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Airbags and permanent auditory deficits. A real correlation ?

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Abstract. *Airbags and permanent auditory deficit. A real correlation ? Objectives* : To evaluate the relationship between airbag-induced noise and individual metabolic risk factors in determining persistent hypoacusia in drivers after road accidents.

Methodology : We selected 22 patients previously involved in a car accident with deployment of airbags. Patients underwent general and audiological clinical history, tonal audiometric examination, vocal audiometric examination, impedance meter examination and blood tests.

Results : We divided patients, according to audiometric data, into 2 groups : group A with no residual otological disturbances (6 subjects) and group B with persistent hypoacusia (16 subjects). Blood parameters were into physiological levels in all group A patients ; on the contrary 12 (subgroup B1) out of 16 group B patients had altered blood levels of glucose, urea and cholesterol, with mean values of 155.8 ± 38.6 mg/dl, 48.2 ± 8.3 mg/dl and 250.8 ± 28.1 mg/dl, respectively, revealing statistically significant differences in these parameters when compared with the other 4 hypoacusic cases (Sub-group B2) and with the normal subjects (Group A) ($p < 0.01$ for glucose, $p < 0.05$ for urea and $p < 0.001$ for cholesterol).

Conclusions : Our findings confirm the transitory otological damage due to airbag deployment : the intensity of the acoustic wave hitting the ear after airbag deployment is responsible for a temporary rise in the acoustic threshold but the persistence of an auditory deficit can be due to co-factors able to interfere with the acute acoustic trauma recovery processes through a metabolic, angiopathic, neuropathic or unknown mechanism. Moreover, also the age of the patients could affect in a significant way the recovery from the acoustic trauma.

Introduction

Car accidents are one of the principal causes of accidental deaths (1) which, for obvious reasons, mainly involve the younger and more active population of industrialized societies. Great importance, from both medical and economical viewpoint, is consequently attributed to all measures able to avoid the loss of human lives on the roads. In combination with safety belts, pneumatic devices known as airbags, which absorb the kinetic effects of impact, have improved the survival rate of drivers and passengers involved in road accidents by preventing the head and body from hitting the steering wheel, windscreen or dashboard. According to studies carried out in the United States (1, 2) the airbag avoided the fatal outcome of motor-car traumas in 21-22% of drivers who had not fastened their safety belts and 9-16% of those who had. A federal law made the installation of airbags on all motor vehicles (3) in the United States compulsory since 1 September 1998. Like any other "remedy", the airbag also has certain side effects. Its actual wide diffusion led to the emergence of a new spectrum of lesions caused by its deployment. In many cases, these can be considered "minor damages", which still amply justify its use. Unfortunately, more severe lesions are occasionally recorded, involving the facial skeleton, (4) ocular structures (5), facial nerve (6) or cervical cord (7, 8). Cause their anatomical location, the management of these lesions often involve the ENT specialist, not only where surgical correction is needed, but also for the evaluation of hearing problems and balance disturbances (9, 10, 11). The aim of the present work was to evaluate the relationship between airbag-induced noise and individual metabolic risk factors in determining persistent hypoacusia in drivers after road accidents.

Material and methods

We retrospectively selected for the present study 22 patients (19 males and 3 females) aged from 18 to 69 years (mean 34), who had been involved in a car accident that had triggered the deployment of the airbag, without suffering traumatic complications,

as documented by their clinical records and medical examination. Specifically clinical files attested a state of psychological agitation and average sensorineural hearing loss of 40 dB mainly on the 4 kHz without vertigo, documented by pure tone audiometry immediately after the accident. All the patients referred a normal audio-vestibular function before the car crash.

To rule out the effects of factors interfering with the interpretation of the fundamental data, the subjects were assessed for acoustic risk factors, such as exposure to noise for the professional risk categories, prolonged use of walkman and/or habitual attendance (at least once a week) of discotheques and use of ototoxic drugs.

All subjects were driving their cars at the time of the accident and declared that they were wearing their safety belts. All the cars had air-conditioning and were being driven with the windows closed.

Subjects underwent the following protocol : general and audiological clinical history, tonal audiometric examination using Amplaid 309, 315 and 420 appliances, vocal audiometric examination, impedance meter examination using Amplaid 720 and 728 appliances, blood tests (blood glucose, urea and cholesterol levels). Informed written consent was signed by all the patients. All the measured data underwent a statistical analysis by using Student T-test.

Results

Patients demographic data and basic information on the accident modalities are summarized in table 1. None of the 22 people enrolled complained of balance disturbances.

Table 1
Subjects enrolled and accident modalities

Patient	Sex	Age	Accident	Type of airbag
AM	m	34	F	D
FP	m	25	F	D + P
VD	m	18	FL	D + P
FG	m	24	FL	D + P
MM	f	69	FL	D + S
AG	m	20	F	D
EF	m	20	FL	D
EZ	m	27	FL	D
AF	m	55	F	D
GP	f	30	FL	D
GR	m	30	F	D + P
AR	m	50	F	D
AP	m	44	F	D
PR	m	21	FL	D + P + S
SA	m	19	F	D
VT	m	49	F	D
AA	m	54	F	D
VS	m	24	F	D
SS	m	43	F	D + P
ST	m	24	FL	D
FS	f	30	F	D
KD	m	32	F	D

m: male; f: female; F: frontal, FL: frontolateral; D: driver; P: passenger; S: lateral airbags.

According to audiometric data, we divided patients into 2 groups : group A with no residual otological disturbances (6 subjects) and group B with persistent hypoacusia (16 subjects). Specifically, group B patients resulted affected by a bilateral neurosensory hypoacusia with a mean sensorineural loss at 4 kHz of 45.6 ± 5.1 dB for the left ear and of 46.9 ± 5.7 dB for the right one (see table 2 for details). We didn't measure any statistically significant differences between the left and right ears hearing losses, in these patients, for all the tested frequencies. We further divided group B patients according to the abnormality (subgroup B1 : 12 patients) or normality (subgroup B2 : 4 patients) of blood parameters. These two subgroups didn't statistically differ for what concern the severity of the hearing losses.

Blood parameters were into physiological levels in all group A patients (Table 3) ; specifically, mean glucose, urea and cholesterol values in this group were 85.8 ± 9.2 mg/dl, 34.2 ± 4.2 mg/dl and 94.2 ± 14.3 mg/dl, respectively (Glucose, urea and cholesterol normal values : 65-110 mg/dl, 10-40 mg/dl, < 240 mg/dl, respectively).

12 (subgroup B1) out of 16 group B patients showed altered blood levels of glucose, urea and cholesterol (table 4), with mean values of 155.8 ± 38.6 mg/dl, 48.2 ± 8.3 mg/dl and 250.8 ± 28.1 mg/dl, respectively. On the contrary 4 out of 16 patients with persistent hearing losses, had blood parameters within the physiologic values. The statistical analysis of these data showed that blood parameters of subgroup B1 patients differed in a significant way from those of both group A and subgroup B2 patients ($p < 0.01$ for glucose, $p < 0.05$ for urea and $p < 0.001$ for cholesterol).

Table 2

Group B Audiometric data

Patients	3 kHz		4 kHz		5 kHz	
	Left ear	Right ear	Left ear	Right ear	Left ear	Right ear
AF	30 dB	35 dB	40 dB	45 dB	20 dB	25 dB
MM	35 dB	35 dB	45 dB	45 dB	10 dB	10 dB
AP	30 dB	40 dB	50 dB	55 dB	15 dB	20 dB
AR	45 dB	40 dB	55 dB	50 dB	15 dB	5 dB
AA	20 dB	20 dB	45 dB	45 dB	20 dB	20 dB
VT	30 dB	30 dB	45 dB	45 dB	30 dB	30 dB
GP	30 dB	35 dB	45 dB	55 dB	35 dB	40 dB
AM	30 dB	30 dB	40 dB	50 dB	20 dB	25 dB
FS	35 dB	30 dB	50 dB	40 dB	25 dB	20 dB
KD	45 dB	40 dB	45 dB	40 dB	15 dB	10 dB
GR	35 dB	35 dB	40 dB	40 dB	5 dB	5 dB
SS	35 dB	35 dB	55 dB	55 dB	5 dB	5 dB
FP	35 dB	35 dB	40 dB	40 dB	25 dB	25 dB
EZ	30 dB	35 dB	40 dB	45 dB	25 dB	30 dB
FG	35 dB	30 dB	50 dB	45 dB	20 dB	20 dB
AG	35 dB	45 dB	45 dB	55 dB	30 dB	35 dB
Mean ± SD	33.4 ± 5.8 dB	34.4 ± 5.7 dB	45.6 ± 5.1 dB	46.9 ± 5.7 dB	19.7 ± 8.6 dB	20.3 ± 10.9 dB

Data indicate the hearing loss, measured in decibel, at 3, 4 and 5 kHz for left and right ears.

Table 3

Blood parameters of group A patients

Patients	Age (years)	Glucose (mg/dl)	Urea (mg/dl)	Cholesterol (mg/dl)
EF	20	80	40	80
VD	18	75	35	90
SA	19	90	30	120
PR	21	100	32	100
ST	24	90	38	85
VS	24	80	30	90
Means ± SD	21 ± 2.5	85.8 ± 9.2	34.2 ± 4.2	94.2 ± 14.3

Glucose normal values : 65-110 mg/dl ; Urea normal values : 10-40 mg/dl ; Cholesterol normal values : < 240 mg/dl.

At last, Sub-group B1 patients were significantly older (mean age : 43.3 ± 12.5 years) than sub-group B2 or group A patients (mean age : 24 ± 2.9 years and 21 ± 2.5 years respectively) ($p < 0.01$).

Discussion

Among the potential negative effects of the airbag's deployment, otological damage has been more and more frequently described in the literature (12). Though relatively limited, it deserves attention on account of the potential permanent rise in the auditory threshold (13). In fact, in addition to the transmissive hearing loss attributable to perforation of the tympanic membrane, sensorineural hearing loss has also been described. The combination of the two types of damage suggests that tympanic rupture fails to protect the inner ear, as reported in a number of studies (3, 14). The high and medium frequencies are the more frequently impaired, as confirmed by experimental research on animals (15) and volunteers (16).

According to surveys carried out by the motorcar industries, airbag deployment noise is of high intensity and short duration, with peaks even exceeding 170 dB. As expected, the inflation of double or multiple airbags proportionally increases the risk of acoustic damage with the increase in the pressure wave (17, 18). However, the recording of otological consequences is relatively recent and infrequent, probably because there is only a temporary rise in hearing threshold in most cases, while a permanent auditory deficit is rare (12).

Although the protection offered by the airbag is far greater than the risks of side effects, in the United States, where it is now compulsory on all motor-cars, people have been demanding devices able to disconnect the airbag if its presence is considered inappropriate. This led to a more careful and scientific assessment and, in July 1997, precise indications on the

Table 4
Blood parameters of group B patients

	Patients	Age (years)	Glucose (mg/dl)	Urea (mg/dl)	Cholesterol (mg/dl)
Sub-group B1	AF	55	140	50	260
	MM	69	200	55	280
	AP	44	140	50	280
	AR	50	160	25	300
	AA	54	120	50	250
	VT	49	120	45	260
	GP	30	130	48	250
	AM	34	200	50	240
	FS	30	240	50	220
	KD	32	120	45	210
	GR	30	120	50	210
	SS	43	180	60	250
Sub-group B2	FP	25	80	40	70
	EZ	27	100	35	120
	FG	24	110	40	160
	AG	20	80	28	140
Means ± SD					
Sub-group B1		43.3 ± 12.5	155.8 ± 38.6	48.2 ± 8.3	250.8 ± 28.1
Sub-group B2		24 ± 2.9	92.5 ± 15	35.7 ± 5.7	122.5 ± 38.6

Glucose normal values : 65-110 mg/dl ; Urea normal values : 10-40 mg/dl ; Cholesterol normal values :< 240 mg/dl.

advisability of disconnecting the airbag under certain circumstances emerged from the National Conference on Medical Indications for Airbag Disconnection : given its undeniable capacity to save lives in potentially fatal accidents, the airbag's disconnection is not recommended in relation to potential audiological damage, both in normal hearers and in people already suffering from hypoacusia or tinnitus (19).

In fact, although airbag deployment produces a noise nearing 170 dB, according to the conclusions of the Conference, any hypoacusia, because of the difficulty in isolating the effect of the airbag from the other noise produced by the accident, cannot be automatically attributed to this instrument. In our sample, the clinical histories and the finding of a sensorineural loss after the trauma with dips at 4 kHz were suggestive of an acoustic lesion, but none of the patients showed laceration of the tympanic membrane, ossicular dislocation, or labyrinth rupture with perilymphatic fistulas.

According to our data, the hypoacusia tends to be temporary and reversible in persons in good metabolic condition (group A patients). On the contrary, a significant proportion of patients with persisted hearing loss (Sub-group B1) showed altered metabolic pictures. In a smaller proportion of cases (4 patients, Sub-Group B2), the hearing loss was irreversible despite a good metabolic state ; in these cases an accentuated individual susceptibility may be involved. Moreover, also the age of the patients could affect in a significant way the recovery from the acoustic trauma.

Conclusions

Our findings confirm the transitory otological damage due to airbag deployment : the airbag-related noise causes a hearing loss that could become irreversible in some cases.

The intensity of the acoustic wave hitting the ear after airbag deployment is responsible for a temporary rise in the acoustic threshold, but the persistence of an auditory deficit can be due to co-factors able to interfere with the acute acoustic trauma recovery processes through a metabolic, angiopathic, neuropathic or unknown mechanism.

In conclusion, remaining the airbag an indispensable protective aid, audiometric tests have to be performed in all the subjects involved in accidents triggering the inflation of the airbag and, in patients suffering for airbag-related transient hearing loss, co-factors that may contribute to make the auditory deficit permanent should be promptly investigated and, if necessary, corrected.

References

1. Zador P. L., Ciccone A. Automobile driver fatalities in frontal impacts : air bags compared with manual belts. *Am. J. Public Health*, **83** : 661-666, 1993.
2. Lund A. K., Ferguson S. A. Driver fatalities in 1985-1993 cars with air bags. *J. Trauma*, **38** : 469-475, 1995.
3. McFeely W. J., Bojrab D. I., Devis K. G., Hegyi D. F. Otologic injuries secondary to air bag deployment. American Academy of Otolaryngology Head and Neck Surgery Foundation Annual Meeting. San Antonio (TX) September 13-16, 1998.
4. Scott I. U., John G. R., Stark W. J. Air bag-associated ocular injury and periorbital fractures. *Arch. Ophthalmol.*, **111** : 25, 1993.
5. Blacksin M. F. Patterns of fracture after air bag deployment. *J. Trauma*, **35** : 840-843, 1993.

6. Bedell J. R., Malik V. Facial nerve paresis involving passenger airbag deployment : a case report. *J. Emerg. Med.*, **15** : 475-476, 1997.
7. Maxeiner H., Hahn M. Airbag-induced lethal cervical trauma. *J. Trauma*, **42** : 1148-1151, 1997.
8. Giguere J. F., St-Vil D., Turmel A., Di Lorenzo M., Pothel C., Manseau S., Mercier C. Airbags and children : a spectrum of C-spine injuries. *J. Pediatr. Surg.*, **33** : 811-816, 1998.
9. Morris M. S., Borja L. P. Air bag deployment and hearing loss. *Arch. Otolaryngol. Head Neck Surg.*, **124** : 507, 1998.
10. Morris M. S., Borja L. P. Noise levels associated with airbag deployment may result in occupants experiencing irreversible hearing loss. *J. Trauma*, **44** : 238-239, 1998.
11. Kramer M. B., Shattuck T. G., Charnock D. R. Traumatic hearing loss following airbag inflation . *N. Engl. J. Med.*, **337** : 574-575, 1997.
12. Saunders J. E., Slattery W. H. 3rd, Luxford W. M. Automobile airbag impulse noise : otologic symptoms in six patients. *Otolaryngol. Head Neck Surg.*, **118** : 228-234, 1998.
13. Beckerman B., Elberger S. Air bag ear. *Ann. Emerg. Med.*, **20** : 831-832, 1991.
14. Chait R. H., Casler J., Zajtchuk J. T. Blast injury of the ear : historical perspective. *Ann. Otol. Rhinol. Laryngol.*, Suppl. **140** : 9-12, 1989.
15. Price G. R., Rouhana S. W., Kalb J. T. Hearing hazard from the noise of airbag deployment. Acoustical Society of America. *J. Acoust. Soc. Am.*, **99** : 2464, 1996.
16. Nixon C. W. Human auditory response to an airbag inflation noise. Wright Patterson Air Force Base, Ohio : Biological Acoustics Branch, 6570th Aerospace Medical Research Laboratory, 1969.
17. Rouhana S. W., Webb S. R., Wooley R. G. *et al.* Investigation into the noise associated with airbag deployment : Part 1-Measurement technique and parameter study. Proc from 38th Stapp Car Crash Conference, Fort Lauderdale, Florida, p. 191-212, 1994.
18. Price G. R. Airbag noise hazard examined with mathematical model of the human ear. *J. Acoust. Soc. Am.*, **102** : 3201, 1997.
19. National conference on medical indication for airbag disconnection. *Ann. Emerg. Med.*, **31** (6) : 782-3, 1998.

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