Canadian Acoustics / Acoustique Canadienne 20(1) 21-25 (1992) *Research article / Article de recherche*

THE EFFECTS OF INHALATION OF OXYGEN AND CARBON DIOXIDE MIXTURES ON NOISE-INDUCED TEMPORARY THRESHOLD SHIFT IN HUMANS

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ABSTRACT

Sixteen subjects breathed either oxygen, carbogen, air with 5% carbon dioxide or air while subjected to 10 min of 100dB noise and for a further 10 min in order to explore the effect of the various gasses on the establishment and recovery of noise-induced temporary hearing threshold shift. Oxygen and carbogen were found to be effective but not different in effectiveness in reducing the establishment of temporary threshold shift suggesting that the role of carbon dioxide in the process is minimal. No evidence was found to suggest that oxygen or carbogen inhalation assists in recovery from temporary threshold shift.

SOMMAIRE

Seize sujets ont respiré soit de l'oxygène, du carbogène, de l'air avec 5% de dioxyde de carbone ou de l'air pendant 10 min alors qu'ils étaient exposés à un bruit de 100 dB, et pendant les 10 min subséquentes, afin d'explorer l'effet des différents gaz sur la manifestation des troubles temporaires du seuil de l'ouïe, et sur le rétablissement consécutif. L'oxygène et le carbogène sont également efficaces mais non-différents pour réduire l'apparition à la fatigue auditive de l'ouïe ce qui suggère que le rôle du dioxyde de carbogène est minime dans ce processus. Les résultats ne permettent pas de suggérer que l'inhalation d'oxygène ou de carbogène aiderait au rétablissement suite à la fatigue auditive.

1. INTRODUCTION

Inhalation of either a high concentration of oxygen or a 95% oxygen 5% carbon dioxide mixture (carbogen) has been shown to reduce hearing threshold shift due to intense noise in various animal studies (Joglekar, Lipscomb, & Shambaugh, 1977; Witter, Deka, Lipscomb, & Shambaugh, 1980; Brown, Vernon, & Fenwick, 1982); Brown, Meikle, & Lee, 1985) and also with humans (Joglekar et al., 1977; Patchett, 1980; Witter et al., 1980; Joyce & Patchett, 1982; Lindgren, Dengerink, & Axelsson, 1989).

Since Joglekar et al. (1977) considered that a more substantial diminution of temporary threshold shift (TTS) from noise exposure was shown when their subjects breathed carbogen rather than pure oxygen, most researchers have used this gas to explore the parameters of the effect. Since an increase in arterial carbon dioxide tension (PCO_2)is known to cause some cerebral vasodilation (Comroe, 1974), various investigators (e.g., Brown et al., 1985) have suggested that the carbon dioxide component of the carbogen may be the main element in the mechanism by which carbogen reduces noise induced TTS. This suggestion follows the proposition of Lawrence, Gonzales and Hawkins (1967) and Hawkins (1971) that noise induced threshold shift could be caused by vascular insufficiency in the inner ear possibly as a result of intense noise stimulation. Thus raised PCO_2 in the blood could counteract noise induced cochlear capillary constriction allowing greater passage of oxygen and other nutrients to inner ear areas being depleted in the course of high metabolism.

It is not yet clear what effect sound has on the cochlear vasculature, the evidence is conflicting. As a result of noise exposure, Lipscomb and Roettger (1973) found evidence suggesting constriction of cochlear capillaries and Dengerink, Axelsson, Miller, and Wright (1984) found a reduced blood supply to the cochlea in guinea pigs. However, Perlman and Kimura (1962) and Prazma, Rogers, and Pillsbury (1983) noted an increase of cochlear blood

flow after noise exposure. Oxygen tension (PO_2) in both endolymph (Misrahy, Shinabarger, & Arnold, 1958) and perilymph (Nuttall, Hultcrantz, & Lawrence, 1981) have been reported to be diminished under noise exposure suggesting a lessened supply. On the other hand, a reduced oxygen presence in cochlear fluids could be the likely result of heightened metabolism at the level of the organ of Corti (Ward, 1970) rather than an indication of reduction of supply.

The question as to whether artificially increased carbon dioxide in the blood has an effect of dilating cochlear blood vessels, or mediating a cochlear blood flow increase by some other means in the process whereby carbogen reduces noise induced TTS is also still open. Miles and Nuttall (1988) found a small dilation in stria vascularis capillaries of guinea pigs breathing carbogen. However, they found no dilation changes in animals breathing carbon dioxide in air but measured a 50% cochlear blood flow increase in these subjects. Similarly, Hultcrantz, Larsen, and Angelborg (1980) found a considerably greater cochlear blood flow with rabbits inhaling 7% carbon dioxide in air than carbogen. Cochlear blood flow increases with carbogen have been reported by Dengerink et al. (1984) with guinea pigs in a post-mortem histological study, but in a recent study by Miller, Bredsberg, Grenman, Suonpaa, Lindstrom, and Didier (1991) using a laser-Doppler flow meter found that as far as they could ascertain, carbogen inhalation had little if any influence on human cochlear blood flow.

From evidence such as the above, it does seem that carbon dioxide might alter cochlear blood flow but the studies on blood flow with carbogen rather suggest that this mechanism may not be the primary one to affect noise induced TTS. Carbogen clearly has an effect, possibly not as an agent in altering blood transport rate, but principally for its oxygen content.

The following study was mounted to compare the effects of the inhalation of oxygen, carbogen and carbon dioxide on noise induced TTS in order to attempt to assess the relative contribution that these gasses might make in reduction of TTS and in its recovery.

2. METHOD

Subjects

Twenty-four paid male college student volunteers, all with hearing threshold levels (HTLs) better than 20dB at 4kHz served as subjects.

Procedure

Subjects located in an I.A.C. sound attenuated chamber completed four sessions, each separated by at least 24hrs. At each session, HTLs at 4kHz of the subjects' previously established better ears were measured with pulsed tones beginning at 40dB HL on a Grason-Stadler E-800 Bekesy audiometer. The mean of the six-pen excursions following 1 min of recording was calculated as HTL.

Sixteen subjects were then exposed for 10 min to 100dB SPL of narrow band noise centred at 3kHz generated by an Alison Model 22 audiometer and narrow band filter Model 26, produced binaurally through TDH39 ear-phones. The noise was expected to produce a TTS 2 min after noise cessation (TTS_2) of approximately 15 dB at 4kHz (Ward, Glorig, & Sklar, 1958). The traditional test frequency for TTS studies of 4kHz, expected to show the greatest shift with noise (Ward et al. 1958) was chosen. TTS was measured with the Bekesy audiometer at 2 min, 5 min and 10 min after cessation of the noise using the same procedure as for the pre-test. The audiometer began at 40 dB HL 1 min after the noise cessation and six pen excursions, three each side of the 2 min point, were used to calculate TTS_2. The audiometer was then turned off and restarted at the 4 min point and again at the 9 min point to measure TTS_5 and TTS_{10}.

At each of the four sessions, subjects breathed a different gas either medical grade air, 100% oxygen, 95% oxygen with 5% carbon dioxide (carbogen) or 95% medical air with 5% carbon dioxide. The gas was delivered at a flow rate of 12 l/min through an Ohio Medical Products respiratory therapy nebulizer to provide humidity into a disposable plastic respiratory mask which the subject wore from the beginning of the pre-test until TTS_{10} had been measured. Presentation of gasses across subjects was organized in a Latin square design to control for order effects. Subjects were not made aware of which gas they breathed at any session and subsequent questioning indicated that they were not able to guess successfully.

To establish whether inhalation of the gasses used affected TTS rather than altered basic HTLs, the remaining eight subjects underwent the same procedures except that they were not subjected to the 100dB noise nor was their hearing retested 15 min or 20 min after the pre-test. They attended four sessions, each time breathing a different gas, were pre-tested at 4kHz in the manner described above then tested again 12 min after the pre-test.

Calculation of HTLs and TTSs were performed by an independent observer not aware of the experimental conditions under which the hearing test records were obtained.

3. RESULTS

TABLE 1
Average Threshold Shift in dB at 4kHZ
After 10 min Gas and Noise Exposure (N=16)

Gas	Mean TTS_2	SD	Mean TTS_5	SD	Mean TTS_{10}	SD
Air	13.27	4.53	10.75	4.14	7.43	3.62
Oxygen	9.97	4.45	6.94	5.14	4.80	4.23
Carbogen	8.94	5.78	6.90	3.94	5.22	4.60
Air+5% CO_2	10.89	4.94	8.31	4.46	5.91	4.20

The results for the subjects exposed to noise while breathing the various gasses are summarized in Table 1. A one-way repeated measures ANOVA revealed that at the 2 min point after noise exposure, average threshold shifts differed according to the gasses inhaled [$F(3,45) = 5.094$, $p < .05$]. Tukey post-hoc comparisons to reveal which avage threshold shifts were different from others indicated a significant difference between the average TTS_2 after air inhalation and both carbogen inhalation ($p < .01$) and oxygen inhalation ($p < .05$). None of the other comparisons showed significant differences. Inhalation of either 100% oxygen or carbogen, then, produced the least amount of threshold shift to the noise compared with inhalation of air. Although the difference did not reach an acceptable level of significance, compared with air inhalation, inhalation of 5% CO_2 and air, may also have shown a reduced threshold shift ($t = 1.37$, $p < .10$).

Table 2 shows averaged threshold shifts after 10 min of gas inhalation but no noise.

TABLE 2
Average Threshold Shift in dB at 4kHz
After 10min Gas Inhalation (N=8)

Gas	Mean	SD
Air	-3.66	5.17
Oxygen	0.80	5.27
Carbogen	1.08	3.27
Air + 5% CO_2	2.67	2.96

One sample *t* tests indicated that none of the mean threshold shift measurements differed significantly from zero. The apparent differences were no more than expected by measurement error, given this number of subjects. Thus no evidence was found that gas inhalation alters auditory thresholds.

On Figure 1 are plotted the TTS recovery curves following the cessation of the noise exposure. If inhalation of a particular gas resulted in a more rapid recovery than others, the curve for that gas would be expected to descend more steeply.The recovery curves in the figure appear to follow a parallel course with no indication that inhalation of a particular gas before and during recovery altered the rate of TTS recovery.

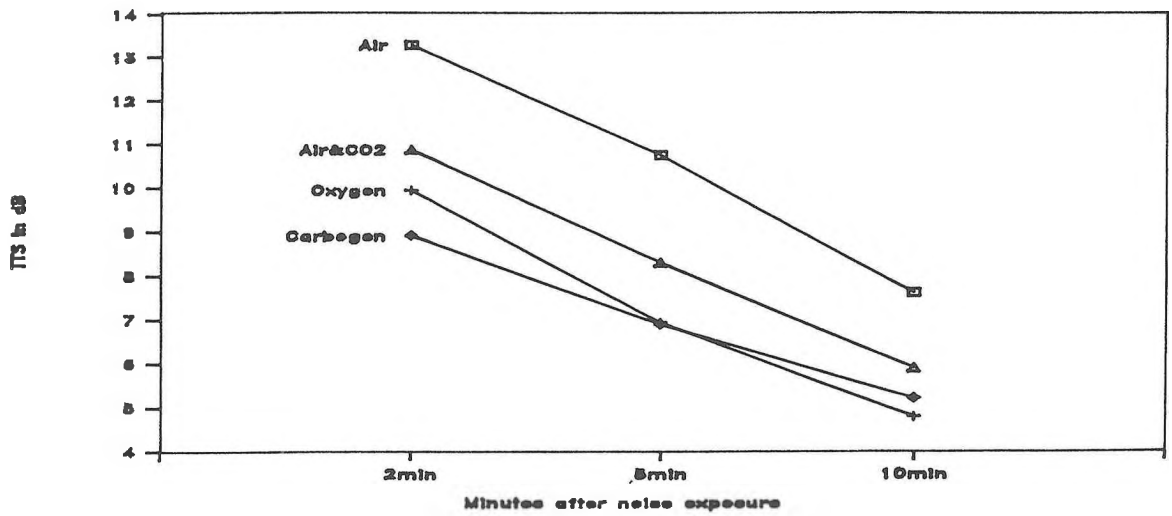

FIGURE 1: Average threshold shift recovery in dB for 16 subjects after exposure to 100dB of noise for 10 min with gas inhalation.

4. DISCUSSION

Inhalation of oxygen or carbogen reduced noise induced threshold shift, according to the results. Since oxygen and carbogen had no apparent effect on HTLs, it is reasonable to conclude that the effect of gas inhalation was to reduce noise induced threshold shift rather than to raise basic auditory acuity. The addition of carbon dioxide to the inhaled oxygen did not seem to make a difference. These findings coincide generally with those of Joglekar et al. (1977) who observed a beneficial effect of inhalation of both oxygen and carbogen on sound induced TTS, but do not agree with their observation that the effect of carbogen was superior. However, Joglekar et al. reached their conclusion about the superiority of carbogen from a consideration of a graphical representation of their results rather than from a statistical analysis.

Although strong efforts were made in this study to control sources of measurement error inherent in any research involving auditory threshold measurements, the influence of such error on the results cannot be discounted. Thus it is difficult to draw conclusions from the finding that there was no significant difference between TTS with carbogen inhalation as against oxygen inhalation. The data from the subjects exposed to the noise while breathing a mixture of air and carbon dioxide does suggest that carbon dioxide could possibly have some beneficial effect on TTS. However, it is clear that since TTS results with carbogen and oxygen were very similar, the added carbon dioxide played a minor rather than a dominant role in the TTS reduction. This conclusion does not agree with those of Joglekar et al.(1977), Degerink et al.(1984) nor that of Brown et al.(1985) all of whom ascribe a major role to the carbon dioxide in carbogen in the reduction of TTS. They propose that carbon dioxide dilates cochlear blood vessels allowing more blood and thus more oxygen to reach depleted areas in the cochlea during intense stimulation. The conclusion is consistent, however, with a more parsimonious explanation that raised blood PO_2 allows for more oxygen than usual to reach areas in the cochlea depleted in the course of intense metabolic activity.

There was no evidence that recovery from TTS was assisted differentially by the inhalation of the gasses. The rate of recovery appeared to be related to the magnitude of the original TTS_2, an observation which coincided closely with that of Ward, Glorig, and Sklar (1959) in their examination of TTS recovery. The finding that TTS recovery rate did not appear to depend on gas inhaled did not agree with the interpretation that Joglekar et al. (1977) made of their data. Those investigators considered that carbogen inhaled during recovery had a beneficial effect on recovery since their carbogen treated subjects recovered more rapidly than those inhaling air. However, an examination of the graphs provided by Joglekar et al. suggests that it is possible that they did not take into account the different levels of TTS at the beginning of the recovery period. Their graphs appear to show parallel recovery. Also, rather in contradiction to their interpretation that carbogen assists TTS recovery, Joglekar et al. did point out that recovery rate appeared to be similar whether carbogen was administered during recovery or only while the subject was exposed to the noise suggesting, as does the present study, that enhanced oxygen inhalation can reduce the level of TTS due to noise but may not assist in the recovery process. In a follow-up study of the Joglekar group's work in the same laboratory with human and chinchilla subjects, Witter et al. (1980) also considered that they had evidence that recovery from TTS was faster after inhalation of carbogen than after inhalation of air. In their case, the carbogen was administered for various periods 30 min before noise exposure. Again, it would seem possible that in their interpretation of results, they neglected to take into account the original TTS magnitudes when they compared rates of recovery. Their graphs also show parallel TTS recovery curves. It would seem, then, that although oxygen or carbogen inhalation can reduce TTS in the first place, resulting in faster recovery, breathing such gasses while recovering may have little effect. It is possible that recovery from TTS depends on processes other than oxygen dependent ones. The suggestion of Ylikoski and Lehtosalo (1985) that recovery from auditory fatigue may depend on replenishment of neuropeptides in the area of outer hair cells before these cells can regain functioning capacity after intense stimulation, a process not involving a direct supply of oxygen, is interesting.

The findings of this study suggest that some of the hearing loss effects from loud sound can be offset by inhalation of either oxygen or carbogen at the time of noise exposure but gas administration after the noise in an effort to speed recovery may not be effective. Since Fisch, Marata and Hossli (1976) have indicated that 30 min of oxygen inhalation increases the oxygen tension of human perilymph to its maximum extent, studies are needed to explore the possible prophylactic effect of longer periods of oxygen administration on the development of TTS. Information about a relationship between oxygen supply and TTS has practical implications. For instance, by monitoring and regulating oxygen levels in noisy industrial situations and even augmenting oxygen supplies for individuals, perhaps some of the effects of such noise on hearing could be ameliorated.

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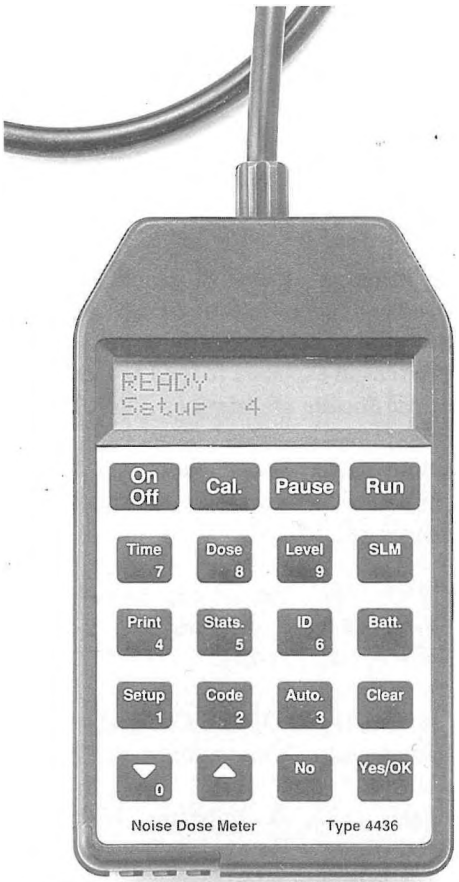

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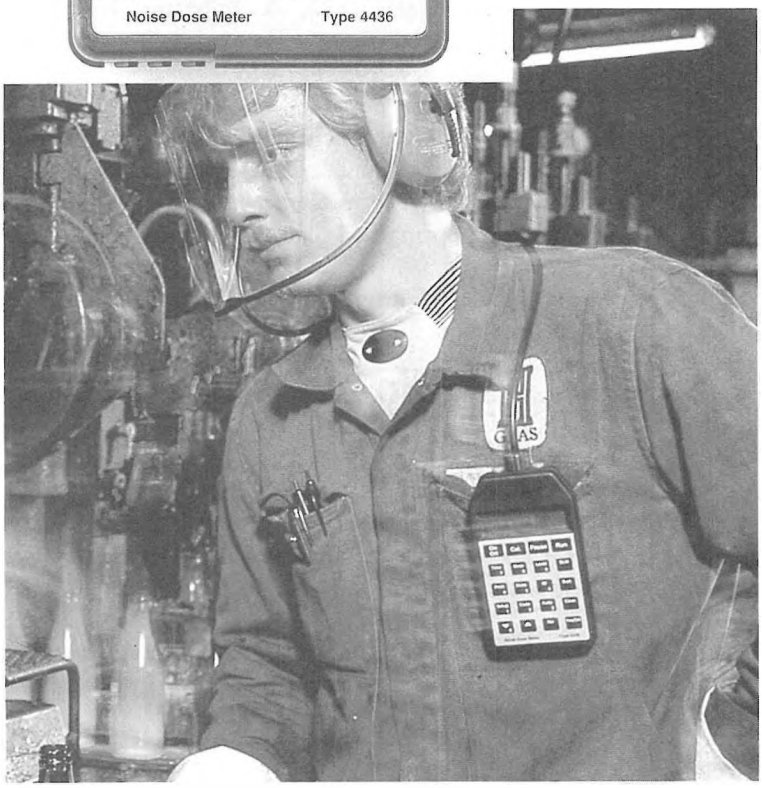

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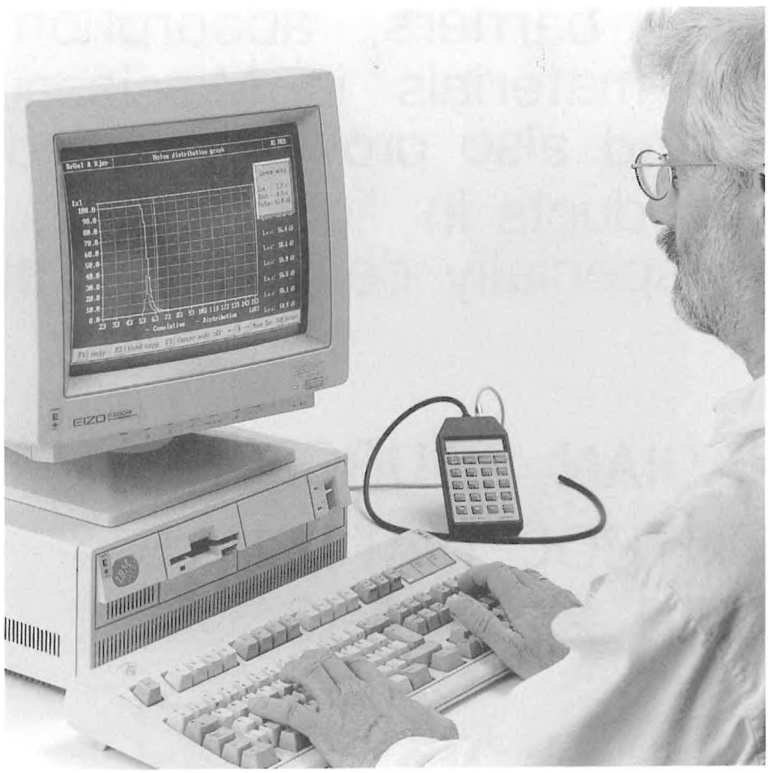

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