NON-ACOUSTIC ENVIRONMENTAL FACTOR INFLUENCES ON OCCUPATIONAL HEARING IMPAIRMENT: A PRELIMINARY DISCUSSION PAPER*

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ABSTRACT

Recently, several review papers have been published on environmental factors affecting hearing. But no mention was made of the ototoxic substances or agents to which industrial workers may be exposed apart from noise. This paper is a preliminary discussion of data on the numerous agents which ototoxicity is suspected or demonstrated on animals and on humans and on their probable site of disorder. These include carbon monoxide, heavy metals such as lead, arsenic and mercury, organic solvents such as toluene, xylene and stryrene. Allergenic chemicals and climatic conditions are also considered as being possibly associated with occupational middle ear dysfunction. Effects of vibration on hearing and synergistic interactions between noise and vibration are also examined. Research priorities are discussed in terms of the likelyhood of exposure and suspected toxicity. Based on known mechanisms of ototoxicity, it is suggested that potent nephrotoxic substances are also strong toxic agents to the inner ear. The risk of damage to the ear of the foetus from noise exposure of the pregnant woman is also considered. It is concluded that systematic investigation of potential ototoxic chemicals from the workplace should be conducted as it was done for drugs for which case studies showed damage to hearing.

SOMMAIRE

L'analyse des données disponibles concernant les facteurs de l'environnement pouvant affecter l'audition a récemment fait l'objet de plusieurs publications. Toutefois, mis à part le bruit, on n'y fait pratiquement aucune mention des substances ou agents ototoxiques auxquels les travailleurs industriels peuvent être exposés. Cet article constitue une analyse préliminaire

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des données sur l'ototoxicité présumée ou confirmée de plusieurs de ces substances ou agents ainsi que du site probable de lésion. Il est question du monoxide de carbone, des métaux lourds tels que le plomb, l'arsenic et le mercure, de solvents organiques tels que le toluène, le xylène et le styrène. Des substances allergènes ainsi que des conditions bio-climatiques sont mis en cause en regard des troubles de l'oreille moyenne d'origine professionnelle. Les effets des vibrations sur l'audition de même que leur action synergique avec le bruit sont également abordés. La probabilité d'exposition et le degré présumé de toxicité ont servi de base à la discussion de priorités de recherche. Des mécanismes connus d'ototoxicité permettent de poser l'hypothèse à l'effet que des substances fortement néphrotoxiques sont également de puissants agents toxiques pour l'oreille interne. L'analyse des données disponibles conduit à la conclusion qu'une étude systématique des produits chimiques potentiellement ototoxiques du milieu de travail mérite d'être entreprise comme ce fut le cas pour les médicaments pour lesquels des études de cas avaient révélé des atteintes auditives.

INTRODUCTION

Occupational hearing loss (OHL) has been studied for more than a century. One hundred years ago, Barr published an occupationnaly based comparative study of hearing loss (Barr, 1886). He considered excessive noise as the major toxic agent to the ear; but he noted that exposure to dust and fumes were also associated with hearing loss. Most contemporary studies of OHL have focused on the influence of noise exposure, ignoring or excluding other environmental causes of hearing impairment. The resulting bulk of data gives the impression that noise is the only possible ototoxic agent in the workplace. This view, shared by acousticians, otologists and audiologists, is clearly expressed in recent review papers on environmental factors affecting hearing (Mills and Going, 1982; Mills, 1985). Reviews of interactions between noise and other agents are somewhat limited to exposure to drugs that independantly affect hearing (Humes, 1984). Such an approach is restrictive; for example, in a well controlled epidemiological study of OHL in heavy industry, age and noise exposure accounted for only 40% of the variance of hearing levels (Phaneuf, 1982).

This paper presents a case for the necessity of consideration the effects of non-acoustic environmental factor influences on hearing loss in addition to that of noise exposure. The theoretical relationship of joint exposures has been described elsewhere (Phaneuf and Hétu, 1986). These contributions to the occurence of OHL can be additive or interactive, depending on the nature of the ototoxicity. The determinants to OHL other than noise are reviewed below. Recommendations are made for further research.

REVIEW OF EVIDENCE ON OTOTOXIC AGENTS OTHER THAN NOISE

The term ototoxicity in this paper refers to an adverse structural or functional effect of an agent on the auditory system, including the middle and the inner ear, the auditory pathways and the cortex. To affirm that occupational exposure to a substance or to an agent is ototoxic, two conditions are required: (a) it must induce a consistent acute and/or chronic response on the human auditory system and (b) this response can be reproduced experimentally in animals and observed in humans. Few agents actually meet these conditions but several can be seriously considered as possibly toxic to hearing in the context of occupational exposure.

Carbon monoxide

Several case reports have been published on acute and chronic effects of carbon monoxide on hearing (Lumio, 1948a, b; Wagemann, 1960; Kawata et al., 1964; Taniewski and Kugler, 1964; Zenk, 1965; Baker and Lilly, 1977). The hearing loss appears to be reversible in most cases and is associated with a central nervous system toxicity. In an extensive epidemiological investigation, CO was reported as a frequent cause of "industrial toxicosis" (Surgen et al., 1973). Laboratory exposure to 200 ppm during 4 hours failed to show any effect on the temporary threshold shift (TTS) induced by a loud tone exposure (Haider, 1973). However, auditory perception tasks appeared consistently sensitive to acute effects of exposure levels between 0.50 to 250 ppm during 2 to 5 hours (Beard and Wertheim, 1967, 1969; Groll-Knapp et al., 1972; Fodor and Winneke, 1972). Acute CO poisoning of guinea pigs demonstrated severe but reversible loss of auditory sensitivity which was clearly associated with cortical and to a lesser extent subcortical dysfunction (Makishima et al., 1977).

The possibility of a permanent damage to hearing should be tested on animals using low level chronic exposures to CO. A retrospective epidemiological study of workers exposed to carbon monoxide, after adjusting for noise exposure and age, may confirm laboratory experimentation. Several occupations involve low level chronic exposure to CO and high noise levels, including welders, foundry workers and diesel operators.

Heavy metals

Heavy metals are traditionally mentioned as possible ototoxic agents (see Quick, 1982) but very few studies have verified this possibility.

Hearing disability has been reported as a symptom associated with lead-poisoning (Ciurlo and Ottoboni, 1956). Two studies conducted on guinea pigs confirmed this contention, showing VIII nerve axonopathy, the inner ear appearing otherwise intact (Gozdzik-Zolnierkiewicz and Moszinski, 1969; Yamamura et al., 1984).

Inner ear disorders have been repeatedly observed following arsenical intoxication of animal subjects (Ruedi, 1951; Leonard et al., 1971; Anniko and Wersall, 1975). The damage seems to appear in

the stria vascularis followed by disorders in the various components of the organ of Corti. Sensorineural hearing loss (more pronounced in the low frequencies) has been measured in a significantly higher proportion of children exposed to environmental arsenic than in a group of controls. No comparable study have been conducted among the various groups of workers chronically exposed to arsenic (see Landrigan et al., 1982).

Mercury is considered as ototoxic from the results of the study on the people from the Minamata Bay in Japan who showed hearing losses in a large majority (Kurland e al., 1960). Results from a study on guinea pigs intoxicated with methyl mercury demonstrated considerable sensory cell destruction in the inner ear (Falk et al., 1974). A study of hearing loss among industrial workers has suggested a combined effect of noise and mercury exposure (Eggemann et al., 1977). Data on the effects of chronic exposure to other suspected toxic metals such as gold, zinc or manganese have not been found in this preliminary review of litterature.

Organic solvents

Case reports of organic solvent ototoxicity among industrial workers were published more than 30 years ago (Lehnardt, 1965) but experimental studies were initiated only recently. They originated from an extensive investigation of the possible damage resulting from solvent abuse among volunteer inhalers (Rebert et al., 1983). This led to a demonstration of the toxicity of toluene (1200 ppm, 14hr/day, 7 days/week for 5 weeks) on the sensory cells of the inner ear of new born and adult rats (Pryor et al., 1984). Comparable tests on xylene and styrene showed much higher toxicity of these two substances (Pryor and Rebert, 1984). Yet no epidemiological study on workers chronically exposed to these chemicals has been undertaken to assess their ototoxicity. Vestibular disorders have been reported among painters exposed to white spirit (Arlien-Soborg et al., 1981), but no data on hearing assessment is given. Results from cross-sectional studies on relatively small samples of workers suggest that carbon disulfide, carbon tetrachloride, trichlorethylene, and n-butanol induce sensorineural hearing loss (see Barregard and Axelsson, 1984).

Reviews of published data on the neurotoxicity of organic solvents suggest that sensorineural hearing loss can likely be an early indicator of organic solvents neuropathies (Chong, 1982; Spencer and Schaumburg, 1985).

Proved neurotoxicants such as trichlorethylene and carbon disulfide (Spencer and Schaumberg, 1985) may also interact with the effect of noise by acting on the VIIth cranial nerve, involved in the activation of the acoustic reflex. Noise would have more deleterious effects in the absence of the protection normally afforded by this reflex (Bobbin and Kiniel, 1980). Thus on physiological grounds, noise and solvents should produce synergistic interactions.

Allergenic chemicals

A number of substances, though not directly toxic to auditory system, may cause allergic reactions in the upper respiratory tract.

This may contribute to middle ear disorders such as chronic otitis media causing conductive hearing loss. Families of chemicals that are more frequently associated with these allergic reactions include formaldehyde, alcohols and phenols (Boyles, 1985). These substances are frequently found in the workplace. Formaldehyde is also suspected to be toxic to the inner ear (Quick, 1982).

No data are available to quantify the prevalence of OHL associated with respiratory allergies from exposure to chemical substances in the workplace. Nevertheless, this could account for the increased prevalence of conductive hearing loss among coal miners (NIOSH, 1976) as well as among foundry workers (Burns et al., 1977) exposed to dust particules and to vapors of metal scouring solutions. In a Quebec foundry, we have found 9 out 12 (75%) workers exposed to such vapors with middle ear disorders, whereas the proportion of the 212 workers not exposed to these irritants was 19%. Although it is not a conclusive proof as the cause of occupational middle ear disorder, this suggests its plausibility.

Bio-climatic conditions

In the same way as for allergic reactions, exposure to very humid and to cold temperature (as in refrigerated areas of the food industry) as well as contaminated recirculated air in buildings could result in occupational middle ear dysfunction or disease and conductive hearing loss. This is again an undocumented possibility, but we have received several reports of symptoms related to middle ear diseases from people employed in slaughterhouses exposed to cold, humid and possibly contaminated air.

Vibrations

Exposure to whole body vibrations appears to induce, at least in some conditions, a measurable TTS though of small magnitude (Kile and Wurzbach, 1980). Combined with noise, it has consistently been associated with increases in TTS (Okada et al., 1972; Yokoyama et al., 1974; Okada et al., 1984; Manninen, 1986).

A synergistic effect on permanent threshold shift was also observed in animals exposed to high levels of impulse noise (Hamernick et al., 1980) or to low levels of impact noise over several consecutive days (Hamernick et al., 1981). Considerable permanent damage to the middle and the inner ear have been observed in animals exposed to high intensity infrasound (Lim et al., 1982).

Few field studies have been conducted on the possible contribution of occupational vibration exposure to hearing impairment. Vestibular dysfunction has been reported in almost one out of two among a group of workers testing diesel engines and being exposed to 10 Hz vibrations (Aantaa et al., 1977) but no data on hearing was presented. The combined noise and vibration exposure appears to induce hearing losses that are more pronounced in the low frequencies than is the case for noise exposure alone (Sulkowski, 1980). In two well controlled studies in the lumbering industry, workers suffering from vibration induced white fingers had more hearing loss than

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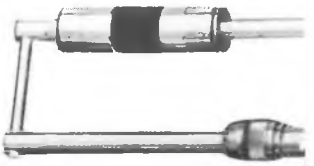

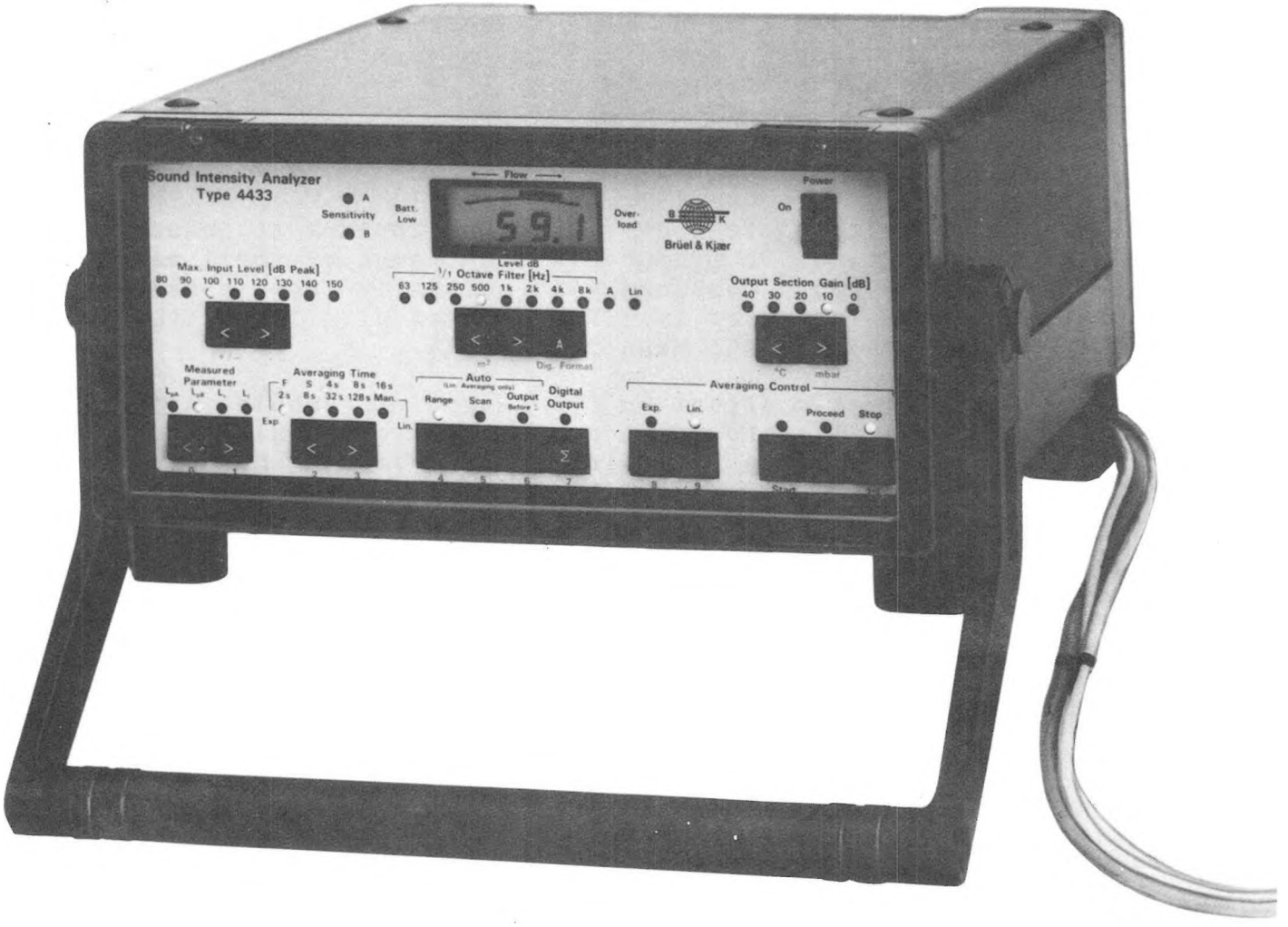

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controls exposed to the same cumulative amount of noise (Pyyko et al., 1981; Iki et al., 1985).

As vibration exposure is frequently associated with noise exposure in industry, it could account for a significant portion of the variance of hearing loss measured in cross-sectional studies.

The above review, summarized in Table 1, confirms the need to consider other agents than noise as environmental factors that may have adverse effects on hearing in the workplace. It also stresses the need for a systematic investigation of the various suspected ototoxic agents.

Table 1. Agents which ototoxicity is suspected (?), demonstrated on animals (*) and for humans (x) and their probable site of disorder.

Suspected agent	Probable site of disorder			
	middle ear	inner ear	auditory pathway	auditory cortex
Gases				
Carbon monoxide				x *
Heavy metals				
Lead			*	
Arsenic		x *		
Mercury		x *	?	?
Organic solvents				
Toluene		*		
Styrene		*		
Xylene		*		
White spirit (mixed solvents)		?		
Carbon disulfide		?		
Carbon tetrachloride		?		
Trichlorethylene		?		
n-butanol		?		
Allergenic chemicals				
Formaldehyde	?	?		
Alcohols	?			
Phenols	?			
Strong acids	x			
Climatic conditions				
Temperature variations	?			
cold damp air	?			
contaminated air	?			
Vibrations				
Whole body		x *		
hand/arm		x		

RESEARCH PRIORITIES AND STRATEGIES

In view of the number and the diversity of the agents that need to be investigated in order to confirm and characterize their potential ototoxicity, priorities should be defined. These could be based on the following two principles: (a) the substances or agents most widespread in the working environment should be considered first; (b) those for which presumed toxicity is more potent should receive priority.

The first principle calls upon knowledge of the industrial processes and statistics on exposures to the different agents considered. Industrial hygiene measurements may be helpful for determining the priorities. Stated in broad terms, substances such as organic solvents and heavy metals are sufficiently widespread to deserve consideration.

The second principle put forward raises more difficulties as it implies making assumptions on the damaging mechanism of the different agents suspected to be ototoxic. One assumption that we wish to propose is that substances that are potent nephrotoxics are also strong toxic agents for the inner ear. This is based on four line of reasoning:

1) Several substances that act upon the renal function also affect the inner ear and vice versa. It is the case for diuretic drugs (e.g. ethacrynic acid) that inhibit reabsorption in the loop of Henle; their effects are observed in the stria vascularis of the inner ear (Prazma, 1981a), causing temporary and, in some instances, permanent hearing losses. Mercurial diuretics also fall in this category. In addition, aminoglycosides, which are powerful toxicants to the inner ear, are also nephrotoxic (Prazma, 1981b).

2) The damaging power of proven ototoxic substances, such as aminoglycoside antibiotics and loop diuretics, strongly interact with renal dysfunction (Prazma, 1981a,b; Quick, 1982). Moreover, it is generally considered that loop diuretics can induce permanent damage to the inner ear only among those patients suffering from renal insufficiency.

3) Several of the suspected or confirmed ototoxic substances found in the workplace are nephrotoxic. It is the case for heavy metals such as lead and mercury (Buchet et al., 1980), and for organic solvents such as toluene, xylene and styrene (Askergen, 1984; Lauwerys et al., 1985). It is noteworthy that exposure in the workplace to such substances, which share the same sites of damage, are often combined (Orbaek et al., 2985; Winchester, 1985). This implies that they could be toxic even though each is present in concentrations below the permissible level.

4) Drug ototoxicity is a direct function of their renal clearance (Quick, 1982), which in turn depends on the integrity of the renal function. The same could be true for ototoxic chemicals from the workplace; moreover if the serum half-life of the substance is long enough, it could prevent complete elimination during the hours separating two work-days. This appears to be the case for toluene even under low levels of exposure (Brugnone et al., 1985). Accumulation in the blood could take place and a threshold of

ototoxicity could be reached when the renal clearance is insufficient.

One particular condition of possible ototoxicity of the working environment has been overlooked; it is the possible damage to the ear of the foetus from exposure of the pregnant mother at work (Lalande et al., 1986; Marien, 1986). Arsenic, lead, mercury and cadmium have been shown to cross, by passive diffusion, the placental barrier; it is also the case for liposoluble substances such as organic solvents (Barlow and Sullivan, 1982). The immaturity of the renal function probably accounts for the greater susceptibility of the foetus to ototoxic antibiotics (Henry, 1983). Consequently, it is urgent to undertake the investigation of the toxicity of the nephrotoxic agents of the working environment on the developing ear of the foetus.

From the above proposed priorities of research, strategies of investigation must be considered. Experimental studies on animal models should be developed as has been profitable with ototoxic drugs. This would allow one to confirm the ototoxicity, to obtain a dose-response curve, to estimate the relative potency and to understand the damaging mechanism of the suspected substances. This approach is essential knowing the relative insensitivity of epidemiological procedures to identify a more or less small excess of disease in the presence of many confounding factors. However, epidemiological data are necessary to validate the risk factors and the exposure limits and to explore the possible interactions with noise exposure. In a context where the neurotoxicity of substances such as solvents is of increasing concern, thus making it the object of numerous epidemiological studies, it is tempting to include hearing assessment in such studies. But the confounding variables involved differ with the outcome considered. The association between hearing loss and organic solvents exposure should be the object of specific study designs.

CONCLUSION

In conclusion, the above preliminary review of evidence provide sufficient grounds to contemplate occupational hearing loss in a different perspective. Noise is certainly a preponderant cause of OHL; but several other agents of the working environment can affect hearing in isolation or in conjunction with noise. Such a perspective calls for a new research effort in the field of occupational toxicology and epidemiology.

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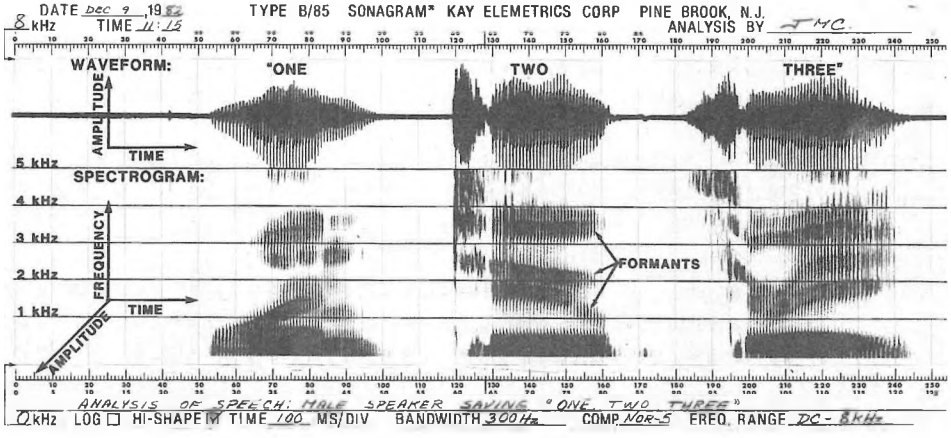
The Digital Sona-Graph® model 7800 is a DC-32kHz spectrum analyzer designed for the analysis and display of non-stationary signals (such signals include speech, Doppler shifts from Ultrasound and telemetry signals, sonar and radar returns, animal sounds, music, heart sounds or any sound that has frequency characteristics which change over time). The complex nature of these signals requires an analyzer which incorporates the features of a sound spectrograph, a waveform printer, signal splicer/editor and a spectrum analyzer.

SOUND SPECTROGRAPH

The 7800 captures and stores the input signal to a real time rate of 32kHz in a large 128K word (each word is 11 bits wide) solid state memory (RAMs). The 7800 can accept single or dual channel input. Once stored, the signal(s) can be analyzed (to a 60dB dynamic range), displayed and printed in many ways. The spectrographic display is most revealing for non-stationary signals which are characterized by spectral (frequency) changes over time. This 3-D display (amplitude vs. frequency vs. time) is shown to the right.

- **Print Resolution: 1000 (H) X 400 (V) X 23 Grey Scale Levels**



SIGNAL SPLICER/EDITOR

The large 128K word (by 11 bit) memory can record and store 5.12 seconds (in the 8kHz range) of input signal. The splicer/editor feature lets the user listen to or remove, selected portions of the audio signal or splice separate segments together.

AUDIO WAVEFORM PRINTER

Print the entire stored waveform or just selectively expand portions of the stored signal. Unlike expensive high speed oscillographs, the 7800 makes it easy to select only certain segments for display (see the sample Sonagram® above). A rapid yet significant transition can be easily isolated and viewed in detail. The waveform can also be printed with the corresponding spectrogram.

APPLICATIONS

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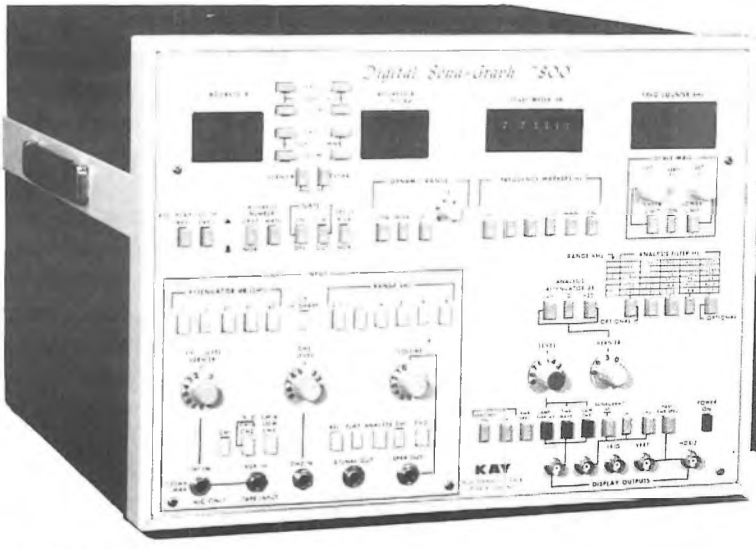

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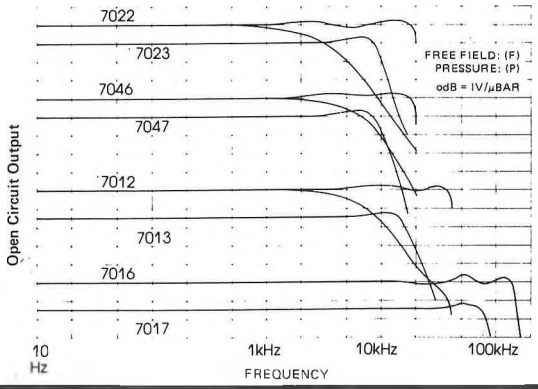

Cross Reference Table

ACO	B&K	GenRad	Ivie
7012	4133	1560-9532*	1133*
7013	4134	1560-9533*	1134*
7016	4135	1560-9534*	—
7017	4136	1560-9535*	—
7022	4145	—	—
7023	4144	—	—
7046	4165	—	—
7047	4166	—	—
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