Changes in deep body temperatures and auditory thresholds following exposure to noise and carbon monoxide at various ambient temperatures

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Abstract

This article consists of two separate laboratory experiments. In experiment 1 deep body temperatures (dbt-values) of 72 male Long-Evans hooded rats were determined immediately before and after a four hour exposure to 18 combinations of noise, carbon monoxide (CO) and ambient temperatures. The noise levels were no extra noise, broad band noise of 90 dBA and 105 dBA. The ambient temperature levels were maintained within chamber at 20 °C, 35 °C and 40 °C. CO levels were 0 and 750 ppm. Dbt alteration utilized as an index of general load produced by these burdens. The results showed that the dbt-values changed least when rats were exposed solely to 20 °C (x = + 0.1°C). When adding either noise of 90 dBA or 105 dBA or 750 ppm CO to the 20 °C ambient temperature condition the decrease of the mean dbt-values was 1.5 °C to 1.8 °C. However, the greatest mean decrease in the dbt-values were found when rats were simultaneously exposed to 750 ppm CO and noise of 90 dBA (x = -2.8°C) and 750 ppm CO and noise of 105 dBA (x = -2.6°C). At 35 °C no similar changes were found. At 40 °C the tendency had totally reversed such that under both noise exposures rats gained heat (x = 0.6 °C to 0.8 °C) and especially under the combination of 105 dBA and 750 ppm CO the mean dbt-values increased by 1.6 °C. In experiment 2 hearing threshold shifts (dB) at 10 kHz and 40 kHz of 16 male Long-Evans hooded rats were determined for six days before and after a four hour exposure to ambient temperature of 20 °C, to a 105 dBA noise at 20 °C, to a 105 dBA noise at 35 °C, and to a 105 dBA noise with 750 ppm CO at 35 °C. The results showed that the mean loss of hearing was greatest at 40 kHz after exposure to a 105 dBA noise with 750 ppm CO at 35 °C.

Key words: Carbon monoxide — Noise — Ambient temperature — Combined effects — Deep body temperature — Hearing threshold shift

Introduction

Noise, carbon monoxide and temperature are very common environmental factors, to which millions of people are daily exposed in their working environments or during their leisure in their living and traffic environments (Manninen 1988a). It is also generally known that in many working places and vehicles noise is present in different qualities and quantities. On the other hand, air temperature may also rise in working places and vehicles beyond the range that feels comfortable due to deficient or lacking ventilation and the heat generated by machines.

As these environmental factors are often present simultaneously in the same environments and working situations, it is important both for medical reasons and considering public health to find out the combined effects of these three factors, in particular. A question not only of scientific interest but also of relevance in everyday exposure situations is whether an elevated ambient temperature (and body temperature) may potentiate a noise and carbon monoxide induced hearing loss, in other words, whether an elevated temperature is an additional risk factor in connection with exposures to noise and carbon monoxide?

Previous research has shown that a carbon monoxide exposure of 1200 ppm lasting 90 minutes before and a carbon monoxide exposure lasting 120 minutes together with an exposure to a 110 dBA noise brings
forth considerably greater changes in hearing than a noise exposure alone. (Young et al 1987). It has therefore been assumed that a blood hypoxia following a carbon monoxide exposure would promote the development of changes in hearing. According to Zorn (1968) and Zeigelschafer (1968) the effects of noise and carbon monoxide might be cumulative.

The very new research results show that the differences in the blood counts of smokers and non-smokers are particularly outstanding when the subjects are exposed to a whole body vibration simultaneously with the noise exposure (Manninen 1988a). Together with some previous observations (Manninen 1983, 1986, 1988b, 1990) this finding suggests that temporary changes in hearing are connected to metabolic and body temperature changes. For example, the sublingual temperature and temporal temperature dropped most either due to mere sinusoidal 5 Hz vibration or due to a simultaneous whole body vibration and a 95 dBA wide band noise. On the other hand, it is generally known that a carbon monoxide exposure results in a reduced blood oxygen level and a decrease in body temperature. The decrease in temperature slows metabolism and oxygen consumption down. If metabolism is increased by adding the strenuousness of physical work, the effects of noise and vibration on the TTS values can be temporarily inhibited (Manninen 1984). All in all, in humans these changes in the hearing threshold are most distinct at the frequencies of 4 and 6 kHz.

Fechter (1988) has also shown that a carbon monoxide exposure may promote the development of noise-induced damages at high frequencies and histopathology in the basal region of cochlea. Researchers find it quite probable that cells at the basal region of cochlea would have a higher metabolic demand than cells located in the apex (Fechter et al 1987). Therefore changes in those environmental factors would be reflected most clearly in the operation of basal cells.

Summarizing the above brief review it can be assumed that increasing the body core temperature (by increasing the temperature of the environment) can to a certain degree compensate for the decrease in body temperature, deteriorated metabolism and further the increase of the hearing threshold (resulting from simultaneous noise and carbon monoxide exposure). When the body core temperature due to heat grows higher than what the exposure situation and related compensation need require, the blood oxygen consumption goes up under the effect of heat combined with the noise and carbon monoxide exposure, metabolism decreases and hearing dysfunctions grow stronger.

To prove this hypothesis true the following two experiments with animals were conducted.

Material and methods

Experiment 1 was planned to characterize changes in the deep body temperatures (dbt-values) during exposures and Experiment 2 was planned to characterize changes in hearing thresholds due to the exposure combinations.

Experimental subjects

In both experiments subjects included male Long-Evans hooded rats (Blue-Spruce; Altamont, N. Y.) 250 mg upon arrival. The subjects were housed in plastic shoebox cages in a colony room with a 12/12 hour light/dark cycle (lights at 7.00 am); all animals were provided food and water ad libitum except during exposure and testing. In experiment 1 there were altogether 72 rats and in experiment 2 altogether 16 rats.

Experimental design

Experiment 1 was realized as a randomized 2-3-3 type factorial test whereas the experiment 2 was based on the block design test. Experimental protocols used in the two experiments were quite similar. Following the initial hearing threshold determinations one subject from each squad of four animals was assigned to one of the experimental conditions: total number of exposure conditions was 18 in experiment 1 and in experiment 2 the corresponding number of exposure conditions was 4.

In experiment 1 the noise categories were 1) no noise, 2) 90 dBA broad band stable noise, and 3) 105 dBA broad band stable noise. Dry bulb temperature inside the exposure chamber was 1) 20 °C, 2) 35 °C and 3) 40 °C. The carbon monoxide content in the exposure situation was 1) 0 ppm CO, and 2) 750 ppm CO. In experiment 2 exposure conditions were 1) 20 °C (control condition), 2) 105 dBA broad band stable noise at 20 °C, 3) 105 dBA broad band stable noise at 35 °C, and 4) 105 dBA broad band stable noise with 750 ppm CO at 35 °C. In both experiments subjects were exposed once to one of the exposure conditions for four hours.

Exposure chamber

Exposures to noise, carbon monoxide at various temperature levels took place in an IAC sound attenuating chamber designed for small animal experiments, which was itself enclosed in an IAC sound attenuating room. The dimensions of the animal
chamber were 40 cm deep x 60 cm wide x 36 cm high. The interior surface of the chamber was stainless steel, providing a reverberant environment.

Dry bulb temperature inside the chamber was produced through an electric heater that was regulated by variable resistor. The variable resistor was situated in the observation room where the researchers worked, too. Temperature levels inside the chamber was monitored through a digital Micronta indoor/outdoor thermometer.

Teflon tubes from carbon monoxide tanks were directed via microvalves and filters to the middle part of chamber. The tip of gas outlet was fixed above the tip of gas inlet and connected to infusion pump of the carbon monoxide analyzer. During the exposure period fluctuation of carbon monoxide content was kept as small as possible, at least within $\pm$ 50 ppm from the desired exposure level (ie 750 ppm CO). When needed interior content of carbon monoxide was corrected through regulating the flow rate of the infusion pump of the carbon monoxide analyzer.

Subjects were exposed in a wire-mesh cage (8cm x 17cm x 19cm) and sound levels were monitored with a Quest model 215 type 2 sound level meter with the microphone at the approximate level of the rats' ears. The noise had maximum intensity between 4 and 8 kHz. Noise was produced by a laboratory-built noise source, amplified by a SAE model 201 amplifier. The loudspeaker located right above the cage where the subjects were staying during the exposure period.

**Determination of dbt-values**

Right before and after the exposure the deep body temperatures (dbt-values) of the subjects were measured by inserting a probe of a thermometer in the rat's rectum. For each measurement the probe was inserted five centimetres in the rectum and it was kept stable until the reading of the meter stabilized completely, which usually took 2 minutes. Dbt-determinations were done exactly in the same way each time. To eliminate the effects of the season and of the day, the subjects were exposed to several combinations during each exposure period. The combinations were allotted.

**Reflex modulation audiometry**

The application of reflex modulation procedures to the assessment of auditory function has been described in previous papers (Young and Fechter 1983). Unlike other reflexive test methods for assessing threshold this procedure uses the inhibition of the reflex produced by low-intensity pure-tone prestimuli rather than absolute reflex amplitude, to estimate auditory thresholds.

Briefly, rats were brought into an audiometric chamber (IAC model 1200) containing four test boxes, each of which housed a wire mesh cage (22x10x9.5 cm) suspended on a frame by four metal springs; a cylindrical magnet protruded from the bottom of the cage. The interior of each test box was 61 cm deep, 72 cm wide and 118 cm high. Centred beneath each cage is a wire coil connected to a nine volt amplifier that behaved as accelerometer to vertical movement of the cage, reflecting ballistic startle movements of the rat to white noise bursts over a 50 msec interval following noise burst onset. Three piezo-electric speakers mounted directly above and to the sides of each cage deliver the acoustic stimuli. The two speakers on either side of the cage delivered the noise burst, while the speaker on top delivered the pure tone stimuli. The speakers had a reasonably flat response between two and fifty kHz.

On a given trial, a 20 msec white noise burst (105 dBA), which reliably elicits a large magnitude reflexive startle response, was given either alone or preceded (210 msec) by a pure tone prestimulus at either ten or forty kHz; these frequencies sample mid and high frequency hearing, respectively, in the rat. Low intensity prestimuli have been shown to attenuate the magnitude of the startle response relative to that elicited by noise bursts alone, the magnitude of reflex diminution being proportionate to prestimulus intensity.

The present investigation provided 450 trials per day to each subject, in six randomly assigned blocks of 75 trials (60 prestimulus, 15 noise alone), presented randomly at mean intervals of 45 seconds. The trials consisting of noise bursts alone without pure tone prestimuli yielded baseline values to which trials with prestimuli were related. Trials with prestimuli included the two frequencies at each of 19 attenuation steps (0-90 dB SPL at five dB intervals) in a balanced design so that three repetitions each of 20 frequency x attenuation step combinations were presented in a given block. The entire regimen lasted six hours and was presented daily during every seven day test period.

Each day, rats were brought from the vivarium into the behavioral laboratory. A 1/2" Brüel & Kjaer (model 4199) microphone was centred in one cage and connected to a Brüel & Kjaer (model 2610A) measuring amplifier and Hewlett-Packard (model 5210A) frequency meter. A computer then presented several white noise and pure tone trials in order to calibrate stimulus frequency and intensity, as well as to test the responsiveness of each cage's accelerometer. The subjects were then placed into the cages and given ten 'warm-up' trials to permit them to accli-
mate to the test situation; these trials were not analyzed. A computer-driven program then delivered the six trial blocks, recorded individual responses and performed descriptive statistics on the data. Upon completion of testing, the animals were returned to the vivarium, and the test cages readied for the session.

All animals were pretested for seven days in order to determine pre-exposure acoustic thresholds. Animals were then exposed for four hours for one day to the four exposure combinations mentioned previously. Immediately after this the rats were again tested for seven days in order to determine post-exposure hearing sensitivity.

Auditory thresholds were determined by averaging a subject's responses to each frequency x attenuation combination across all trials for a given week. First day responses for given week of both pre- and post-exposure tests were omitted because previous studies have shown uncharacteristically high response levels on the initial day in a series. Threshold was operationalized as the prestimulus intensity level in dB SPL that produced 15% attenuation in startle amplitude relative to baseline trials. Threshold shift values, which represent the change in acoustic thresholds from pre-exposure levels, were subjected to statistical analysis.

Analysis of data

The results are presented as arithmetic means (x) of differences and standard errors of the corresponding means (SEM) in classes of independent variables. The differences were obtained through subtracting the post-exposure values from the pre-exposure values. Calculations were performed over the whole material and by exposure combinations. To depict single and combined effects three-way variance analyses (F-ratios) were calculated.

Results

Figure 1 shows the mean differences in dbt-values by exposure combinations. The shaded diagrams depict exposure combinations including a simultaneous carbon monoxide exposure (750 ppm CO). The numbers on the horizontal axis in the middle of the figure indicate the intensity of the noise used (0, 90, and 105 dBA). At the bottom of the figure there are the exposure chamber temperatures (20, 35, 40 °C).

A look at the left part of the figure reveals that 750 ppm CO reduced the dbt-values at 20 °C. The reduction was especially high when the subjects were exposed simultaneously either to a 90 dBA or to a 105 dBA noise. When the temperature was risen to 35 °C and the rats were simultaneously exposed to noises or simultaneous noises and 750 ppm CO, changes in the dbt-values were rather small and unsystematic in comparison to changes at 20 °C. When the temperature inside the chamber was risen to 40 °C the increase in the dbt-values was the greater the more intense noise was used. In particular when the subjects were exposed simultaneously to 750 ppm CO and a 105 dBA noise at 40 °C, the increase in dbt-values was very significant.

Table 1 shows that carbon monoxide content (factor A), noise (factor B) and temperature (factor C) have very significant single effects on the variation of the dbt-values. Furthermore, the results of the variance analyses showed that the combinations of any two factors (carbon monoxide and noise, carbon monoxide and temperature, and noise and temperature) have a very significant combined effect on the variation of the dbt-values.

Figure 2 shows that the mean loss of hearing is greater after exposure to a 105 dBA noise and 750 ppm CO at 35 °C than after any other exposures. Especially, the results show that an ambient temperature of 35 °C with 105 dBA noise was decreasing the sensitivity of hearing more than the temperature of 20 °C together with a 105 dBA noise. However, the deviation of threshold shift values was marked.

Discussion

Numerous previous studies on thermal climate and thermal physiology (eg Berglund et al 1991) unanimously point out that for cold environments, a heat loss greater than heat production due to a variety of activities and shivering causes the body core temperature to fall. On the contrary, if the heat loss is insufficient, the core temperature will increase above the controlled level. As demonstrated here in complex exposure situations environmental temperature plays a dual role. However, the more interesting is that not only the ambient temperature but also the carbon monoxide content and noise will enhance the same short-term falling or rising of the deep body temperatures; the normal functioning of the cooling system of the body is disturbed at high temperatures in particular where the subjects are simultaneously exposed to an intense noise. Especially, a simultaneous carbon monoxide content seems to increase the thermal load of the body when the subjects are exposed to carbon monoxide at high temperature. Correspondingly, the temperature regulation system of the body is also disturbed at a low temperature when the subjects are simultaneously exposed to intense noise. In other words, at low environmental
temperatures the effects of cold and at high environmental temperatures the effects of heat are enhanced by noise and carbon monoxide.

In an exposure situation similar to this the state of the systemic circulation and height of the systemic blood pressure might be important for the demonstration of sympathetic stimuli on inner ear function. So far circulatory disorder may be one of the major pathogenetic factors responsible for sudden loss of hearing. It could be stated that depending on the ambient temperature level hypotension in one hand, hypertension, in the other hand, is prerequisite for the effect of extreme sympathetic stimulation on diminution of inner ear blood flow, probably either through vasoconstriction or vasodilation and sludge formation. However, the observation that carbon monoxide is ineffective in producing a clearer auditory impairment, is surprising even though the mean shift of hearing thresholds at 40 kHz were highest after an exposure combination including 750 ppm CO. As a matter of fact it seemed that very elevated ambient temperature has the most dramatic influence on hearing sensitivity. As Figure 2 presents by increasing temperature from 20 °C to 35 °C and exposing subjects simultaneously to intense noise, loss of hearing is happening in concrete way. It is also worth while noting that the potentiation of heat-induced auditory dysfunction under noise exposure appears to be frequency specific. This finding is parallel to the previous findings of Young et al (1987).

Table 1. Single and combined effects of carbon monoxide (factor A), noise (factor B) and ambient temperature (factor C) on the dbt-values. Results are obtained from the three-way variance analyses

<table>
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<th>SUM SQUARES</th>
<th>D. F.</th>
<th>MEAN SQUARE</th>
<th>F RATIO</th>
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<td>71</td>
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On the whole, environmental temperature seems to be a considerable additional risk in exposures. This additional risk should be taken into account in everyday life and particularly in traffic and work places. To reach a better understanding of this phenomenon, future studies should examine how changes in deep body temperature are reflected in other functions of the body. Other questions worth of further studies are, does the changes in the deep body temperature appear in the same way when the carbon monoxide content or the temperature is higher or lower than in this study, and when the duration of exposure is different from what was used here.

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