One-Year Vestibular and Balance Outcomes of Oklahoma City Bombing Survivors

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Abstract

This multisite investigation assessed subjective, behavioral, and objective balance function in 30 blast survivors. Subjects with vertigo, dizziness, or imbalance were screened (n = 6) or evaluated (n = 27) during 1 year. Tests included a questionnaire, electronystagmography (ENG), and computerized dynamic posturography (CDP). Ninety-seven percent of subjects were located inside a building during the blast, and 63 percent of subjects experienced dysequilibrium within 48 hours. Forty-three percent of symptoms could not be attributed to head injury. Sixty percent of subjects had abnormal ENG and/or CDP; ENG abnormalities mostly were peripheral or nonlocalizing, whereas CDP patterns were “vestibular,” “surface dependent,” and “physiologically inconsistent.” At 1-year postblast, 55 percent of initially abnormal CDP results were normal, and 72 percent of subjects said symptoms were unchanged or occurred intermittently. A serial, test battery approach is recommended to assess symptoms. Blast-related dysequilibrium had clinically significant manifestations and should be considered a valid component of aural blast injury.

Key Words: Acoustic trauma, computerized dynamic posturography, dizziness, electronystagmography, imbalance, vertigo, vestibular blast injury

Abbreviations: BPPV = benign paroxysmal positioning vertigo, CDP = computerized dynamic posturography, dB pSPL = decibel re: peak sound pressure level, ENG = electronystagmography, MCT = motor control test, psi = air pressure measured in pounds per square inch, Q = quarterly evaluations, SHA = smooth harmonic acceleration, SOT = sensory organization test, TNT = trinitrotoluene

Despite evidence that the nonauditory labyrinth can be activated with sound and air pressure variations (see Parker, 1976; Britton, 1986; Hinchcliffe et al, 1992, for reviews), most aural blast injury papers focus on auditory sequelae. Consequently, human vestibular blast trauma has been inadequately described and evaluated. The literature that acknowledge vestibular symptoms discuss causation without formally reporting evaluation methodology. These papers attribute symptoms primarily to associated head injury (McReynolds et al, 1949; Kerr and Bryne, 1975; Kerr, 1978) but also to perilymph fistula (Singh and Ahluwalia, 1968), temporary endolymphatic hydrops (Korkis, 1952), “pressure-related damage” (Kerr, 1978, 1980), and psychological overlays (Pahor, 1981).

Only one study has conducted controlled vestibular evaluations following blast exposure. Shupak et al (1993) performed electronystagmography (ENG) and rotatory-chair smooth harmonic acceleration (SHA) tests on five Israeli patrol boat crewmen exposed to a close-range explosion. This study is significant since none of the men suffered head injury, yet three subjects had evidence of peripheral vestibulopathy within...
1 week of the blast. Follow-up was limited to 1-month postblast, but all three subjects were symptom free within 2 weeks of the first evaluation.

No study has examined postblast vestibular pathology with a test battery that includes age-appropriate functional balance, investigated change throughout a year, or systematically compiled associated signs and symptoms. The April 19, 1995 terrorist bombing of the Oklahoma City Federal Building provided an opportunity to formally evaluate and follow vestibular and balance outcomes of blast exposure in a large, varied sample. This explosion resulted from detonation of a 4800-pound ammonium nitrate truck bomb and was directly liable for 592 injuries and 167 deaths (Malonee et al, 1996). The purpose of the multisite longitudinal investigation was to describe subjective, behavioral, and objective vestibular and balance function in a sample of survivors to document patterns of deficit and recovery.

METHOD

Subjects

Subjects were recruited from a companion study evaluating audiologic outcomes of the Oklahoma City bombing (Van Campen et al, 1999). As part of this investigation, subjects were queried about vestibular symptoms and observed for abnormal gaze (i.e., disconjugate or nystagmus). Subjects were asked to participate if they had nonrecorded gaze abnormalities and/or one or more episodes of vertigo or ongoing imbalance. Subjects reporting only temporary imbalance without vertigo were given a more thorough screening (described in this section) to determine eligibility.

Thirty-two of 83 audiologic subjects (39%) with imbalance, vertigo, or abnormal gaze signed a written, informed consent to participate. Data from two subjects, however, were excluded due to observable, nonorganic behavior (described in the results section). At the time of the blast, all subjects were located within the downtown area that sustained 90% of the structural damage (roughly 0.9 mile east-west by 1.3 miles north-south). Ages ranged from 25 to 63 years (mean = 43 years). Fifty percent of subjects were female and 50 percent were male.

Stimulus

Individual sound and air pressure exposure levels were infeasible to calculate due to unknown exact 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 (Source: Wilfred Baker Engineering, Inc.). Incident blast overpressure ranged from 3000 pounds per square inch (psi) at 10 feet to 25 psi at 100 feet for a person perpendicular to the blast. Corresponding decibel levels would have been approximately 235 dB pSPL and 199 dB pSPL, respectively. Ground-reflected estimates are higher (Van Campen et al, 1999). Overpressure duration was approximately 2.8 msec at 10 feet and 27 msec at 100 feet. Calculations were based on published blast overpressure data (Baker, 1973; U.S. Department of the Army, 1990).

Procedure

Volunteers were instructed to terminate intake of tranquilizers, antihistamines, alcohol, and caffeine 48 hours prior to all testing. During their first visit, subjects completed a 21-item, closed-set, written questionnaire, addressing the time course, character, and associated signs and symptoms of the dizziness/imbalance, previous imbalance, head injury history, current use of medication and alcohol, and ear history (Appendix). During subsequent visits, subjects were queried regarding persistence of symptoms.

Screening protocol included nonrecorded gaze testing, the Dix-Hallpike maneuver, and all conditions of the sensory organization test (SOT) and motor control test (MCT) for computerized dynamic posturography (CDP). (See Tables 1 and 2 for Dix-Hallpike protocol and criteria for abnormality.) Posturography was done with a Neurocom EquiTest system. Testing was performed without shoes, with feet at a comfortable stance and arms at the side. The subject was secured in a harness, and sensory and motor control test conditions were described before a trial was initiated.

Subjects who passed all screening components did not receive further evaluation. A full balance assessment was scheduled if there were gaze abnormalities, if any part of the CDP was abnormal for a subject's age, or there was recurring imbalance. If a subject had a positive Dix-Hallpike, a canalith repositioning maneuver
Table 1  Electronystagmography Protocol

<table>
<thead>
<tr>
<th>Subtest</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonrecorded gaze</td>
<td>30° horizontal, vertical, and off-axis gaze for 20–30 seconds</td>
</tr>
<tr>
<td>Recorded gaze</td>
<td>Eyes open at 20° horizontal and vertical gaze for 20–30 seconds</td>
</tr>
<tr>
<td>Fistula testing</td>
<td>Eyes closed, +400 mmH₂O of pressure for 15 seconds, then −400 mmH₂O of pressure for 15 seconds. Patient was queried regarding sensation of vertigo.</td>
</tr>
<tr>
<td>Random horizontal saccades</td>
<td>DIX-Hallpike maneuver</td>
</tr>
<tr>
<td>Varying-frequency horizontal smooth</td>
<td>Supine, head-hanging position with eyes open for at least 30 seconds.</td>
</tr>
<tr>
<td>pursuit</td>
<td>Thirty-second waiting period before repeating the maneuver for the same ear or between ears.</td>
</tr>
<tr>
<td>Positional tests</td>
<td>Eyes closed and mental tasking for at least 30 seconds in sitting, supine, head right/left, and body right/left positions</td>
</tr>
<tr>
<td>Bithermal caloric irrigations</td>
<td>Head and shoulders elevated 30°, water temperatures of 30° and 44° C. Each ear stimulated with 250 cc for 30 seconds. Recording done with eyes closed and mental tasking for approximately 1 minute and 15 seconds following irrigation cessation. Fixation suppression checked at that time. Order of irrigation was right-then-left warm, followed by left-then-right cool. Five-minute waiting period between ears, and 10 minutes between temperatures.</td>
</tr>
</tbody>
</table>

was scheduled. The technique used for repositioning (e.g., Epley vs Semont) was left to the discretion of each participating clinic. If a subject had two unsuccessful repositioning maneuvers, a full assessment was scheduled.

Subjects meeting criteria for full balance assessment (n = 27) were tested quarterly (Q) by an audiologist at one of two collaborating clinics throughout 1 year: April to June 1995 (Q1), July to September (Q2), October to December 1995 (Q3), and April to June 1996 (Q4). Appointment reminder letters were mailed each quarter; however, return attendance was variable, as was participation in both parts of the test battery. Five subjects came for all four evaluations. Sample size (n) collapsed across both tests was Q1 = 9, Q2 = 18, Q3 = 22, and Q4 = 24.

The first balance assessment included immittance audiometry, ENG, and CDP. Immittance audiometry included tympanometry and calculation of equivalent volumes to document intact tympanic membranes. ENG was performed with commercial computerized equipment (ICS Chart or Nicolet Nystar) and was composed of subtests delineated in Table 1. All ENG recordings were made with bitemporal surface electrodes and one vertical electrode pair. If CDP had been done for screening, it was

Table 2  Electronystagmography Criteria for Abnormality

<table>
<thead>
<tr>
<th>Subtest</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonrecorded gaze</td>
<td>Continuous, observable beats in a given direction of gaze</td>
</tr>
<tr>
<td>Recorded gaze</td>
<td>Recordable beats (&gt;6° per second) in a given direction of gaze</td>
</tr>
<tr>
<td>Fistula testing</td>
<td>Recordable beats and/or report of vertigo</td>
</tr>
<tr>
<td>Random horizontal saccades</td>
<td>Velocity, accuracy, or latency outside the range of age-matched normative data provided with system software</td>
</tr>
<tr>
<td>Varying-frequency horizontal smooth</td>
<td>Velocity gain outside the range of age-matched normative data provided with system software</td>
</tr>
<tr>
<td>pursuit</td>
<td>Observable, latent, fatigueable, rotary nystagmus with subjective vertigo</td>
</tr>
<tr>
<td>Dix-Hallpike maneuver</td>
<td>Recordable beats, present as long as the subject remained in a given static position with eyes closed</td>
</tr>
<tr>
<td>Positional tests</td>
<td>Caloric weakness was an intereard difference of 25% or greater. Directional preponderence was an intereard difference of 30% or greater. Failure of fixation suppression was failure to reduce caloric nystagmic velocity by at least 40% with eyes opened and gaze fixated 18° away.</td>
</tr>
<tr>
<td>Bithermal caloric irrigations</td>
<td></td>
</tr>
</tbody>
</table>
not repeated. Otherwise, all conditions of CDP were completed.

Follow-up balance assessments included all conditions of CDP, nonrecorded gaze testing, recorded gaze testing, Dix-Hallpike maneuver, and recorded positional testing. Oculomotor tests were repeated if they were abnormal previously for the patient's age. Caloric irrigations were not repeated since ear weaknesses typically do not improve, even with resolution of signs and symptoms.

Data were analyzed qualitatively for patterns and quantitatively with descriptive statistics. ENG abnormality criteria are defined in Table 2. Decisions regarding normalcy for CDP sensory and motor conditions were based upon age-matched normative data provided with system software.

Whenever SOT was borderline abnormal, had nontraditional patterns (Cyr, 1991; Nashner, 1993b), or had inconsistencies within conditions, data were subjected to a three-tiered assessment. Analysis was done by the first author, who was knowledgeable about the subjects' history, symptoms, and ENG results; an expert judge blinded to these factors; and a mathematical equation (stepwise linear discriminant analysis) proposed by Cevette et al (1995) for classification of SOT data as "normal," "vestibular," or "nonphysiologic." This is reported to have an accuracy of 95.5% (Cevette et al, 1995). Both judges had access to and used motor control data and raw data for decision-making purposes. Final SOT pattern decisions were based on agreement of two of the three methods.

For the judges, one or more of the following defined "physiologically inconsistent" or "nonphysiologic" results (Cyr, 1991; Nashner, 1993b; Cevette et al, 1995; Hamid, 1996): (1) abnormal scores for less challenging SOT conditions (e.g., 1, 2, and 3) accompanied by normal scores for more difficult conditions (e.g., 4, 5, and 6); (2) evidence of voluntary control of posture, such as prolonged motor latencies or rhythmic or increased sway patterns; (3) inconsistent performance across trials and within conditions; and (4) evidence of subject anxiety, such as increased strength and decreased latencies for MCT.

RESULTS

Screening

Six subjects were screened. One subject passed the screening and was not evaluated further. Five subjects failed the screening and three elected to continue in the study.

Full Assessment

Twenty-seven subjects were evaluated, including the three who failed the screening. Five subjects were seen during all four quarters. Of these, only one had an ENG abnormality. For this subject, benign paroxysmal positioning vertigo (BPPV) was detected in Q1 and resolved spontaneously by Q2. Four subjects had abnormal SOT patterns, one surface dependent and three vestibular. All but one subject were normal by Q4. The subject with BPPV had an unresolved vestibular pattern. Results for all 27 subjects are described below. Sample size for ENG was Q1 = 9, Q2 = 16, Q3 = 20, Q4 = 24. Sample size for CDP was Q1 = 7, Q2 = 17, Q3 = 19, and Q4 = 21.

Electronystagmography

Table 3 shows ENG results for individual subjects. The majority of findings were normal by the second quarter: 3 subjects were normal for Q1 (33%), 13 for Q2 (81%), 14 for Q3 (70%), and 19 for Q4 (79%). ENG abnormalities were predominantly peripheral or nonlocalizing, with positional nystagmus being the most common finding. Four subjects had positive findings that appeared inconsistently during the year. Specific results for the year are delineated below and summarized in Table 4.

Nonrecorded Gaze Nystagmus. One subject (4%) had direction-fixed horizontal gaze nystagmus (detected during a Q1 hearing evaluation and resolved by Q2).

Recorded Gaze Nystagmus. One subject (4%) had recordable down-beating nystagmus (Q4).

Fistula Test. There were no positive fistula tests.

Oculomotor Tests. One subject (4%) had abnormal smooth pursuit. This subject also had the down-beating nystagmus, sustained head injury, and had a mild, bilateral hearing loss. No subjects had saccadic abnormalities.

Dix-Hallpike. There were three cases of BPPV (11%). Two subjects who sustained head injury had positive Dix-Hallpike maneuvers in Q1. One case self-resolved by Q2, and the other
Table 3  Individual Results (n = 27) for Electronystagmography (ENG), Posturography, Sensory Organization Test (SOT), and Subjective Perception of Dizziness

<table>
<thead>
<tr>
<th>ID #, Hearing Loss, Head Injury, and Age</th>
<th>ENG</th>
<th>SOT</th>
<th>Subjective Perception</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First Exam</td>
<td>Last Exam</td>
<td>First Exam</td>
</tr>
<tr>
<td>1 B, 25</td>
<td>N²</td>
<td>Positional nystagmus³</td>
<td>DNT²</td>
</tr>
<tr>
<td>6 U, 45</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>11 B, HI, 48</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>15 N, HI, 27</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>16 B, HI, 48</td>
<td>Smooth pursuit¹</td>
<td>Smooth pursuit, down-beating nystagmus²</td>
<td>N²</td>
</tr>
<tr>
<td>18 N, HI, 31</td>
<td>BPPV¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>22 B, HI, 49</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>34 N, 50</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>35 B, HI, 52</td>
<td>Nonrecorded gaze nystagmus¹, positional nystagmus, unilateral weakness²</td>
<td>N²</td>
<td>5, 6³</td>
</tr>
<tr>
<td>39 B, 41</td>
<td>Unilateral weakness¹</td>
<td>Positional nystagmus⁴</td>
<td>CNT³</td>
</tr>
<tr>
<td>40 N, 33</td>
<td>N²</td>
<td>N²</td>
<td>4, 5, 6³</td>
</tr>
<tr>
<td>41 N, 47</td>
<td>Positional nystagmus³</td>
<td>N³</td>
<td>5¹</td>
</tr>
<tr>
<td>42 N, 40</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>44 B, HI, 41</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>45 B, HI, 44</td>
<td>N¹</td>
<td>N¹</td>
<td>N¹</td>
</tr>
<tr>
<td>46 B, 50</td>
<td>N²</td>
<td>N²</td>
<td>4, 5, 6²</td>
</tr>
<tr>
<td>48 U, 51</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>58 B, HI, 45</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>62 U, HI, 28</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>72 B, HI, 63</td>
<td>BPPV¹</td>
<td>N³</td>
<td>N³</td>
</tr>
<tr>
<td>74 U, HI, 25</td>
<td>Positional nystagmus¹</td>
<td>N³</td>
<td>N³</td>
</tr>
<tr>
<td>81 B, 49</td>
<td>Positional nystagmus¹</td>
<td>BPPV¹</td>
<td>N²</td>
</tr>
<tr>
<td>83 B, HI, 61</td>
<td>N³</td>
<td>N³</td>
<td>N³</td>
</tr>
<tr>
<td>86 N, HI, 29</td>
<td>N²</td>
<td>N²</td>
<td>N²</td>
</tr>
<tr>
<td>87 N, HI, 32</td>
<td>Positional nystagmus¹</td>
<td>Positional nystagmus¹</td>
<td>4, 5, 6¹</td>
</tr>
<tr>
<td>88 U, 54</td>
<td>Positional nystagmus²</td>
<td>N¹</td>
<td>5, 6²</td>
</tr>
<tr>
<td>92 B, HI, 49</td>
<td>DNT²</td>
<td>N²</td>
<td>N²</td>
</tr>
</tbody>
</table>

Under the subject identification column, hearing loss is defined as age-corrected thresholds ≥ 22.5 dB HL for one or more frequency(s) during the first audiologic visit (Q1 or Q2). U = unilateral hearing loss, B = bilateral hearing loss, N = normal hearing bilaterally. HI = head injury incurred the day of the bombing. Under ENG and SOT columns, N = normal results, DNT = did not test, CNT = could not test. Superscripts refer to test quarter.

resolved by Q3 after a canalith repositioning procedure during Q2. The third subject had a positive Dix-Hallpike for the first time during Q4 and did not have head injury.

Positional Tests. Eight subjects had positional nystagmus during the year (30%). It was present for one subject (11%) in Q1, two (13%) in Q2, five (25%) in Q3, and two (8%) in Q4. Resolution of nystagmus was documented in four cases. One subject had resolution of nystagmus and vertigo after doing Cawthorne-Cooksey exercises (Cawthorne, 1944; Cooksey, 1946) three times per day for 2 weeks.

Caloric Irrigations. Two subjects (7%) had right unilateral caloric weakness. The first subject also had mild bilateral sensorineural loss at 250 Hz but no head injury. The second subject suffered a skull fracture and loss of an eye on the left side. He reported no vestibular symptoms but had the direction-fixed left-beating gaze nystagmus during Q1. Gaze nystagmus resolved by Q2, but ENG showed positional nystagmus. This subject also had mild-to-severe bilateral high-frequency sensorineural hearing loss. There were no subjects with directional preponderance or failure of fixation suppression.
Table 4 Electronystagmography

<table>
<thead>
<tr>
<th>Subtest</th>
<th>Number of Subjects with Positive Findings</th>
<th>Percent of Subjects with Positive Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonrecorded gaze nystagmus</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Recorded gaze nystagmus</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fistula</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal horizontal saccades</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Disorganized smooth pursuit</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Benign paroxysmal positioning vertigo</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Positional nystagmus</td>
<td>8</td>
<td>30</td>
</tr>
<tr>
<td>Bithermal caloric weakness</td>
<td>2</td>
<td>7</td>
</tr>
</tbody>
</table>

Computerized Dynamic Posturography

Two subjects had obvious nonorganic signs and were eliminated from the database. One demonstrated apparent mental confusion and inability to follow instructions, and the other was uncooperative. For the latter, observed behavior included free falling forward and hanging in the harness. Upon reinstruction, performance was within normal limits for subsequent trials within the same condition.

The sample size for CDP was 25 since two subjects had physical restrictions and could not be evaluated. One subject had preexisting neuromuscular disease and the other had excessive weight.

Sensory Organization Test

Table 3 shows SOT results for individual subjects. Fourteen subjects (56%) had clear, easily interpreted SOT patterns. Eleven subjects (44%) had SOT results that were borderline abnormal, had nontraditional patterns (Cyr, 1991; Nashner, 1993b), or had inconsistencies within conditions for any given quarter. These data were not eliminated from the database due to clinical impressions that subjects were trying to give a best performance. Consequently, data were subjected to the three-tiered evaluation process.

The agreement rate between judges was 92%, while each judge agreed with the equation method 60% of the time. All three methods agreed upon SOT classifications only 56% of the time. There was no instance where none of the methods agreed.

Averaged across quarters, a normal pattern was predominant (68%), followed by vestibular (15%), surface-dependent (13%), and physiologically inconsistent patterns (4%). Results for each quarter are seen in Figure 1. For subjects who had head injury, 69% of 42 evaluations (29) were normal, 17% were surface dependent (7), 12% were vestibular (5), and 2% were nonphysiologic (1).

Of subjects with abnormal patterns during the year (physiologically consistent or inconsistent) (n = 12), two returned to normal by Q2, one by Q3, and three by Q4. Therefore, 50 percent of subjects with abnormal results were normal by 1-year postblast. For two subjects, physiologically consistent abnormalities were not detected until Q4. No subject was nonphysiologic for more than one test session. Two subjects had variable findings between quarters (i.e., surface dependent, then nonphysiologic; and nonphysiologic, normal, then vestibular).

Motor Control Test

Motor control results also were predominantly normal. There were four cases of notable, probably neurologically based MCT abnormalities. One subject had bilateral, bidirectional, prolonged latencies and during Q1 had a "5" SOT pattern. Although he had head injury, he also had auditory signs and disconjugate gaze that predated the explosion. The second subject had bilateral, bidirectional, elevated amplitude scaling. He was the subject with down-beating nys-
tagmus, abnormal smooth pursuit, and a “5” SOT pattern. He sustained head injury from the blast. The third subject also had head injury. His MCT abnormalities were bilateral, bidirectional, prolonged latencies and poor toes-down adaptation. This is the same subject with direction-fixed gaze nystagmus, positional nystagmus, and a unilateral weakness. The fourth subject had bilateral, bidirectional, elevated amplitude scaling, poor toes-down adaptation, and huge raw MCT responses overlaid with tremor. She had positional nystagmus during Q3 but did not report head injury.

Five other subjects had some elevated amplitude scaling sporadically throughout the year, which was thought to be related to test anxiety. As a group, mean MCT latency and amplitude data agreed well with published norms (Nashner, 1993a). There were no group changes across quarters that did not fall within the range of published standard deviations.

**ENG and CDP**

Both tests detected deficits equally well. Eleven of 27 subjects (41%) had positive ENG findings and 11 of 25 subjects (44%) had physiologically consistent CDP abnormalities for at least one quarter. Fifteen of 25 subjects (60%) were abnormal for one or both tests. Nine of 25 subjects (36%) were abnormal for only one test. Electronystagmography and CDP both were abnormal for 6 of 25 subjects (24%). One of the subjects who could not be tested with CDP had a negative ENG and the other had a positive ENG.

Results were very similar for subjects with reported head injury (one of whom could not do CDP). Six of 16 subjects (38%) had ENG abnormalities and 7 of 15 (47%) had CDP abnormalities for at least one quarter. Nine of 15 subjects (60%) were abnormal for one or both tests. Four of 15 subjects (27%) were abnormal for only one test. Electronystagmography and CDP both were abnormal for four subjects (27%).

**Questionnaire**

Twenty-seven of 30 subjects who were screened or fully evaluated completed a questionnaire during their first visit.

**Location**

There was no obvious relationship between precise location and vestibular symptoms or test results. However, of 83 subjects in the audiologic study (Van Campen et al, 1999), 34 of 36 people reporting dizziness or imbalance were located inside buildings at the time of the blast. Of 30 subjects screened or evaluated for vestibular/balance disturbance, 29 were inside a building. These buildings were fairly evenly distributed around ground zero. Ten of these subjects were from the Federal Building. The furthest distance from ground zero that resulted in a complaint of dizziness was eight blocks north. This individual was a pedestrian and also suffered sensorineural hearing loss, presumably from reflections off a nearby building. Figure 2 graphically depicts location information.

**Time Course of Dizziness**

Table 5 summarizes the time course and character of reported dizziness. Thirteen of 27 subjects (48%) reported dizziness the day of the explosion. Four people reported dizziness the following day, so that 63% of subjects had their first episode within 48 hours of the event. Nine subjects had their first episode sometime after 48 hours. Two of the nine were unsure about the exact onset of symptoms. Seven subjects had their first episode at 5-, 6-, 11-, 12-, 13-, 61-, and 68-days postblast. The subject with positive nonrecorded, direction-fixed gaze nystagmus, positional nystagmus, and unilateral weakness did not experience symptoms.

The duration of reported dizziness ranged from seconds (n = 12), minutes (n = 9), hours (n = 1), or days (n = 4). All but four subjects reported freedom from dizziness between attacks.

Persistence of symptoms over time was addressed only with subjects who were fully evaluated each quarter. They were asked if the symptoms were the “same” or “worse” than when first noted, or if they “come and go” or were “gone.” Responses for each quarter are shown in Figure 3. The predominant response during each quarter was that the symptoms would come and go. At 1-year postblast, 67% of subjects queried (16 of 24) reported that symptoms either came and went or were the same as when first noted.

Analyzing fourth quarter data by head injury did not illuminate why symptoms were uncompensated. Figure 4 illustrates that subjects with and without head injury had similar test results and perceptions of dysequilibrium. In fact, proportionately more subjects with head injury reported their symptom(s) to be gone than subjects without head injury. Age also did
Figure 2  Aerial view of geographic area that sustained 90% of building damage. 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. Icons represent locations for 83 subjects enrolled in the complimentary audiologic study (Van Campen et al., 1999) from which vestibular subjects (n = 30) were recruited. Red denotes subjects who were evaluated for dizziness. Yellow denotes subjects not evaluated for dizziness. Orange denotes locations that had both vestibular and non-vestibular subjects. Person icons designate pedestrians, bulls-eyes designate subjects inside a vehicle, and houses designate subjects inside a building. House icons may represent more than one subject.
not seem to be a factor since those with and without perceived dizziness had almost identical means (43.7 years and 43.2 years, respectively).

**Character of Dizziness**

Eighteen of 27 subjects (67%) reported spinning vertigo. Other subjects reported imbalance, light-headedness, or near-syncope. Fifteen subjects (56%) felt that their symptoms occurred in certain positions, and 23 (85%) reported no warning before an episode (see Table 5).

**Associated Signs and Symptoms**

Fifteen of 27 subjects reported nausea with their dizziness; however, only three had episodes of vomiting. Tinnitus was reported by 21 subjects (78%), aural pressure for 15 subjects (56%), and otalgia for 12 (44%). Twenty-two of 30 subjects (73%) had documented age-corrected-thresholds hearing loss of ≥22.5 dB HL at one or more frequency(s) for at least one ear during Q1 or Q2 (auditory methodology is delineated in Van Campen et al, 1999). Other reported symptoms included aural drainage, pressure inside of head, headache, double vision, blurred vision, spots before the eyes, facial numbness, facial tingling, limb numbness, limb weakness, limb clumsiness, mental confusion, difficulty with speech, and difficulty swallowing.

**History**

Two subjects had a previous episode of dizziness, one 22 years and one 5 years prior to the blast. Seventeen of 30 subjects (57%) reported sustaining a head injury on April 19, 1995. Specific head injuries, as shown in Figure 5, included skull fracture, concussion, aural bleeding, head and face lacerations, and loss of consciousness.

One subject reported having a myringotomy and tubes in the past; otherwise, there were no significant otologic histories. Medication use included one subject on dilantin and one on a tranquilizer. Both of these subjects had normal ENG and CDP results for all evaluations. No sig-
Vestibular injury presents even greater assessment challenges since preblast data are seldom available, and since symptom descriptions can vary widely between subjects. Furthermore, current test methods have limited ability to detect pathology/dysfunction, and factors such as anxiety, sleep deprivation, and diet can influence vestibular assessments. Longitudinal studies have additional constraints due to inconsistent subject participation. Nonetheless, this investigation revealed clinically relevant vestibular sequelae to a large blast.

The occurrence of vestibular symptoms following a single blast is unclear from previous literature. Percentages have restricted meaning since methodology and definitions were not delineated. Vestibular disturbances have been reported as "uncommon" (Kerr and Byrne, 1975) and having rates of 1, 7, 13, 60, and 100 percent (McReynolds et al, 1949; Pahor, 1981; Shupak et al, 1993; Singh et al, 1983; Chandler and Edmond, 1997). From the Oklahoma City blast, 44 percent of 82 subjects queried (Van Campen et al, 1999) reported postblast symptoms of dizziness/light-headedness. This occurrence rate coincides nicely with results of an extensive survey conducted by the Oklahoma State Department of Health. In their interviews with 494 bombing survivors, 166 (34%) reported a new onset of "dizziness/light-headedness" since the blast.

The occurrence of empirical evidence of vestibular dysfunction is even less well known. McReynolds et al (1949) briefly mentioned that 18 of 143 (13%) subjects had caloric weakness, and Shupak et al (1993) documented vestibulopathy in three of five subjects (60%). While our occurrence of caloric weakness was lower (7%), there were 41 percent positive ENG findings and 44 percent positive CDP results. For the combined test battery, there was a 60 percent rate of dysfunction. These figures are strikingly higher than the 5 percent occurrence of imbalance in the general, asymptomatic population (Nashner, 1993a), lending support to the conclusion that exposure to the blast overpressure was responsible for reported and documented vestibular dysfunction.

Physiologic findings were mostly peripheral or nonlocalizing and agreed with those previously reported: horizontal gaze nystagmus,
Vestibular Outcomes of Oklahoma City Bomb/Van Campen et al

BPPV, positional nystagmus, and caloric weakness (McReynolds et al, 1949; Kerr and Byrne, 1975; Pahor, 1981; Shupak et al, 1993). Downbeating nystagmus and disorganized smooth pursuit have not been reported previously, but these likely were due to head injury.

Positional nystagmus was the most common finding, which is not surprising since it can be measured in asymptomatic individuals (Barber and Wright, 1973). Rather than adopt a more stringent criterion for abnormality, however, we decided that any positional nystagmus would be considered aberrant so as not to overlook a significant sign that might correlate with patient complaints. Cyr (1990) and Brookler and Rubin (1993) state that all nystagmus is abnormal, whether or not it reaches clinical significance, since it reflects asymmetric vestibular output.

The time course and character of postblast dysequilibrium also is not well documented in previous literature. Only Chandler and Edmond (1997) reported immediate vertigo. Three of Shupak et al's (1993) five subjects reported vertigo within a week of blast exposure. In the present study, 63 percent of subjects had their first episode of dizziness or vertigo within 48 hours, but the range was within 24 hours to 68 days. Episodes occurred spontaneously in most subjects (85%) and/or in certain positions (56%) and lasted from seconds to days. Our occurrence of spinning vertigo (67%) agrees well with Shupak et al's (1993) 60 percent rate (three of five). Chandler and Edmond (1997), however, had a 100 percent rate (five of five subjects). Other articles do not differentiate “dizziness” or “giddiness” from “vertigo.”

It was surprising that 67 percent of Oklahoma City subjects felt that their symptoms either were unchanged or still occurred intermittently at 1 year. Additionally, only 6 of 12 subjects (50%) with abnormal SOT results were normal at 1-year postblast. Oklahoma State Department of Health data (see footnote 1) corroborate this finding since 81 percent of people reporting dizziness/light-headedness felt that the symptom had not resolved at the time of interview (September 1996 to September 1998).

Vestibular compensation clearly should be active within this period (Igarashi, 1984; Smith and Curthoys, 1989) and is given as an explanation for the lack of symptomatology following traumatic aural injury or chronic noise exposure (Shupak et al, 1993, 1994). Pahor (1981) did report a case of “giddiness” with a unilateral caloric weakness that took 2 years to resolve. In contrast, all of Shupak et al’s (1993) and Chandler and Edmond’s (1997) subjects were symptom free within 3 weeks of injury.

The persistence of symptoms in the current study could be due to several factors. A little over half of the subjects reported head injury, and it is possible that some resulted in central nervous system dysfunction that would inhibit central compensation. However, fourth quarter data do not indicate a strong difference between subjects with and without head injury regarding perceived disability or behavioral performance. Another possibility is that the psychological impact of the event altered the subjects’ activity level, anxiety level, and/or sleep patterns. Although this is conjecture, it seems plausible since reduced physical energy or mental motivation can inhibit compensation (Bailey et al, 1993; Sullivan et al, 1993).

Two pathophysiologic explanations could be undetected perilymphatic fistulas or post-traumatic endolymphatic hydrops. Both conditions produce a fluctuating baseline of vestibular output that will not trigger the central compensation process. The possibility of fistulas in our sample is very low since there were no positive fistula tests with ENG, and since patients did not report exacerbation or initiation of symptoms with increased intracranial pressure. It is more likely that some subjects suffered from post-traumatic endolymphatic hydrops. The high occurrence of spontaneous vertigo (85%), delayed initiation of symptoms (e.g., up to 68-days postblast), and cases of variable positive test findings would support this conclusion.

Animal and human studies provide evidence that intense acoustic stimulation can result in damage to cells responsible for production or absorption of endolymph or disruption of the endolymphatic sac (Clark and Rees, 1977; Paparella and Mancini, 1983; Ylikoski, 1988; Kumagami, 1992). Of particular interest is that intense infrasound (<20 Hz), which is heavily weighted in a bomb explosion, produced hydrops in chinchilla (Lin et al, 1982). Reportedly, vestibular susceptibility to infrasound is due to the resonant frequency range of semicircular and otolithic structures, which encompass frequencies < 20 Hz (Evans, 1976).

Animal and human data also indicate that acoustic trauma to vestibular apparatus is secondary to severe injury to the organ of Corti (Mangabeira-Albernaz et al, 1959; Man et al, 1980). Our high occurrence of hearing loss (73%) and tinnitus (78%) in dizzy subjects upholds this relationship with blast exposure. This injury
progression seems to be supported by our locale information as well.

There was no obvious relationship between individual location relative to the blast and subsequent signs, symptoms, or test results. However, 94 percent of people reporting dizziness from the corresponding audiologic study (Van Campen et al, 1999) were in buildings. Additionally, 97 percent of subjects screened or evaluated were located inside a building during the blast. Since blast overpressure and subsequent sound pressure levels can increase within closed spaces (Yelverton et al, 1996), it may be postulated that some subjects in certain buildings experienced vestibular symptoms due to pressure levels that exceeded those outdoors, inside vehicles, or inside other buildings. Future research may want to explore the structural characteristics of these particular buildings.

Some papers dismiss postblast dizziness as being related to head injury and postconcussion syndrome (McReynolds et al, 1949; Kerr and Byrne, 1975; Kerr, 1978). As such, it would be considered a secondary (resulting from ballistic environmental materials) or tertiary (resulting from whole-body displacement) blast injury. Although dizziness certainly can follow concussion (Barrett et al, 1994) and head injury (Rubin et al, 1995), it is noteworthy that 43 percent (13 of 30) of the present study's symptomatic subjects did not report head injury, and seven of them had abnormal ENG or CDP results. Even more striking is that none of Shupak et al's (1993) subjects had head injury, yet three of five men reported vertigo with subsequent peripheral abnormalities on ENG and SHA testing. These findings are highly supportive that vestibular blast injury can be pressure induced and, therefore, can be considered a primary blast injury.

Furthermore, many ENG abnormalities were peripheral in origin and there were no traditional head injury patterns on CDP (3, 6 or 3, 5, 6) (Cyr, 1991). Most CDP test results from head injury subjects were normal (69%). Two subjects did have "multisensory" patterns (2, 3, 4, 5, 6 and 2, 4, 5, 6), which were judged to be predominantly surface-dependent patterns, and three had motor control involvement. The predominant deficits, therefore, were either vestibular in origin or revealed a dependence on somatosensory information. The latter (4, 5, 6 pattern) can be seen with a weakened vestibular response but is an unusual manifestation. Encumbered performance on condition 4 could represent poor selection processing resulting from both brain and aural concussion. As a result, the individual has difficulty suppressing irrelevant information and processing salient cues (Cyr, 1991; Nashner, 1993b).

A test battery approach is supported by the fact that both tests detected disorders equally well and contributed either novel or supportive information. Computerized dynamic posturography was included in the battery to differentiate between forms of postural ataxia, such as vestibular deficits or multisensory postconcussion syndrome (Lipp and Longridge, 1994). While CDP is not site specific (i.e., diagnostic), it is nevertheless system specific, and added valuable information to the gestalt of balance.

During the year, CDP detected deficits in 20 percent of subjects when ENG was normal. Conversely, ENG detected abnormalities 16 percent of the time when CDP was normal. Of 10 subjects with a normal test battery at every evaluation, 5 continued to report subjective dysequilibrium at 1 year. This illuminates the limitations of current tests, especially for cases with spontaneous vertigo that were symptom free between attacks. Inclusion of both tests improved the detection of dysfunction by almost one-third. Quarter 4 ENG and CDP accurately demonstrated normalcy for three of five subjects (60%) who reported resolution of symptoms.

The cases of physiologically inconsistent SOT findings for CDP were thought provoking. As reported earlier, there were only two (6%) cases of obvious nonorganic behavior. The other cases were not straightforward. These individuals had consistent case histories and seemed to try to give a best performance. As far as the authors were aware, there were no legal suits pending at the time so financial compensation did not seem to be a factor. This leads to the prospect that test anxiety played a role in performance. Many subjects were noticeably distressed with movement of the visual surround or platform, possibly due to the sensory similarity to an explosion experience. Additional evidence that these performances were not "nonorganic" in the traditional sense is that no one performed in this manner consistently across quarters.

Subsequent vestibular blast injury studies should investigate in more depth the compelling issues of physiologically inconsistent patterns on CDP, fluctuating perceptions of dizziness, variations in test results, and poor functional compensation (both subjective and objective). These might be addressed best by correlating the results of vestibular studies with formal neurologic and psychological examinations and with a
standardized tool for self-reported dizziness handicap. Additionally, controlled study of balance rehabilitation protocols would be beneficial.

CONCLUSION

This study showed that vestibular blast injury is a demonstrable outcome of exposure to blast overpressure and clarifies clinical presentations of survivors with vestibular complaints. Vestibular symptoms occurred as early as the first 24 hours or took as long as 2.5 months to develop. Empirical documentation of symptoms may be difficult to achieve due to a predominantly spontaneous, intermittent nature of the deficit. A serial, test battery approach is recommended to increase detection sensitivity.

For 60 percent of 27 survivors, blast-induced, nonauditory, labyrinthine injury had clinical significance for ENG and/or CDP. Although 59 percent of these subjects had head injury, typical head injury patterns for ENG or CDP were not documented, lending support to the idea that vestibular trauma can be directly related to blast overpressure (i.e., a primary blast injury). Especially for cases without clinically positive findings, subjective reporting of symptoms provided valuable information as to the character and persistence of the deficit and may be an important indicator of functional compensation.

Continued study of the nature and etiology of vestibular blast trauma is imperative to better serve future blast victims. Toward this end, vestibular trauma needs to be considered a valid component of aural blast injuries and should be included routinely in the evaluative and rehabilitative process.

Acknowledgment. The authors sincerely appreciate the subjects who volunteered their time and effort. We also thank David Cyr, Patricia Burnett, Jim Saunders, Mark Wood, and Mark Stephenson for constructive ideas and reviews; Reginald Tempelmeyer for data management and form designs; Bobbie Borchardt, manager of Oklahoma City's Geographic Information Systems, for mapping the area; Quentin Baker from Wilfred Baker Engineering, Inc., San Antonio, TX, for calculating the bomb's physical characteristics; and Lewis Nasher for serving as a blind judge.

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REFERENCES


APPENDIX

I. Time Course of Dizziness

1. When did your dizziness first occur?
   
   
   
   Month Day Year

2. How long did the first attack last?

   A  Seconds
   B  Minutes
   C  Hours
   D  Days

3. Is your dizziness constant?
   No  Yes

4. Is your dizziness in attacks?
   No  Yes

5. On the average, how long do the attacks last?

   A  Seconds
   B  Minutes
   C  Hours
   D  Days

6. How long is the period between attacks?

   A  Hours
   B  Days
   C  Weeks
   D  Months

7. Are you completely free of dizziness between attacks?
   No  Yes

II. Characteristics of Dizziness

8. Do you experience any of the following sensations when you are dizzy? (Check all that apply).

   A  Looking up
   B  Looking down
   C  Looking right
   D  Looking left
   E  Bending over
   F  Sitting to lying
   G  Lying on back
   H  Rolling to or lying on right side
   I  Rolling to or lying on left side
   J  Lying to sitting
   K  Sitting to standing

9. Does your dizziness occur in certain positions?
   No  Yes
   If yes, please specify: ___________________

10. Do you get dizzy when you:

    No  Yes

   A  Blow your nose
   B  Cough
   C  Exert yourself
   D  Hear a loud sound

11. Does your dizziness ever occur without warning?
   No  Yes

12. Does anything make your dizziness better or make it go away?
   No  Yes
   If yes, please specify: ___________________

13. Does anything make your dizziness worse?
   No  Yes
   If yes, please specify: ___________________
III. Associated Signs and Symptoms

14. Please check any of the following symptoms that you have experienced. Please check whether the symptom is constant or in episodes.

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<tr>
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<th></th>
<th>Constant</th>
<th>Episodes</th>
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<tbody>
<tr>
<td>A</td>
<td>Nausea</td>
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<tr>
<td>B</td>
<td>Vomiting</td>
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<td></td>
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<td>C</td>
<td>Impaired hearing, right ear</td>
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<td>D</td>
<td>Impaired hearing, left ear</td>
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<td>E</td>
<td>Noises in right ear</td>
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<td>F</td>
<td>Noises in left ear</td>
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<td>G</td>
<td>Pressure or fullness in right ear</td>
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<td>Pressure or fullness in left ear</td>
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<td>I</td>
<td>Pain in right ear</td>
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<td>Discharge from right ear</td>
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<td>Discharge from left ear</td>
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<td>Pressure in head</td>
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<td>Headache</td>
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<td>O</td>
<td>Double vision</td>
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<td>P</td>
<td>Blurred vision or blindness</td>
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<td>Q</td>
<td>Spots before eyes</td>
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<tr>
<td>R</td>
<td>Numbness in face</td>
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<td>S</td>
<td>Tingling around mouth</td>
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<td>T</td>
<td>Numbness in arms or legs</td>
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<td>U</td>
<td>Weakness in arms or legs</td>
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<td>V</td>
<td>Clumsiness in arms or legs</td>
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<td>W</td>
<td>Confusion</td>
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<tr>
<td>X</td>
<td>Difficulty with speech</td>
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<tr>
<td>Y</td>
<td>Difficulty with swallowing</td>
<td></td>
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</table>

IV. General

15. Have you ever had a head injury(s)?
   ___ No   ___ Yes   If yes, when?
   ___________ ___________ ___________
   Month  Day  Year
   ___________ ___________ ___________
   Month  Day  Year
   ___________ ___________ ___________
   Month  Day  Year

16. Have you had any of the following injuries?
   No  Yes
   ___  ___  
   A  Skull fracture
   B  Concussion
   C  Bleeding from ears
   D  Cuts or lacerations to face or scalp
   E  Loss of consciousness

17. Have you ever had a neck injury?
   ___ No   ___ Yes   If yes, please specify: ____________________________

18. Have you ever had a back injury?
   ___ No   ___ Yes   If yes, please specify: ____________________________
19. Do you use alcohol?
   ____ No   ____ Yes   If yes, how much per day? ____________________________

20. Are you currently taking any medication?
   ____ No   ____ Yes   If yes, please specify: ________________________________

21. Have you ever had ear surgery?
   ____ No   ____ Yes   If yes, please specify: ________________________________

Thank you for filling out this questionnaire.