74% and 81%, respectively). However, significant gender effects persisted with case histories as covariates. Additionally, there were no significant differences in CF or EHF thresholds for subjects (mixed gender) with positive or negative histories for hearing disorders or noise exposure.

CONCLUSION

The Oklahoma City experience highlights the important role of audiologic management in the aftermath of blast injury. In this sample of survivors, auditory deficits presented a need for both immediate assessment and ongoing care. While previous reports emphasize medically manageable sequelae, such as tympanic membrane perforations and conductive hearing loss, this study demonstrated that aural blast injury is a multifaceted pathology resulting from exogenous and endogenous factors.

A test battery assessment was very useful to appropriately discern patient complaints and patterns of deficit. In particular, EHF testing showed evidence of acoustic trauma even when
CFs fell within normal limits. Based upon present data, it would be appropriate to counsel patients that recovery from tinnitus, sensorineural deficit, loudness sensitivity, and otalgia is limited after 6 months. Advance discussion about the possibility of assistive listening or tinnitus management devices can enhance later acceptance. Patients should be followed for 1 year to document change across time and to provide supportive rehabilitation.

Although aural blast trauma is not life threatening, it can be life changing and disconcerting. Until there are definitive medical therapies that promote healing of inner ear structures, professionals need to understand the nature of the injury in order to provide state-of-the-art counseling and intervention. Renewed awareness of terrorist activity should prompt professional commitment to preparedness. Research endeavors such as assessing discrete changes in auditory processing and biochemical and molecular mechanisms of blast-overpressure-induced injury will enhance comprehension of functional and physiologic pathology.

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REFERENCES


APPENDIX

Questionnaire

1. Were you injured and given medical treatment due to the Oklahoma City Federal Building explosion on April 19, 1995?
   No
   Yes

2. Were you at work when the explosion occurred?
   No
   Yes
3. We need to know where you were when the explosion took place. Please check one.
3A. _____ Inside a building
   Name of building/business ____________________________
   Street address ____________________________
   Floor of building ____________________________

   Were you in a room with windows?
   _____ No
   _____ Yes

   Did the windows in your room break?
   _____ No
   _____ Yes

   Was the building damaged?
   _____ No
   _____ Yes
   _____ Outside

   Specify location as best as you can: ____________________________

   _____ Inside a vehicle
   Specify location as best as you can: ____________________________

   Please check where you were seated in the vehicle:
   ____________________________
   No   Yes
   A  Driver’s side
   B  Front passenger
   C  Back left passenger
   D  Back right passenger

   Were the windows open?
   _____ No
   _____ Yes  If yes, please specify which one(s) ____________________________

   Did any of the windows break?
   _____ No
   _____ Yes  If yes, please specify which one(s) ____________________________

3B. Do you remember which direction you were facing during the explosion?
   _____ No
   _____ Yes  If yes, check the direction:
   A  North
   B  South
   C  East
   D  West
   E  Northeast
   F  Northwest
   G  Southeast
   H  Southwest

4. This question has three parts:
   Part 1  What were your initial symptoms after the explosion?
   Part 2  How soon after the explosion did you first notice the symptoms?
   Part 3  How would you describe your symptoms now as compared to when you first noticed them?
### Part 1

<table>
<thead>
<tr>
<th>Check all that apply</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Noise (or ringing) in right ear</td>
</tr>
<tr>
<td></td>
<td>Noise (or ringing) in left ear</td>
</tr>
<tr>
<td></td>
<td>Muffled or distorted hearing in right ear</td>
</tr>
<tr>
<td></td>
<td>Muffled or distorted hearing in left ear</td>
</tr>
<tr>
<td></td>
<td>Pain in or around right ear</td>
</tr>
<tr>
<td></td>
<td>Pain in or around left ear</td>
</tr>
<tr>
<td></td>
<td>Sensitivity to loud sounds in right ear</td>
</tr>
<tr>
<td></td>
<td>Sensitivity to loud sounds in left ear</td>
</tr>
<tr>
<td></td>
<td>Headache</td>
</tr>
<tr>
<td></td>
<td>Dizziness</td>
</tr>
<tr>
<td></td>
<td>Vomiting</td>
</tr>
<tr>
<td></td>
<td>Other</td>
</tr>
</tbody>
</table>

### Part 2

<table>
<thead>
<tr>
<th>Check only one for each symptom</th>
</tr>
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<tbody>
<tr>
<td>Already</td>
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</table>

### Part 3

<table>
<thead>
<tr>
<th>Check only one for each symptom</th>
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</thead>
<tbody>
<tr>
<td>Comes and</td>
</tr>
</tbody>
</table>

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5. **Before** the explosion,

5A. Did you have any previous ear or hearing disorders?

- [ ] No
- [ ] Yes If yes, please describe: ____________________________

5B. Did you have any previous dizziness or balance disorders?

- [ ] No
- [ ] Yes If yes, please describe: ____________________________

---

6. **Before** the explosion, did you ever have a previous hearing test?

- [ ] No
- [ ] Yes If yes, when was your last test? ____________________________

6A. Do you remember the results?

- [ ] No
- [ ] Yes If yes, what were they? ____________________________

6B. Are the results available?

- [ ] No
- [ ] Yes If yes, where? ____________________________

---

7. **Since** the explosion, have you had a hearing test?

- [ ] No
- [ ] Yes If yes, where? ____________________________

  When? ____________________________

7A. Do you remember the results?

- [ ] No
- [ ] Yes If yes, what were they? ____________________________

7B. Are the results available?

- [ ] No
- [ ] Yes If yes, where? ____________________________

---

8. **Before** the explosion, did you have any ringing or noises (tinnitus) in your ears?

- [ ] No
- [ ] Yes If yes, which ear(s)? _____ right _____ left _____ both

8A. Does the noise sound different **now** after the explosion?

- [ ] No
- [ ] Yes If yes, please describe: ____________________________
9. **Before** the explosion,
   9A. Did you have any work-related noise exposure?
      - No
      - Yes  If yes, for how long? ________ number of years
      What type of noise? ____________________________________________
      Did you wear ear protection? ______ sometimes
      ______ always
      ______ never
   9B. Did you have any recreational noise exposure?
      - No
      - Yes  If yes, for how long? ______ number of years
      What type of noise? ____________________________________________
      Did you wear ear protection? ______ sometimes
      ______ always
      ______ never

10. **During** the explosion, did you sustain a head injury?
    - No
    - Yes  If yes, check the injuries that apply:
      A  Skull fracture
      B  Concussion
      C  Bleeding from ears
      D  Cuts or lacerations to face or scalp
      E  Loss of consciousness

11. **Before** the explosion, did you ever have any head injuries?
    - No
    - Yes  If yes, check the injuries that apply:
      A  Skull fracture
      B  Concussion
      C  Bleeding from ears
      D  Cuts or lacerations to face or scalp
      E  Loss of consciousness

12. **Before** the explosion, did your physician ever tell you to take (check all that apply):
    - No
    - Yes  
      A  Strong antibiotics
      B  Diuretics
      C  Chemotherapy
      D  Large amounts of aspirin

13. Are you currently taking any medication?
    - No
    - Yes  If yes, please specify: ________________________________________

14. **Before** the explosion, how would you describe your health in general? (Check one)
    - Poor
    - Fair
    - Good
    - Excellent

15. Does your family have a history of hearing loss?
    - No
    - Yes  If yes, specify relative(s) _______________________________________
      Specify type of hearing disorder _______________________________________

Thank you for filling out this form. Your answers may be able to benefit others in the future.