韓國感性科學會誌
Korean Journal of The Science of Emotion & Sensibility
2000. Vol.3. No.1. 37-42

Autonomic, Respiratory and Subjective Effects of Long-term Exposure to Aversive Loud Noise : Tonic Effects in Accumulated Stress Model *

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Abstracts Long-term exposure to loud noise affects performance since it changes arousal level, distracts attention, and also is able to evoke subjective stress accompanied by negative emotional states. The purpose of the study was to analyze dynamics of subjective and physiological variables during a relatively long-lasting (30 min) exposure to white noise (85 dB[A]). Physiological signals were recorded on 15 college students during 30 min of intense auditory stimulation. Autonomic variables, namely skin conductance level , non-specific SCR number, inter-beat intervals in ECG , heart rate variability index (HF/LF ratio of HRV), skin temperature, as well as respiration rate were analyzed on 5 min epoch basis. Psychological assessment (subjective rating of stress level) was also repeated every 5 min. Statistical analysis was employed to trace the time course of the dynamics of subjective and autonomic physiological variables and their relationships. Results showed that the intense noise evoked subjective stress as well as associated autonomic nervous system responses. However it was shown that physiological variables endured specific changes in the process of exposure to the loud white noise. Discussed were probable psychophysiological mechanisms mediating reactivity to long-term auditory stimulation of high intensity, namely short-term activation, followed by transient adaptation (with relatively stable autonomic balance) and then a subsequent wave of arousal due to tonic sympathetic dominance.

Introduction

Long-term exposure to intense noise is stressful to people and is usually accompanied by annoyance and loweres performance on ongoing tasks (Boucsein, Ottman, 1996; Kryter, 1970; Loeb, 1986; Miller, 1974; Poulton, 1979). Studies on nonauditory physiological concomitants of noise were reported by many research groups (Cohen, 1980; Jennings, 1986; Peterson et al., 1981), but most of these data were published many years ago and were limited by the small number of the

* This research was supported by '99 Chungnam National University Research Fund ('99 충남대학교 자체연구비)

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used autonomic variables (Berg et al., 1975; Hanson et al., 1993; Kryter, 1980; Poulton, 1979). Generally these studies reported about laboratory experiments, mostly noise of relatively short duration, but some are field and epidemiological investigations of aftereffects of noise on phychophysiological responses, along with the considerable suggestive evidence that long-term intense noise impairs health (Boucsein, Ottman, 196; Cohen, 1980; Peterson et al., 1981; Sohn et al., 1999).

Many of the studies on alteration of psychophysiological state as a result of long-term exposure to noise have been related to changes in autonomic indices such as heart rate, respiration, skin conductance, and blood pressure, i.e., variables that usually are also sensitive as the measures of physiological arousal (Loeb, 1986). From the very early studies it was known that repeated (or long-lasting) stimulation (70-90dB range) may lead to adaptation of most physiological responses, however, sometimes habituation effects of certain variables (respiration, pulse volume etc.) were not significant (Davis et al., 1955 cited by Loeb, 1986). Most of psychological studies stated that noise intensities above 70dB, if continued for a long time, are becoming subjectively aversive to people (Kryter, 1970; Loeb, 1986). Nevertheless, it should be mentioned that there are also many other factors moderating psychophysiological effects of noise such as psychoacoustic characteristics of noise, availability of avoidance strategies, interference with the ongoing task to perform etc. (Berg et al., 1975; Boucsein, Ottman, 1996; Cohen, 1980; Hanson et al., 1993; Fischer, 1972; Loeb, 1986; Sokhadze et al., 1999).

There still exist many unresolved questions regarding bodily changes evoked by exposure to noise stress and this topic continues to attract interest of psychophysiologists. In our previous studies we demonstrated that intense white noise exerts effects on autonomic balance while subjects were performing a cognitive task with noise background and suggested what autonomic mechanisms were mediating the observed changes of physiological activity during acute stress episodes (Sohn et al., 1999; Sokhadze et al., 1999), and also identified indicators of attention and emotions during loud affective auditory stimulation (Sokhadze et al., 1998). Nevertheless, mechanisms of physiological responses during long-term exposure to noise, more relevant to chronic accumulated stress, might be different from those manifesting during short-term acute stress episodes. Namely, we found out in the study using stress modeled by cognitive load with noise background (Sohn et al., 1999) that during 40 s of stress physiological activity was indicating a phasic concurrent activation of sympathetic and parasympathetic branches of the autonomic nervous system. We interpreted this result as manifestation of compensatory effects directed to prevent sympathetic over-arousal and as process of optimization of functional state during performance on metal stress task. However, it was not clear how long this adaptive mechanisms are functioning.

The purpose of this study was to analyze of the dynamics of tonic physiological changes and subjective evaluation of stress evoked by long-term (30 min) exposure to intense aversive auditory stimulation with 85dB white noise.

Method

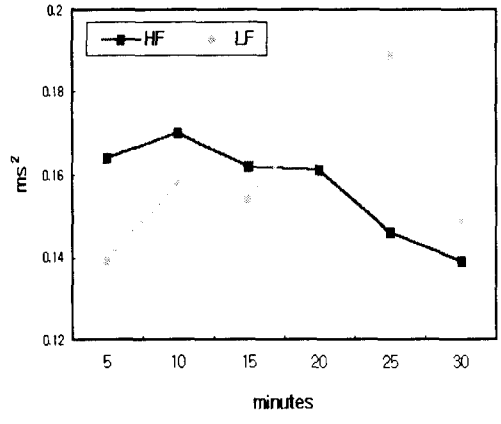
The study was conducted on 23 college students (19-24 years old). As the instruction stated the subjects had an

option to quit the experiment if they found it too stressful. And eight subjects dropped out. Thus, reported are data only on the 15 subjects who completed the experiment.

Physiological signals (ECG, electrodermal activity, skin temperature, and respiration) were acquired by BIOPAC MP100WS, Grass Neurodata System and AcqKnowlwdge 3.5 software. Following physiological variables were recorded: electrodermal activity (EDA), e.g., skin conductance level (SCL), SCR number (N-SCR); cardiovascular activity, namely, inter-beat intervals (RR intervals of ECG), HF (0.15-0.4 Hz) and LF (0.04-0.15 Hz) components of RR intervals power spectrum and HF/LF ratio of inter-beat period variability (HRV) (Bernston et al., 1993), skin temperature (SKT); and respiratory activity, such as respiration rate (RESP) during the baseline resting state (1 min) and 6 sessions of 5 min long exposure to the intensive white noise (85dB[A], delivered through loudspeakers). Subjective rating of experienced stress level (0-100 scale) during test was assessed after each 5-min session. Zero of the scale indicated 'not stressful at all' ;100 indicated 'extremely stressful'.

Results

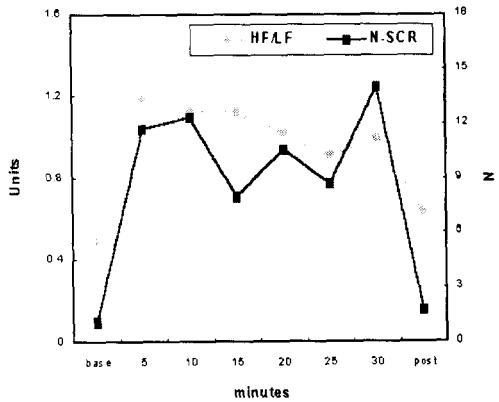
The HF component of HRV (indicator of parasympathetic activity) showed increasing tendency only in the second session (10 min) and then was gradually decreasing (Fig. 1), whereas the LF component of HRV (indicator of mostly sympathetic activity) tended to increase during the first 5 sessions, reaching the peak at 25 min of exposure to WN (Fig 1). During the first 4 sessions (up to 20 min) the HF and LF components (autonomic balance indicator) were featured by high correlation



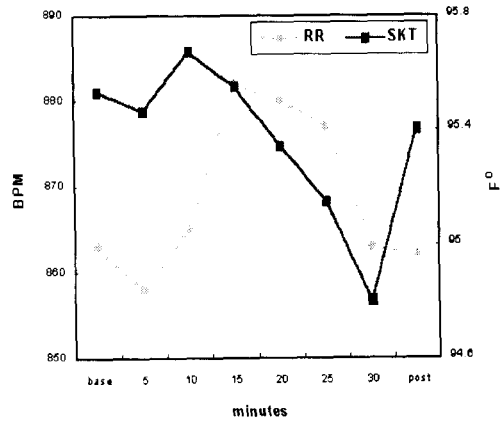
(Figure 1) HF and LF components of HRV during 30 min of exposure to 85dB white noise (N=15). Dissociation of HF and LF components begins from after 20 min and reaches maximum at 25 min of aversive stimulation, whereas changes of HF and LF proceeded in parallel at first 15 min of noise.

(correlation coefficients respectively 0.80, 0.59, 0.93, 0.63, all ps <0.01). However at the 5th session (25 min) correlation was disrupted (r=0.19, p=0.55) and HF and LF dissociated (Fig 1). The HF/LF ratio of HRV showed this trend in the declining values across sessions, thus indicating the take over of LF (and dominance of sympathetic drive) in the cardiac autonomic balance (Fig 2). The same trend was typical for correlation of subjective stress rating scores vs. LF , which was significant (r=0.64, p=0.023) only on 25th min of noise stress.

The profile of changes of HF/LF balance is comparable qualitatively to RR interval dynamic. Inter-beat intervals became shorter in the first 5 min, then followed by adaptation (increase 10-15 min) and the second wave of decrease and total recovery in post-stimulation period (dashed line,

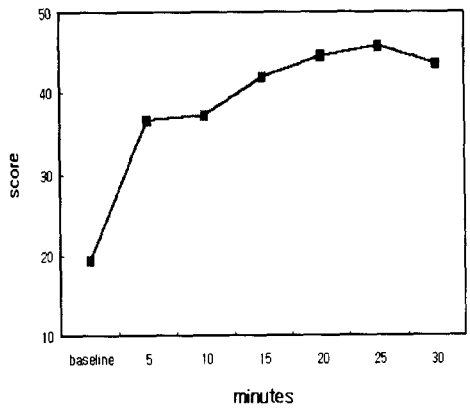


(Figure 2) HF/LF ratio of HRV and N-SCR (number of non- specific SCR) - indicator of tonic sympathetic activation - during 30 min exposure to white noise background (85dB). Both curves show initial and second phases of acceleration with peaks centered at 5th and 30th min of auditory stimulation.



(Figure 3) RR intervals of ECG and skin temperature during baseline, 30 min of exposure to 85dB white noise and post-stimulation period (N=15). Changes of both variables has similar profile - first phasic decrease, followed by rebound and short-term over-compensation and second tonic decrease after 10-15 min of aversive stimulation.

Fig. 3). A similar pattern of two-wave decrease was demonstrated as well by SKT (solid line, Fig. 3). Tonic non-specific electrodermal activity was also featured by the initial and second phases of acceleration with peak on the 5th session (N-SCR on Fig. 2), almost in parallel with the HF/LF ratio curve. Only respiration (RESP) and subjective stress rating demonstrated gradual increase with time of exposure to the intense noise (subjective rating on Fig. 4).



(Figure 4) Subjective rating of experienced stress level during 30 min of 85 dB white noise.

Discussion and Conclusion

The results demonstrated that during long-term exposure to noise physiological variables are exhibiting pattern which is typical of most of recorded variables, namely short-term activation (e.g., RR decrease, SKT decrease, N-SCR increase etc.) followed by adaptation (habituation) and subsequent wave of activation, which resulted in failure to recover in the post-stimulation period for some variables. As it was clearly seen in RR and HRV dynamics, heart rate acceleration response was manifested at the initial stages of

exposure, but then the concurrent activation of parasympathetic and sympathetic inputs resulted in temporal compensation and rebound effect, when RR increased even above the baseline level. However, the cardiac responses demonstrated recovery in the post-stimulation period and did not show significant aftereffects.

These data are comparable to results cited in Jennings (1986) that loud noise (108dB) produced physiological effects (vasoconstriction, SCL increase etc.) and then these effects seemed to adapt completely within 23-25 min of exposure. However in our study with a relatively lower intensity of noise, after 15-20 min of exposure to 85dB WN, sympathetic activation started to dominate and led to the second peak of transient RR decrease. Domination of sympathetic arousal in the 5th - 6th sessions of the experiment was also expressed in such indicators of sympathetic activity as SKT decrease, increased LF of HRV and also increased electrodermal activity (N-SCR, frequency of SCR on per minute basis etc.).

Our results are in accord with some of reports about effects of noise stress, such as rise of activity in some physiological parameters followed by a pronounced decrease and then a sustained rise (Hanson et al., 1993; Peterson et al., 1981), but our results are not consistent with studies where only short-term stress was modeled (Jennings, 1986; Sohn et al., 1999; Sokhadze et al., 1999). Nevertheless, the current experimental manipulation employing long-term exposure to intense noise as accumulated stress model is closer to traditional understanding of stress response, when long-lasting monotonous aversive stimulation may result in subjective evaluation of the situation as stressful (negative affect) and is accompanied with signs of tonic sympathetic domination manifested by increase in physiological parameters, which are usually considered as sympathetic activation markers (LF of HRV, SKT, N-SCR etc.) and decline in tonic activity of parasympathetic system (decrease of HF power of HRV) at later stages of aversive stimulation.

In conclusion it should be stated that relatively long-term (30 min) exposure to intense noise (85dB WN) influence psychophysiological responses, and that the influence was expressed mainly in a three-phase curve, namely short-term activation (5-10 min), followed by adaptation and rebound effect (10-20 min) then a subsequent second wave of activation (20-30 min, peak at 25 min) suggesting that observed effects are evoked by the prevalence of tonic sympathetic arousal. Autonomic, respiratory and subjective measures employed in our study indicated that the exposure to loud noise provoked psychophysiological state with tonic physiological and psychological changes usually associated with accumulated stress.

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