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Effect of noise exposure on gap detection in rats

N. Rybalko *, J. Syka

Institute of Experimental Medicine, Academy of Sciences of the Czech Republic, Vídeňská 1083, 142 20 Prague 4, Czech Republic

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Abstract

The effects of intense (110–120 dB) noise exposure (broadband noise for one hour) on temporal resolution was estimated in rats by measuring the behavioural gap detection threshold (GDT). Changes in GDT after 120 dB noise exposure were compared with changes in the threshold and amplitude of middle latency responses (MLR) recorded in response to tone stimuli. GDT values increased from 1.6 to 4.3 or 7.8 ms after exposure to 110 or 115 dB SPL, respectively; GDT recovered to pre-exposure values in 3–7 days. Three main types of noise-induced changes were observed after 120 dB SPL exposure: (I) GDT changes similar to those following noise exposure to 115 dB SPL and maximal hearing threshold shifts (TSs) at high frequencies of about 45 dB; (II) more pronounced changes in GDT (up to 60 ms) with maximal hearing threshold shifts of about 65 dB and (III) a lack of reliable responses to gap during the first weeks post-exposure with maximal hearing threshold shifts of about 80 dB. An increased GDT was present two months after noise exposure in animals with types II and III post-exposure changes; enhanced MLR amplitudes were also found in most of these in the first post-exposure week. The pronounced deficit in gap detection in some rats after 120 dB SPL noise exposure may signal the presence of a noise-induced tinnitus.

Keywords: Noise exposure; Temporal resolution; Gap detection; Hearing loss; Tinnitus; Rat

1. Introduction

The temporal resolution of the auditory system has an important role in the processing of acoustical signals. The measurement of gap detection threshold (GDT) has been used successfully in clinical practice and in animal experiments for studying auditory temporal resolution. Information about the normative parameters of gap detection is important for revealing temporal resolution pathology and for estimating its severity. On the other hand, the study of impaired gap detection (in clinical cases or in experimentally produced impairments in animals) allows us to obtain additional information for understanding temporal resolution mechanisms. Numerous experiments have shown that gap detection thresholds are dependent on the frequency spectrum and intensity of the continuous noise in which the gaps are embedded (Penner, 1977; Giraudi et al., 1980; Shailer and Moore, 1983; Fitzgibbons, 1983; Snell et al., 1994). Minimal GDTs in humans were recorded when the noise signal encompassing the gap was presented at 30–40 dB SL and included frequencies above 5–6 kHz. GDT in this case amounted to about 2–3 ms (Plomp, 1964; Fitzgibbons, 1983). The results of behavioural tests of gap detection abilities in experimental animals have shown that GDT is 2 ms in CBA mice (Walton et al., 1997), ranges from 2.6 to 3 ms in chinchillas (Giraudi et al., 1980; Salvi and Arehole, 1985), amounts to about 10 ms in ferrets (Kelly et al., 1996)

Abbreviations: GDT, gap detection threshold; MLR, middle latency response; SPL, sound pressure level; SL, sensation level

* Corresponding author. Tel.: +42 02 4106 26 89; fax: +42 02 4106 27 87.
E-mail address: rybalko@biomed.cas.cz (N. Rybalko).

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and reaches 2.5 ms in two bird species (budgerigars and zebra finches) (Okanoya and Dooling, 1990). The lowest GDT for rats, measured under optimal stimulus conditions (in a broadband noise at an intensity above 40 dB SPL) by an operant conditioning method, fluctuated around 1.6 ms (Syka et al., 2002). These values were slightly smaller than those obtained in other investigations using a startle amplitude reduction paradigm (Ison, 1982; Leitner et al., 1993).

It has been reported that subjects with a sensorineural hearing loss have reduced auditory temporal resolution and increased gap detection thresholds (Irwin et al., 1981; Fitzgibbons and Wightman, 1982; Tyler et al., 1982; Florentine and Buus, 1984; Glasberg et al., 1987). These data were supported by the prolongation of GDT in animals after noise exposure (Giraudi-Perry et al., 1982; Salvi and Arehole, 1985).

The worsening of gap detection after acoustic trauma may be due to the presence of tinnitus (Heffner and Harrington, 2002). Heffner and Harrington (2002) have shown that exposure to a loud sound produced a tinnitus in hamsters. The behavioural procedure for detecting the tinnitus consisted of recording the animal's conditional response to the presence of a silent interval in continuous noise, i.e., the procedure was similar to that used to estimate GDT. The idea was that animals with tinnitus would not respond to silent intervals because they would hear their tinnitus instead. The spectral character of the tinnitus could be similar to the spectral content of the carrier noise, and thus the tinnitus could, to a greater or lesser extent, mask the pause in the carrier noise and worsen the animal's performance.

It is known that noise exposure produces disorders not only in the peripheral but also in the central part of the auditory system. Elevated hearing thresholds after acoustic trauma are, to a great extent, related to damage of the inner ear. The enhanced amplitudes of cortical evoked responses, observed in experimental animals after noise exposure, indicate pathological changes at the higher levels of the auditory system including the auditory cortex (Popelář et al., 1987; Syka and Rybalko, 2000). Since gap detection is to some extent dependent on the integrity of the auditory cortex (Kelly et al., 1996; Syka et al., 2002), the impairment of cortical function after acoustic trauma could have effect on gap detection ability.

The aim of the present experiments was to study the effects of intense noise exposure on temporal resolution in rats, estimated by measuring behavioural GDT. For assessing the contributions of the peripheral and central parts of the auditory system to noise-induced impairment of temporal resolution, changes in GDT were compared with changes in the threshold and amplitude of middle latency responses (MLR) recorded in response to tone stimuli.

2. Materials and methods

2.1. Subjects

Behavioural GDTs were measured in 17 adult (8–13 months old) pigmented female rats (strain Long–Evans) before and after a 1-h noise exposure at an intensity of 110 (3 rats), 115 (3 rats) or 120 dB SPL (11 rats). The rats were housed in a plexiglass cage (2–3 rats per cage). GDTs were tested using an operant conditioning procedure with food reinforcement; therefore the rats had free access to water but were restricted in food intake. Twenty-three hours before training or testing session, the rats were completely deprived of food. In rats exposed to 120 dB SPL, in parallel with the testing of behavioural GDT, MLR to tone stimuli were recorded. MLR thresholds were used for estimating hearing sensitivity in the animals. The assessment of hearing sensitivity by electrophysiological methods (recording auditory brainstem responses and MLR) in humans and in animals with normal hearing or with hearing loss proved to be in good agreement with the assessments obtained with behavioural techniques (Davis, 1965; Pratt and Sohmer, 1978; Suzuki et al., 1981; Borg, 1982; Henderson et al., 1983). Post-exposure changes in GDT and the threshold and amplitude of MLR were monitored for two months.

2.2. Behavioural apparatus and procedure

GDT measurements were performed in an anechoic sound-proof room. The test apparatus, training and testing procedures were described in detail previously (Syka et al., 2002). Food-deprived animals were trained to detect the presence of gaps in a continuous noise. The experiments were conducted in a grid test box (size $20 \times 20 \times 20$ cm) with two levers (starting and responding) and a foodcup connected with a food dispenser. Gap carrier noise was presented from a loudspeaker located outside the experimental box, 50-cm in front of the wall with the levers. The continuous noise was switched on before the rat was put in the test box.

The experiment was designed so that 0.5–5 s after pressing the starting lever, a test stimulus consisting of five gaps with a constant duration was triggered. Pressing the responding lever during the time when the gaps were presented or up to one second after the last gap, was scored as a hit reaction and the rat was rewarded with a pellet. For estimating the degree of conjunction of the rat's response with the test stimulus (stimulus control, Hendricks, 1966; Kelly and Masterton, 1977) in 33% of the trials in a session, pressing the starting lever did not result in the appearance of the test stimulus. These were so-called "catch trials". A rat's response during a catch trial was classified as a false alarm. False alarms allowed us to estimate the number of accidental responses among all hit