

Distr.
GENERAL

TRANS/WP.29/GRSP/2000/11
15 September 2000

Original: ENGLISH

ECONOMIC COMMISSION FOR EUROPE

INLAND TRANSPORT COMMITTEE

World Forum for Harmonization of Vehicle Regulations (WP.29)

Working Party on Passive Safety (GRSP)

(Twenty-eighth session, 27 November-1 December 2000,
agenda item 1.)

SOUND EXPOSURE LIMIT FOR AIRBAG DEPLOYMENT

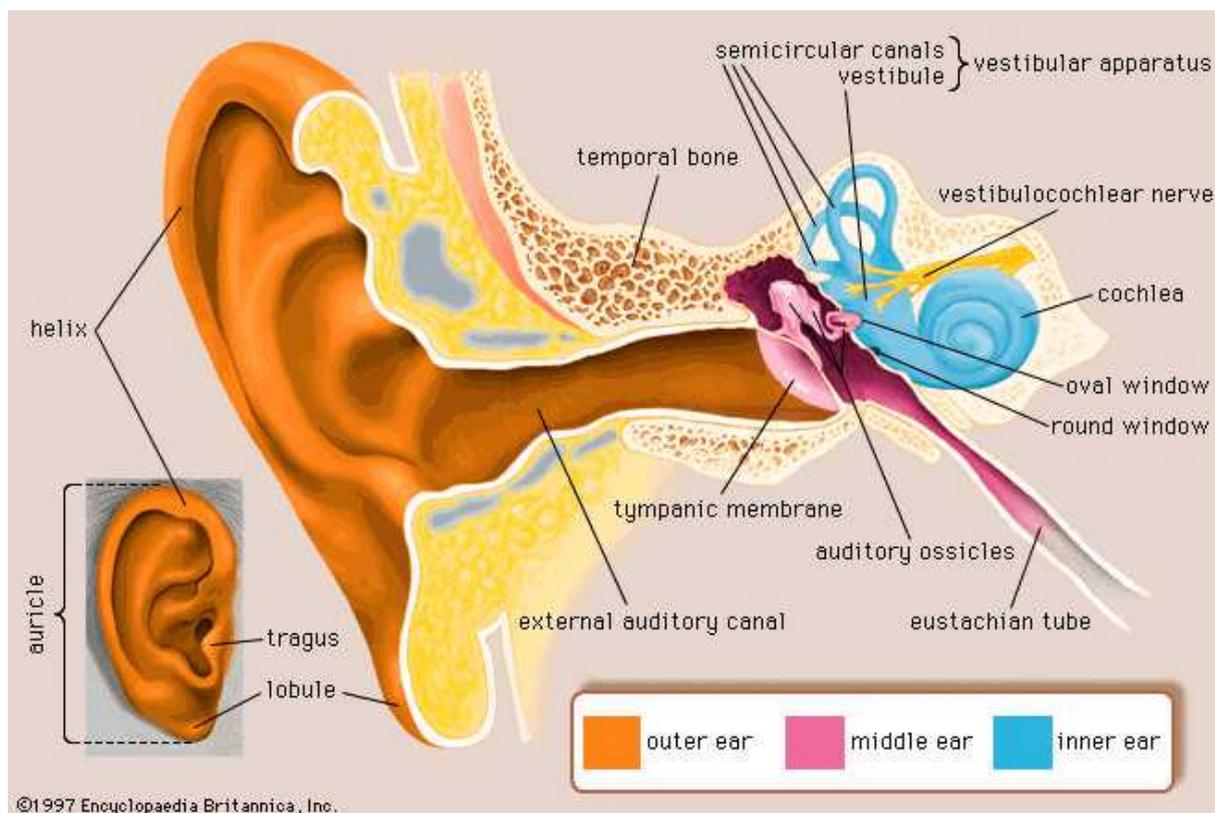
Transmitted by the Expert from France

Note: The text reproduced below was prepared by the expert from France in order to give an explanation of the hearing principle which could help to take a decision by GRSP concerning the measures to be taken to avoid ear injuries caused by the airbag deployment. It is based on a document distributed without a symbol (informal document No. 10) during the twenty-seventh session (TRANS/WP.29/GRSP/27, para. 25).

Note: This document is distributed to the Experts on Passive Safety only.

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Anatomy of the ear [1] and [2]



The human ear is divisible into three parts - the external ear, the middle ear or tympanum, and the internal ear or labyrinth.

The external ear consists of an expanded portion named pinna or auricle, and the auditory canal or meatus. The former serves to collect the vibrations of the air by which sound is produced; the latter conducts those vibrations to the tympanum. The auditory canal is about 25 mm in length. The membrana tympani occupies the termination of the auditory canal.

The middle ear is an irregular cavity filled with air and which communicates with the nasopharynx by the Eustachian tube. The middle ear is traversed by a chain of movable bones, which connect the membrana tympani with the internal ear, and serve to convey the vibrations communicated to the membrana tympani across the cavity of the tympanum to the internal ear. The ossicles of the middle ear are the malleus, the incus and the stapes. They are connected with each other and with the walls of the tympanum by ligaments, and moved by small muscles. The ligaments connecting the ossicles with the walls of the tympanum are 5 in number - 3 for the malleus, 1 for the incus and 1 for the stapes. The muscles of the tympanum are 2: tensor tympani and stapedius. The tensor tympani draws the membrana tympani inward and thus heightens its tension. The stapedius draws the head of the stapes backward, and thus causes the base of the bone to rotate on a vertical axis. It probably compresses the contents of the vestibule.

The internal ear is the essential part of the organ of hearing, receiving the ultimate distribution of the auditory nerve. It is called the labyrinth from the complexity of its shape, and consists of 2 parts: the osseous labyrinth, a series of cavities channelled out of the substance of the petrous bone, and the membranous labyrinth, the latter being contained within the former. The osseous labyrinth consists of 3 parts: the vestibule, semicircular canals, and cochlea. These cavities contain a clear fluid, perilymph, in which the membranous labyrinth is situated. The vestibule is the common central cavity of communication between the parts of the internal ear. The semicircular canals are three bony canals situated above and behind the vestibule. The cochlea bears some resemblance to a common snail-shell: it forms the anterior part of the labyrinth, is conical in form, and placed almost horizontally in front of the vestibule.

Hearing principle: the middle ear transforms the vibrations of the air into mechanical motion of the bones via the membrana typani and serves as an impedance matching device to improve the flow of energy into the cochlea. The stapes is held in the oval window of the cochlea by the annular ligament so that whenever the stapes vibrates, a pressure wave characteristic of the vibration is set up in the fluid. As the pressure wave travels along inside the cochlea, the basilar membrane responds with a wavelike oscillation. Several thousand cells, called hair cells, are found within the organ of Corti which rests on the basilar membrane. When these cells are distorted by the wave motion they cause the auditory nerve to which they are connected to send signals to the brain where they are interpreted as sounds. The basilar membrane's physical properties are smoothly graded and cause it to act like a frequency analyzer with the result that hair cells near the base of the cochlea (close to the oval window) respond to the high frequencies and those at the apex of the cochlea respond to the low frequencies.

Epidemiology

In 1995, Saunders, Slattery and Luxford [3] presented 6 cases of noise-induced hearing loss associated with airbag deployments.

Buckley, Setchfield and Frampton (feb 1999) [4], write that apart from their own report of 2 cases of possible noise trauma, they are aware of only 1 paper reporting that airbag inflation might induce hearing loss [3].

Moreover, SSM Hussein [5], has emphasized that in the 2 cases reported in [4], there is no mention of the time from the accident to when the audiometry was done. On top of that, in case 1 the accident took place in the United States while the post accident audiometry was performed in the United Kingdom, so the patient had a flight from the United States to the United Kingdom. In case 2, the patient already had high frequency hearing loss noted 18 months prior to the accident. So it is unclear if the very small additional loss in the high frequencies was related to the trauma. This patient had treatment for hyperlipidaemia and was 68 years old.

The rarity of similar articles seems to imply that risk of injury is scarce or that injury is occult.

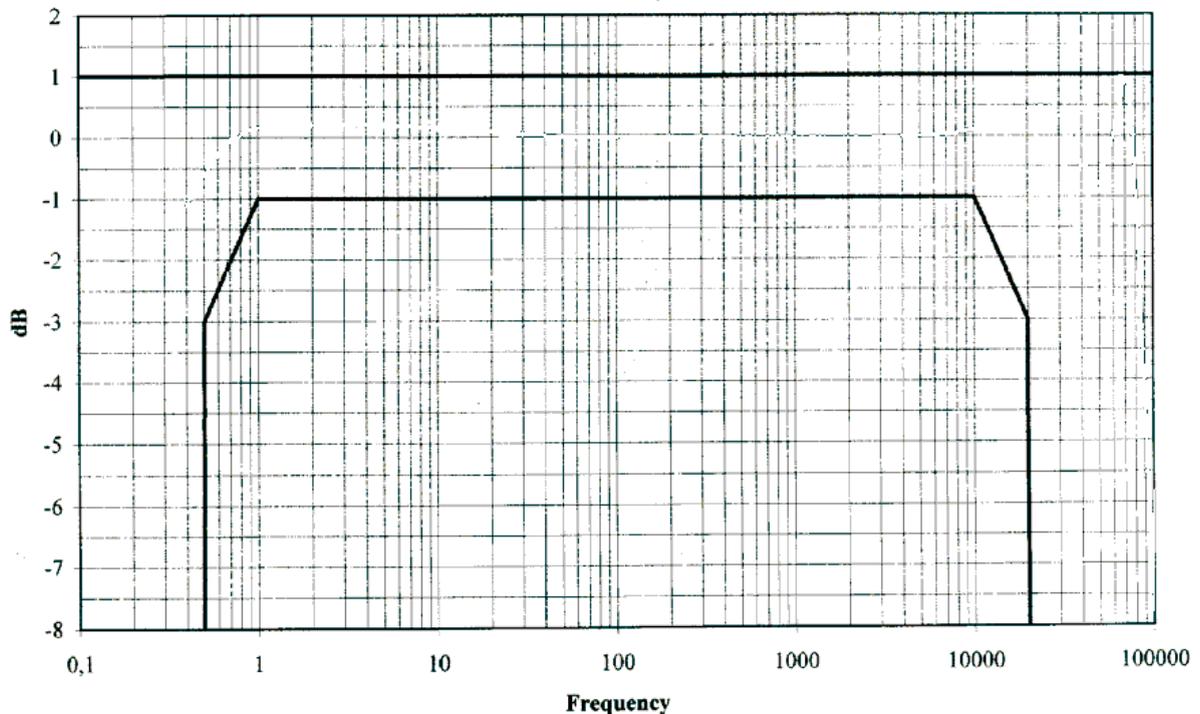
Laboratory data acquisition methods

According to SAE J247, the microphone and preamplifier responses for measuring acoustic impulses within vehicles should meet the requirement illustrated in the graph below.

In order to achieve this large bandwidth, both microphones and pressure transducers are used. Indeed, microphones are more sensitive in high frequencies and pressure transducers in low frequencies. However, new microphones (B&K4938) are within the corridor and are used alone.

The ear range of frequency is well known to extend from 20 Hz to 20,000 Hz and, as a consequence, it is necessary to have a sample rate of 50 kHz.

Microphone and Preamplifier Response for measuring acoustic impulses within vehicles - SAE J247 FEB87



Ear injury mechanisms

Three ear injury mechanisms are usually under consideration:

Blow to the Head: gross mechanical failure of structures

Continuous Exposure to Irritation: long duration (hours, weeks, months) exposures to loud noises. The site of physiological changes is the cells within the cochlea. The mechanism appears to be biochemical or physico-chemical.

Impulse Irritation: mechanical or physical trauma to the cellular structures of the cochlea.

Main already existing criteria [7]

Efforts to develop damage risk criteria for noise induced hearing loss began approximately in 1930. However, researchers mainly focused on continuous noise. In particular, LAeq was developed for factory measurements. Most of the work on impulse noise risk criteria has started since 1960.

1968: The WG17 of the Committee on Hearing and BioAcoustics (CHABA) of the National Academy of Sciences-National Research Council proposed a criteria based on A and B durations which are respectively the low frequency and high frequency pulse durations and the associated pressure peaks. This impulsive criterion was developed for military purposes with exposures expected to occur 100 times daily for many years.

1971: Under a contract from NHTSA, Bolt, Beranek, and Newman (BBN) revised earlier impulse noise assessment criteria to address specifically impulse noise produced by airbag deployment.

1992: based on 20 years of additional data, the National Academy of Sciences reports that the BBN curves are not appropriate for low-frequency impulses such as airbag impulse noise.

1998: Auditory Damage Unit (ADU) Criterion: US Army Research Laboratory (ARL) Model produces a "hazard" function, that estimates that there is an increasing risk of TTS (Temporary Threshold Shift) or PTS (Permanent Threshold Shift - which is a TTS that lasts longer than about 6 months) above 500 ADU. The model is primarily based on a detailed cat ear and aims to assess the mechanical fatigue of the basilar membrane. The model was validated using groups of feline ears in a dozen different experiments. Based on these tests, the correlation between ADU and CTS was $r=0.94$.

Discussion

Crash noise with no airbags was measured in a vehicle at 142 dB

Relationship between TTS and PTS is not clear but it seems that PTS can occur for low TTS.

In 1973, Sommer and Nixon [8] performed volunteer tests with the following results:

No TTS were observed for 165 dB at low frequency

Highest TTS (3dB) were observed for 153 dB at high frequency

Small TTS (1 dB) were observed for 165 dB at mixed frequency

These results are not consistent with BBN or SPL (Sound Pressure Level) based method prediction, but are in agreement with ADU criteria. This can be explained by the behaviour of the ear: the low frequency noise displaces the stapes and bottoms out the annular ligament which is not linear at 30 micrometers. Consequently, high frequencies are clipped and the ear is protected.

It is then necessary to measure properly the complete frequency range, in particular low frequencies should not be missed.

The following hypotheses are to be validated:

The injury mechanism is fatigue

Felidae hearing is similar to the most sensitive humans

Conclusion

Use of ear model is necessary

Measurement method must be specified

The definition of a criteria is premature

References

- [1] Gray's Anatomy , p849-842.
 - [2] Encyclopaedia Britannica 1997.
 - [3] Saunders JE, Slatery WH, Luxford WM « Automobile airbag impulse noise: otologic symptoms in six patients » Otolaryngol Head Neck Surg 1998; 118: 228-31.
 - [4] Buckley G, Setchfield N, Frampton R. « Two case reports of possible noise trauma after inflation of air bags in low speed car crashes » BMJ 1999 ;318: 499-500 (Feb 20).
 - [5] Hussein: response to doc [4] in BMJ
 - [6] Rouhana SW, Webb SR, Wooley RG, McCleary JD, Wood FD, Salva DB « Investigation into the noise associated with air bag deployment: part I - Measurement Technique and parameter study »
 - [7] Rouhana SW, Vaundle CD, Webb SR « investigation into the noise associated with airbag development: part II - Injury risk study using a mathematical model of the human ear » SAE paper 983132.
 - [8] Sommer H.C. and Nixon C.W., « Primary Components of Simulated Airbag Noise and Their Relative Effects on Human Hearing », Final Report, DOT Contratct Number IA-0-1-2160, 1973.
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