Otologic injuries caused by airbag deployment

WILLIAM J. McFEELY, JR, MD, DENNIS I. BOJRAB, MD, KENT G. DAVIS, MD, and **DOUGLAS F. HEGYI, DO,**
Farmington Hills, Southfield, Detroit, and Lapeer, Michigan

Airbags are clearly successful at mitigating injury severity during motor vehicle accidents. Deployment unfortunately has introduced new injury-causing mechanisms. A retrospective review of 20 patients who sustained otologic injuries resulting from airbag inflation was conducted. The most common symptoms were hearing loss in 17 (85%) and tinnitus in 17 (85%). Objective hearing loss was documented in 21 of 24 (88%) subjectively affected ears; this included unilateral and bilateral sensorineural, unilateral conductive, and mixed hearing losses. Ten patients (50%) had dysequilibrium. Four subjects (20%) had a tympanic membrane perforation; each required surgical closure. Ear orientation toward the airbag was found to be associated with hearing loss ($P = 0.027$), aural fullness ($P = 0.039$), and tympanic membrane perforation ($P = 0.0004$). A wide variety of airbag-induced otologic injuries occur and may have long-term sequelae. It is important for health care personnel to be aware of these potential problems. (Otolaryngol Head Neck Surg 1999;121:367-73.)

As of September 1, 1998, federal regulations have required that airbags be installed in all new passenger cars, vans, pickup trucks, and utility vehicles.[1] Airbags clearly are successful at decreasing injury severity during motor vehicle accidents (MVAs).[2,3] Based on all types of crashes, they decrease fatalities by 21% to 22% for drivers not wearing seatbelts and by 9% to 16% for drivers wearing seatbelts.[4-6]

Airbag deployment unfortunately has introduced a new spectrum of injuries; numerous reports of injury patterns exist.[5,7-17] Most are relatively minor and usually consist of erythema, abrasions, and contusions to the face, anterior neck, or upper chest.[5,16] More serious harm is rare, but there are accounts of life-threatening trauma in the literature.[9,10,16] Damage to the eye,[7] cervical spine,[8,9,16] facial nerve,[17] temporomandibular joint,[11] facial skeleton,[7,8] and upper airway[15] are of particular importance to the practicing otolaryngologist. Recent reports have documented a total of 13 persons with otologic symptoms of hearing loss, tinnitus and/or dysequilibrium.[13,14,18-22]

A number of patients with unusual otologic findings after airbag deployment were recently encountered in our practice. Many of these problems are yet to be described in the literature. The purpose of this study is to retrospectively review these patients and to discuss all relevant literature.

METHODS AND MATERIAL

Patients with otologic symptoms after airbag deployment between April 1995 and April 1998 were retrospectively studied. Twenty airbag subjects were extracted from a total of 109 charts of MVA patients. Eighty-nine patients were excluded because of lack of airbag deployment. A retrospective chart review was conducted; a telephone questionnaire was performed to acquire any missing subjective data.

Each patient underwent a complete neuro-otologic examination. Testing was not standardized given the retrospective nature of this study. Pure-tone audiometry and speech audiometry were performed after the accident and periodically after initial evaluation in nearly all subjects. Hearing tests completed less than 1 year before the MVA were retrieved for comparison purposes when possible. Postaccident audiometric testing was divided into 2 groups: early (<1 month after the MVA) and late (≥1 month after the MVA). A 4-frequency (0.5, 1, 2, and 3 kHz) pure-tone average (PTA) was calculated in standard fashion for each audiogram.

A number of statistical methods were used for data analysis. Fisher's exact test was used for independence between categorical variables. Differences between groups were tested with the Mann-Whitney test. The paired *t* test was used for comparison of audiometric PTA scores. Both affected ears (ie, in cases of bilateral involvement) were included in calculations for certain variables when appropriate. Driver inboard (right) ears were considered to have been oriented toward a deploying passenger side airbag when overall head positioning was straight ahead.

From the Michigan Ear Institute and Providence Hospital (Drs McFeely and Bojrab); the Department of Otolaryngology, Wayne State University (Dr Davis); and Lapeer Medical Center (Dr Hegyi).
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Reprint requests: Dennis I. Bojrab, MD, Michigan Ear Institute, 27555 Middlebelt Rd, Farmington Hills, MI 48334.

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Table 1. Subject accident information

Patient no.	Sex	Age (y)	Car type	Collision description	Airbags deployed	Head positioning to airbag	Follow-up (mo)
1	M	50	1996 Dodge Dakota Pickup	Frontal impact with side of car turning in front of him; speed 35 mph	Driver	Left	24
2*	F	45	1996 Ford Contour	Frontal impact with rear of car at rest for light; speed only 5 mph	Both	Right	10
3	M	64	1993 Chrysler LeBaron Convertible	Frontal impact with side of car turning in front of him; speed 20 mph	Driver	Center	9
4†	M	7	1995 Ford Windstar Minivan	Frontal impact with deer; speed 25 mph	Both	Left	17
5†	F	75	1994 Mercury Sable	Frontal impact with side of car turning in front of him; speed 20 mph	Both	Center	11
6	F	72	1996 Honda Accord	Frontal impact with mailbox; speed 25 mph	Driver	Center	15
7	F	57	1991 Dodge Dynasty	Right front impact with front of car that ran red light; speed 40 mph	Driver	Center	19
8	F	36	1996 GMC Safari Minivan	Left front impact with front of car head on; speed 75 mph combined	Both	Left	15
9	F	63	1994 Chrysler Minivan	Frontal impact with front of car head on; speed 60 mph combined	Both	Left	26
10	F	68	1992 Ford Taurus	Right front impact with front of car that ran stop sign; speed 30 mph	Driver	Center	36
11	M	42	1995 Ford Escort	Frontal impact with stopped vehicle; speed 40 mph	Driver	Center	18
12	F	68	1995 Ford Villager	Left front impact with stopped vehicle; speed 25 mph	Both	Center	18
13	F	35	1991 Dodge Stealth	Right front impact with car that ran red light; speed 80 mph combined	Driver	Center	34
14	F	27	1992 Toyota Camry	Right front impact with front of turning car; speed 75 mph combined	Driver	Center	17
15	M	47	1997 Ford Explorer	Frontal impact with telephone pole; speed 40 mph	Both	Center	9
16	F	22	1994 Chrysler LeBaron	Frontal impact with light pole; speed 45 mph	Driver	Center	14
17*	M	18	1993 Dodge Shadow	Frontal impact with stopped vehicle; speed 35 mph	Driver	Center	4
18	M	57	Unknown	Frontal impact with turning vehicle; speed 60 mph combined	Both	Center	19
19	M	46	1992 Ford Aerostar Van	Left front impact with oncoming vehicle; speed 55 mph combined	Driver	Right	40
20	M	57	1995 Nissan Maxima	Frontal impact with car at light; speed 40 mph	Both	Center	4

GMC, General Motors Corp.
*At least 1 window was open 1 to 2 inches.
†Subject was passenger rather than driver.

RESULTS

Subject demographic and accident information is outlined in Table 1. There were 11 female and 9 male patients, with a mean age of 48 years (range 7 to 75 years). Nearly all patients were the driver of the car, and the driver-side airbag deployed in all accidents studied. Passenger-side airbag deployment occurred in 9 of 20 (45%). All patients were wearing seatbelts, although patient 4 (a child) was improperly restrained at time of impact. Follow-up averaged 18 months (range 4 to 40 months).

A wide variety of automobiles were involved. All vehicles sustained a frontal or near-frontal impact. Average speed of impact was 42 mph (range 5 to 80 mph). No patient sustained a skull base fracture or significant closed head injury.

History of previous otologic problems before the MVA was obtained; results are listed in Table 2. Three patients had undergone surgery previously for chronic ear disease. Each of these subjects had been doing well before the accident with no evidence for active disease or perforation. Mild preexisting subjective hearing loss was described in 9 of 20 (45%), infrequent tinnitus in 3 (15%), and mild intermittent dysequilibrium in 1 (5%). No subjective symptom was believed to be bothersome just before the MVA.

New-onset hearing loss in 17 (85%) and nonpulsatile tinnitus in 17 (85%) were the most common symptoms

after airbag deployment. These occurred together on a statistically significant basis ($P < 0.0001$, Fisher's exact test). Of the 17 with subjective hearing loss, it was unilateral in 10 (59%), bilateral in 7 (41%), and persistent in 13 (76%). Tinnitus was unilateral in 11 (65%), bilateral or nonlocalized in 6 (35%), and persistent in 9 (53%). Other common symptoms in our 20 patients included dysequilibrium in 10 (50%) and aural fullness in 6 (30%). Dysequilibrium was temporary in 6 of 10 (60%), persistent in 4 (40%), and delayed in 1 (10%). Aural fullness was associated with occupant head position toward the airbag ($P = 0.039$, Fisher's exact test).

Objective hearing loss (drop of at least 10 dB at 2 frequencies) was documented in 21 of 24 (88%) subjectively affected ears. Of ears with objective hearing loss, the type was sensorineural (SNHL) in 17 (81%), conductive in 2 (10%), and mixed in 2 (10%). High frequencies (>4 kHz) were most commonly affected, followed by middle (2 to 4 kHz) and low frequencies (<2 kHz).

Seven subjects had undergone a hearing test before the MVA. The audiometric configuration grossly changed (ie, from normal to flat or from flat to sloping) in 5 of 9 ears (56%) that had hearing objectively documented before the MVA. The PTA increased significantly after airbag deployment in these 9 ears by 10 dB, both within the first month and 1 month after the accident. This average change was statistically significant for the early ($P < 0.001$, paired t test) and late ($P = 0.002$, paired t test) time periods. Of all tested variables, increased age ($P = 0.028$, Mann-Whitney test), and head position toward the airbag ($P = 0.027$, Fisher's exact test) were statistically related to hearing loss. No significant difference was found between post-MVA hearing levels based on time from the MVA ($P = 0.804$, paired t test).

Of the 10 patients with dizziness, 4 (40%) had caloric weakness, 2 (20%) had benign paroxysmal positional vertigo (BPPV), and 2 (20%) had endolymphatic hydrops. Two subjects (20%) had nonspecific dysequilibrium. Patients with BPPV were treated with a particle-repositioning maneuver. One patient with hydrops did not seek treatment until 2 months after the MVA. Medical therapy was effective in controlling hydrops symptoms. Four of the 10 (40%) with dizziness have chronic symptoms resistant to medical and vestibular therapy.

Three of 20 (15%) described chronic otalgia. Temporomandibular joint (TMJ) dysfunction was diagnosed in all, and it was bilateral in 1. No subject appeared to have this condition before airbag deployment. Pain has been mitigated with conservative treatment alone.

Unilateral tympanic membrane (TM) perforations were documented in 4 patients (20%). Two had mixed and 2 had conductive hearing losses. Three of the affected ears were directly oriented toward the deploying airbag in front. The other was a driver whose right TM was perforated after exposure to dual airbag inflation, despite a neutral head position. Ear orientation was the only statistically significant related variable for TM perforation ($P = 0.0004$, Fisher's exact test). Each patient required a tympanoplasty and showed hearing improvement after surgery.

DISCUSSION

A new assortment of injuries has been documented since the introduction of automobile airbags.[5,7-17] There are only 7 reports[13,14,18-22] of otologic injury caused by airbag deployment. These describe hearing loss, tinnitus, and/or dysequilibrium. The potential risk to the ear from airbag restraint devices appears to be quite low.[19]

A temporary or permanent threshold shift (TS) in hearing has been reported.[13,14,18-22] This study provides documentation of an additional 21 ears that were found to have objective hearing loss after airbag inflation. Most demonstrated SNHLs, but a conductive or mixed loss was noted in all ears with TM perforation. Patient 13 had a mixed hearing loss related to previous chronic otitis media. As reported previously,[23,24] traumatic TM perforation did not appear to protect the inner ear. This is suggested by a sensorineural component in 2 subjects with TM perforations. SNHLs were commonly documented in the mid- and high-frequency range in earlier reports.[13,14,19,20,22] Other airbag studies have shown 6 kHz to be the most common frequency, showing a temporary TS in human volunteers[25] and in cats.[26] Nearly all subjects with SNHL in this study had involvement of both the mid and high frequencies. These results are consistent with those of blast studies demonstrating no single typical audiometric configuration.[23,24]

Our subjects with SNHL generally showed no change in hearing levels after the initial post-MVA audiogram. This is in contrast to the belief that airbags are more likely to produce a temporary TS.[25-27] Increased age was a statistically significant variable associated with hearing loss in our study. Factors such as overall health, smoking, and microvascular disease may predispose to hearing loss.[28] Individual responses to noise and preexisting hearing losses may also affect hearing results in airbag studies.[19,25]

Tinnitus was frequently noted in this study. As previously described in the literature,[23-25,29] tinnitus tends to parallel SNHL. There are only a few known patients with airbag injuries who have persistent tinnitus.[13,14,18,19,22] This report documents an additional 9 with chronic subjective tinnitus. Less than half of those in our study reporting tinnitus noted its resolution.

Table 2. Subjective symptoms and test results

Patient no.	Pre-MVA symptoms	Post-MVA symptoms	Pre-MVA audiogram (PTA), configuration	Early post-MVA audiogram (PTA), configuration
1 (perf)*	HL	LHL, TINN, DIZ, FULL	20 dB (left), flat SNHL	None
2	HL, TINN	RHL, TINN	34 dB (right), sloping SNHL	45 dB (right), falling SNHL
3	HL	RHL, TINN, FULL	35 dB (right), sloping SNHL	46 dB (right), sloping SNHL
4 (perf)	None	LHL, DIZ	None	45 dB (left), flat CHL
5	RHL, TINN	RHL, LHL, TINN, DIZ	23 dB (left) & 45 dB (right), flat SNHL	33 dB (left) & 58 dB (right), flat SNHL
6	LHL	RHL, LHL, TINN	36 dB (left) & 32 dB (right), flat SNHL	40 dB (left) & 41 dB (right), sloping SNHL
7	RHL	RHL, LHL, TINN	None	None
8	None	DIZ	None	None
9	LHL, DIZ	LHL, DIZ, TINN	14 dB (left), sloping SNHL	None
10	None	LHL, RHL, TINN, DIZ, bilateral OTs	None	None
11	None	LHL, TINN, DIZ	None	None
12	None	LHL, RHL	None	39 dB (left) & 39 dB (right), sloping SNHL
13†	HL, TINN	LHL, TINN, DIZ, chronic OTs	11 dB (left), normal	21 dB (left), falling mixed loss
14	None	RHL, TINN, FULL	None	5 dB (right) & 5 dB (left), normal configuration
15	RHL, LHL	RHL, LHL, TINN, DIZ	None	None
16	None	TINN, DIZ, persistent OTs	None	None
17*	None	TINN	None	None
18 (perf)	None	RHL, TINN, FULL	None	35 dB (right), flat CHL
19 (perf)	None	RHL, TINN, FULL	None	45 dB (right), sloping mixed loss
20	None	RHL, LHL, TINN, FULL	None	None

perf, TM perforation; *CHL,* conductive hearing loss; *ENG,* electronystagmography; *ABR,* auditory brain stem response; *ECOG,* electrocochleography; *RHL,* right hearing loss; *LHL,* left hearing loss; *TINN,* tinnitus; *FULL,* fullness; *DIZ,* dizziness; *OT,* otalgia.

*History of left tympanoplasty for a persistent perforation.

†History of a left tympanomastoidectomy for chronic otitis.

Specific dizziness syndromes in our study resolved with nonsurgical therapy. No perilymphatic fistula was diagnosed in this report, but it can occur after minor head injury.[12,30] None of our patients had a significant head injury, although it is possible that drivers of vehicles sustaining a left-frontal impact may have struck the A-pillar between the windshield and driverside window. Stapes displacement with an intense pressure pulse might cause inner ear damage, including fistula formation or intralabyrinthine membrane rupture.[19]

Less common otologic symptoms in this series included aural fullness and otalgia. The fullness sensation resolved in all affected individuals and was usually associated with a TM perforation. Otalgia was always caused by TMJ dysfunction. This is consistent with prior documentation of airbag deployment being associated with TMJ damage.[11] No alternate source of ear pain was discovered.

Airbag-induced TM perforations have not been reported by other authors. The threshold for perforation of a normal TM is thought to be about 180 dB sound pressure level but can be as low as 160 dB with preexisting otologic disease.[12,31] Patient 1 had undergone prior tympanoplasty; this may have lowered the threshold. None of the 4 subjects with perforations demonstrated spontaneous healing. This is in contrast to the combat setting, in which resolution rates vary from 81% to 91%.[29] Larger perforations, those posteriorly located, and those that fail to show healing within a few weeks have a significantly lower rate of closure.[12,23,24,29] All perforations in our study were associated with these negative factors.

Our study suggests that ear positioning toward the airbag was an important factor with regard to hearing loss, aural fullness, and TM perforation. A feline study confirmed increased acoustic trauma associated with ear orientation toward the airbag. This held true for the right (inboard) ears of animals in the driver's seat, which were exposed directly to the passenger side airbag.[26] An ear facing a blast experiences a TM pressure that is approximately double that of an ear not facing the blast.[31] Some clinical cases with hearing loss

Late post-MVA audiogram (PTA), configuration	Frequency affected	Other post-MVA testing
28 dB (left), sloping mixed loss	High (left)	None
33 dB (right), sloping SNHL	Middle & high (right)	Metabolic screen normal
45 dB (right), sloping SNHL	Middle & high (right)	Head CT normal, metabolic screen normal
19 dB (left), flat CHL	All (left)	Head CT normal, head MRI normal
30 dB (left) & 57 dB (right), flat SNHL	Middle (both), high (both)	Head CT normal, head MRI normal, ENG normal
40 dB (left) & 51 dB (right), sloping SNHL	Middle & high (both)	Head CT normal, ABR normal, ECOG normal
25 dB (left) & 36 dB (right), notched SNHL	Middle (left), low (right), high (both)	Head CT normal, plain films negative
8 dB (both), normal	None	Head MRI normal, Hallpike positive (left)
24 dB (left), sloping SNHL	High (left)	ENG & MRI normal, ECOG abnormal, rotary chair abnormal
26 dB (left) & 15 dB (right), sloping SNHL	Middle (left), low (left), high (both)	Head CT normal, ENG normal, ECOG normal
29 dB (left) & 15 dB (right), notched SNHL	High (left)	Head CT normal, head MRI normal
46 dB (left) & 48 dB (right), sloping SNHL	All (both)	None
29 dB (left), falling mixed loss	All (left)	Head CT normal, head MRI normal
None	None	Plain films negative
17 dB (right) & 17 dB (left), falling SNHL bilaterally	Middle (both), high (both)	ENG: positional nystagmus, metabolic screen normal
5 dB (right) & 5 dB (left), normal	None	ENG normal, metabolic screen normal
None	None	None
18 dB (right), normal	All (right)	None
22 dB (right), sloping mixed loss	All (right)	None
23 dB (right) & 14 dB (left), sloping SNHL	High (right)	MRI normal

were associated with the head turned toward the airbag as well.[13,14,18-22] Ear orientation thus may be important with regard to acoustic damage.

Patients with TM perforations in our study had their ears oriented toward the inflating airbag directly in front (patients 1, 4, 19) or the passenger-side airbag (patient 18). The angle of incidence appeared to be important. Air trapping within the external auditory canal from airbag "slap" may also help to explain our observations. On the basis of results of this study, implementation of lateral (side) airbags in new vehicles may increase the chance of TM perforation and other otologic injuries.

Additional factors thought to be important with respect to TM perforation are vehicle interior size and geometry, occupant location, and number of airbags deployed. Engineering studies have documented an effect of vehicle size and window status on the pressure pulse associated with automobile airbag deployment.[32] A smaller effective interior volume and closed windows are expected to augment the potential for acoustic trauma. This does not, however, take into account the protective effect of the low-frequency component inflation noise. This component has actually been found to reduce the effectiveness of the high-frequency component in producing TS in human volunteers[27] and may modulate the flow of energy into the cochlea.[33] Surprisingly, having the windows open might be less protective based on mathematical modeling.[34] No subject with a perforation had the windows open, and none was in a compact car. Interior geometry variation is also known to affect the pressure environment and may contribute to otologic injury.[35]

Deployment of multiple airbags logically increases noise production. Although most perforation subjects were driving, it has been shown that passengers' TMs receive increased sound pressure when both airbags inflate.[32] Dual airbags did deploy in 2 TM perforation subjects; inflation of multiple airbags may have increased the overall pressure wave. Formation of a TM perforation therefore may be multifactorial, with many conditions playing a role.

Car size or type did not prove to be statistically sig-

nificant for any specific otologic injury in this report. Most vehicles were American products; this likely reflects our patient population from the state of Michigan rather than increased danger associated with airbags from US automakers. There is no reason to believe that American airbag restraint systems are not as safe as those produced by foreign car manufacturers.

There are a few limitations to this study. The small number of subjects from a select patient population may not be representative of all persons who sustain airbag-induced otologic injury. Many of the variables studied are subjective and thus difficult to quantify.

The reason that such a high percentage of our subjects had permanent or severe otologic damage may again be related to the nature of our referral population. It is thought that most MVA patients fail to report minor or temporary symptoms such as mild hearing loss or tinnitus. Because studies[25,27] have demonstrated that hearing levels recover within a few hours of airbag deployment, it is possible that most individuals are not bothered by transient symptoms. Hearing loss above the speech frequency range may not be immediately apparent or bothersome.[13] We tended to see only those with persistent symptoms who were eventually referred by local physicians.

The relative incidence of all otologic injuries caused by MVAs is unknown. Airbags may actually be protective for some injury categories. This report is the first to document certain airbag-associated otologic problems. Unilateral TM perforations, unilateral conductive and mixed hearing loss, bilateral SNHL, and vertigo caused by BPPV or endolymphatic hydrops have not been attributed to airbags previously. It should be noted, however, that many patterns of hearing loss have been reported after an MVA without airbag inflation.[36] Furthermore, BPPV and other vestibular abnormalities have been seen after various types of head injury, again unrelated to airbag deployment.[36] It is therefore difficult to establish a direct cause-and-effect relationship between airbags and some of the injuries encountered in this study.

CONCLUSIONS

A wide variety of airbag-induced otologic injuries occur and may have long-term sequelae. These include temporary or permanent hearing loss, tinnitus, dysequilibrium, and otalgia. Four TM perforations were documented, and each required surgical intervention. Ear orientation was found to be a statistically significant variable associated with hearing loss, aural fullness, and TM perforation. On the basis of results of this study, implementation of new lateral (side) airbags may increase the chance of these injuries as deployment conditions change.

It is important for health care personnel to be cognizant of these potential injuries because airbags are now ubiquitous. Recognition of injury patterns is a logical step toward airbag design modification. Refinements may reduce both the incidence and the severity of this new injury spectrum.

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