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NOISE INDUCED STRESS

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Abstract

Noise has the potential to cause stress reaction. Chronic noise induced stress enhance the aging of the myocardium and thus increase the risk of myocardial infarction. The involved path mechanism include acute increase of catecholamines or cortisol under acute noise exposure and an interaction between endocrine reactions and intracellular Ca/Mg shifts. Chronic noise exposure of animals on a diet with suboptimal magnesium content led to increase of connective tissue and calcium, and decrease of magnesium in the myocardium. These changes were correlated to noradrenaline and normal aging. Post mortem studies of hearts from victims of ischemic heart diseases confirmed the importance of Ca/Mg shifts in humans. Recent epidemiological studies support the importance of noise as a risk factor in circulatory and heart diseases, especially in myocardial infarction.

Introduction

There is increasing epidemiological evidence that chronic exposure increase the cardiovascular risk, especially the risk of ischemic heart disease. The theoretical basis of the epidemiological noise and health studies is non-specific stress effect of noise. For methodological reason much of the work was concentrated on noise-induced vasoconcentration and increase of blood pressure. However, the epidemiological evidence is pointing more to a noise related risk increase of myocardial infarction (MI) then of hypertension. Since hypertension is only one of a long list of risk factor in MI, noise seems to affect other risk factors other risk factors more than blood pressure. One of the most important risk factors in MI is age, or more precisely the biological age of heart. In the following we will measure some parameter of acute and chronic noise exposure on several risk factors in MI, including some parameters which are related to the biological age of the heart.

Noise-induced stress reactions

Acute exposure to maximal sound pressure level above 90dB(A) has the potential to cause inner ear hearing loss and to stimulate the sympathetic nervous system into increasing the release of adrenaline and noradrenaline. We found an increase of naradrenaline in persons exposed to habitual work noise. We examine the difference of noradrenaline (NA) in 15 workers who were exposed to noise levels of Leq=94-101 dB(A), when working for one day without ear protectors and Leq=81-98dB(A) and for one day with ear protectors. In the subgroup of persons with low Mg content in the erythrocytes, the noise induced increase of noradrenaline was early double as compared with persons with high erythrocytes-Mg.

In dogs exposed to 3min. Noise of an alarm clock (75 dB) the plasma concentrations of adrenaline, noradrenaline, adrenocorticotropic hormone (ACTH) and cortisol were studied.

In a rat experiment a dose response relationship of stress hormones under exposure to traffic noise was demonstrated. The plasma concentration of cyclic adenosine, 3′, 5′ ,monophosphate (c-AMP) was measured after 4 h. Exposure to recorded traffic noise with level of 60, 80 and 100 dB(A). Traffic noise of 60 dB(A) as well as 80 dB(A) caused a 30% increase of c-AMP while 100 dB(A) traffic noise resulted in an 50% increase of c-AMP.

Stress-Induced Myocardial Injury

A special technique was applied to measure the ECG in conscious unrestricted rats. Each rat was monitored for 1h before exposure, 1h of noise exposure and 2h after the exposure on three consecutive days. In 55% of the rats, the heart rate decrease and the ST interval increase was reproducible during noise exposure and normalized directly after the end of the exposure. The noise-induced increase of the ECG ST segment was similar to ST increase during myocardial ischemia, caused by injections of isoprenaline or occlusion of a coronary artery.

Long term health effects of chronic noise stress in animals

In an animal study with persistently repeated noise exposure a chronic increase of noradrenaline was found. In this experiment, 6 groups of rats were fed diets with normal, suboptimal, or deficient content of Mg respectively, of which 3 groups were used as noise controls. Mg deficiency was used as a model for a stressor which acts synergistically with noise. After one month on these diets, noise exposure of 3 groups was started and continued for 3 months: Traffic noise (maximal level 85 dB) with quit intervals and a mean noise level of 70 dB (frequency weighted according to the frequency characteristic of the rat's hearing threshold) for 2 groups and for the 3rd group a constant noise with a mean level of 74 dB was added.

Mg-deficient diet alone resulted in a marked chronic increase of noradrenaline excretion, which increased further with noise (70 dB: 32%, 74 dB: 76%). This shows that Mg-deficiency and noise are stressors which act synergistically. During the experiment, the body weight of the controls increased from 200g to 402g and 335g respectively, for both of the groups on the suboptimal Mg

diets. However , it was constant in the Mg-deficient group without noise and and was seen to decrease when the noise was applied. Using noradrenaline as an indicator of the total stress, the death rate, ca content and the collagen of the myocard and the quotient of Ca/Mg increased while the Mg-content decressed with increasing stress.

Interaction of stress and Ca/Mg shifts

Further analysis of the experimental results led to an interaction model between chronic stress caused a loss of extracellular and intracellular Mg and an increase of intracellular Ca. A decrease of Mg was correlated to an increased physiological noise sensitivity, i.e. more severe noradrenaline releases in animals and humans under noise exposure. We found a positive feedback mechanism between stress-caused by noise and/or other stressors- and intracellular Ca/Mg shifts which may increase the cardiovascular risk.

Chronic noise stress, Mg-balance and cardiovascular risk

In several studies, it was shown that chronic noise exposure has the potential to cause chronically increased noradrenaline and/or cortisol. Acute and chronic occupational noise exposure led to acute and chronic increase of noradrenaline and cortisol.

Six weeks of experimental night time flight noise exposure(Leq=32 dB(A), Lmax=65 dB(A) 32 times per night) resulted in an acute increase of cortisol excretion, followed by about two weeks of normalization and subsequent long-term increase of cortisol in males. In this experiment blood samples were taken before and after the noise exposure. Among other biochemical parameter the Mg concentration of the erythrocytes (EMg) was analysed by atomic absorption spectroscopy. In one of the 16 test persons EMg decreased, indicating a negative Mg-balance during the 6 weeks of noght time exposure. Five other persons had unusually low EMg already at the beginning of the experiment and it remained low.

A cohort study on the relation of noise, Mg and blood pressure revealed a significant negative correlation between EMg and blood pressure. The pathophysiological mechanisms involved in the development of hypertension under noise exposure, in combination with optimal and sub-optimal Mgintake were studied by Altura et.al.(1992). Besides the already mentioned Ca/Mg shifts in the vascular walls, an increased vasoconstriction under the action of noradrenaline was observed. This effect was confirmed in humans by measuring the increase of the total peripheral resistance (TPR) during infusion of noradrenaline.

The noradrenaline induced TPR increase was reduced by Mg-injections. These injections increased the serum Mg concentration (SMg) to such a degree that also was observed during acute stress of rats on a diet with optimal Mg content. Rats exposed to a combination of sub-optimal Mg input and stress for 12 weeks, had significantly reduced SMg and reacted to acute stress with significantly reduced SMg increase.

Discussion

Long-term health effect of noise can be studied in the animal model, if intensive noise cause directly an increase of stress hormones. Noise with moderate levels has the potential to disturb activities i.e. concentration, communication and sleep and therefore to cause indirectly stress hormones increase. Noise induced indirectly transmitted stress reactions can not be studied in animals as a model for humans; however, the long term health effects of directly and indirectly increased stress hormones will be qualitatively similar in humans and animals. Animal's models can therefore be used to study this part of the problem too.

Several conditions increase the risk for chronic noise-induced stress reaction:

1. Noise is not the only stressor: in combination with other synergic stressor i.e. cold (Heroux et al.,1977), suboptimal Mg intake (Gunther et al.,1978, Ising et al.,1981), nicotine and / or caffeine (Naworth, 1984), overcrowding (Henry and stevens 1977).
2. Persistent noise may lead to an excedence of the individual potential of adaption to stressors.
3. Hereditary defects, for example in spontaneous hypertensive rats lower the stress tolerance.
4. During sleep the levels that cause noise-induced stress reactions are much lower than in the active phase.
5. In humans acute acute and chronic stress hormone increase can be caused indirectly by environmental noise via disturbance of activities such as concentration, communication, circulation and sleep.

Conclusion

From these findings we conclude, that traffic noise exposure, especially at night time, has the potential to cause chronic increase of noradrenaline and cortisol. In a certain percentage of the population cortisol may be increased above the normal range.

Based on the results of the above demonstrated experiment, we developed a model of noise effects and formulated the hypothesis that chronic noise exposure accelerates the aging of the myocardium and the vascular walls, and therefore increases the risk of myocardial infarction.

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