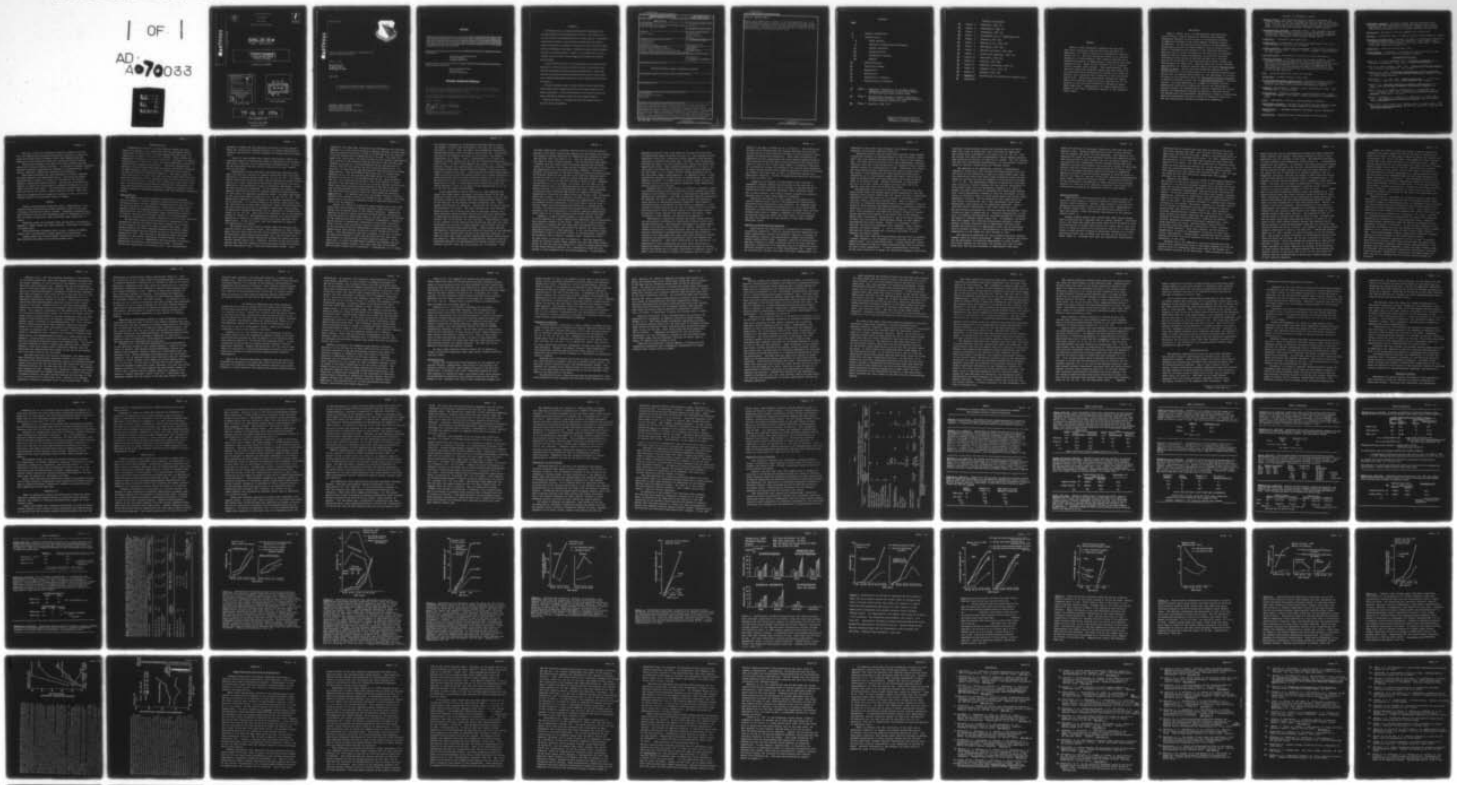
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EXTRA-AUDITORY HEALTH EFFECTS OF INDUSTRIAL NOISE: SURVEY OF FOREIGN LITERATURE

Bruce L. Welch

Welch Associates
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June 1979

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FOR THE COMMANDER

HENNING E. VON GIERKE
Director
Biodynamics and Bioengineering Division
Aerospace Medical Research Laboratory

FOREWORD

The following report is a summary and the author's interpretation of the European literature on the non-auditory effects of noise. The subject area is one in which there is considerable diversity of scientific opinions. Firm positions on the question of whether or not the typical noise exposures experienced by workers in industry or by residents near noise sources cause non-auditory effects have been established by some authors. Definitive conclusions might be premature on the basis of available evidence. However, it is our belief that, as with any scientific article, the report is clear enough that each reader can and should form his opinion.

It very will may be that there is a strong association of noise exposure with hypertension. This does not mean, however, that there is necessarily a cause and effect relationship between noise exposure and hypertension. Noise exposure may very well be associated with lifestyles that are more stressful in themselves. Further epidemiological and laboratory studies addressing these questions are required.

The present contractor report is issued to make the studies reviewed more widely available and to stimulate and facilitate a US research program on non-auditory noise effects. In view of the seriousness of the questions raised in the report, authoritative, definitive studies in this area appear mandatory.

Based on the report, it is not proposed to make any changes in practical exposure criteria and guidelines.

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Based on the literature reviewed in this report there is very substantial evidence that long-term work under industrial sound of 85-95 dBA and above is associated with chronic changes in cardiovascular function, mostly adverse. The best documented change is an impaired regulation of blood pressure, most commonly manifest as an increased prevalance of hypertension in the middle and latter decades of life. There is also evidence for an increased risk of ischemic heart disease. Very conservatively, long-term work under high intensity sound seems associated with an increased risk of cardiovascular disease of at least 60

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percent, quite possible with a doubling or more than doubling of risk. Functional neurological changes are common. Other extra-auditory changes are also reported but too few studies of them have been conducted to adequately assess their importance. Interpretation of these observations is subject to the usual constraints inherent in the epidemiological model.

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Summary

There is very substantial evidence that long-term work under industrial sound of 85-95 dBA and above is associated with chronic changes in cardiovascular function, mostly adverse. The best documented change is an impaired regulation of blood pressure, most commonly manifest as an increased prevalence of hypertension in the middle and latter decades of life. There is also evidence for an increased risk of ischemic heart disease. Very conservatively, long-term work under high intensity sound seems associated with an increased risk of cardiovascular disease of at least 60 percent, quite possible with a doubling or more than doubling of risk. Functional neurological changes are common. Other extra-auditory changes are also reported but too few studies of them have been conducted to adequately assess their importance. Interpretation of these observations is subject to the usual constraints inherent in the epidemiological model.

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Glossary for Nonmedical Readers

Angina pectoris - pain characterized by a sense of tightness and pressure around the chest and radiating from the area of the chest above the heart to the left shoulder and down the arm along the ulnar nerve; due to insufficient blood supply to the muscle of the heart. Common in people who have coronary (=ischemic) heart disease.

Asthenoneurotic syndrome - (astheno = weak) - East European term for complex syndrome including excessive feeling of fatigue, lethargy, irritability, often with complaints of headache and sleeplessness. In West would probably be called chronic anxiety state.

Bradycardia - excessively slow heart rate.

Bradyarhythmia - bradycardia, but often used when heart rate is not only excessively slow but also irregular.

Cardiac ischemia - insufficient blood supply to the muscles comprising the walls of the heart. (Cardiac = heart; ischemic = insufficiency of blood supply).

Cholesterolemia - Excessively high levels of blood cholosterol.

Coronary sclerosis - Sclerotic change in coronary arteries. Atherosclerosis involving the deposit of lipid, calcium and hyaline materials within and beneath the inner muscular coat of the arteries that supply the muscular wall of the heart, accompanied by the proliferation of fibrous connective tissue and resulting in the thickening and loss of elasticity and contractility of the walls of the coronary arteries.

Diel - occurring during the course of each day.

Dysrhythmia - Abnormal rhythm of the heart.

Electroencephalographic hyposynchrony - Abnormal asynchrony (lack of synchrony) in the recorded electroencephalogram. Asynchrony is normally associated with activation and behavioral arousal.

Essential (hypertension) - Without a known physiological cause. Most hypertension is thus described.

Extrasystoles - Premature heartbeats (occurring before their normal time in the rhythm of the heart); usually followed by a compensatory pause.

Frank - unmistakable, obvious, clearly manifest clinically.

Hyperdynamic response - increase in the heart rate and systolic blood pressure with increase in pulse pressure and cardiac output.

Hyperreflexia - Reflexes abnormally excitably, usually abnormally strong.

Hypertension - abnormally high blood pressure for sex and age.

Hypodynamic response - decrease in heart rate and systolic blood pressure with decreased pulse pressure and cardiac output, often superimposed upon (and likely in reflexive response to) elevated diastolic blood pressure due to increased cutaneous vascular tone.

Hypotension - abnormally low blood pressure for sex and age.

Lability - Instability; given to easy and erratic displacement or change.

Splanchnic vascular tone - tonicity, or degree of maintained contraction of muscular walls controlling the diameter of blood vessels that supply the viscera (major organs of the lower body cavity - stomach, intestines, panchreas, spleen, etc).

Tachycardia - excessively rapid heart rate.

Tachyarhythmia - tachycardia, but often used when heart rate is not only excessively fast but also markedly irregular.

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Hurst, J. W., R. B. Logue, R. C. Schlant and N. K. Wenger (Eds), 1974. The Heart, Arteries and Veins, McGraw-Hill, New York. (There may be more recent edition).

This is a review of accessible technical evidence, pro and con, for associating long-term occupational exposure to industrial sound with human health disorders other than impaired hearing. Attention is restricted to morbid and clearly pre-morbid change.

Circulatory disorders have been more often studied than any other kind of extra-auditory disorder. In studies of general morbidity the most consistent pathological findings are cardiovascular. This review, therefore, emphasizes cardiovascular morbidity.

Since most available data are published in foreign languages the most important are summarized herein. Data from key papers are presented in tables or graphs in a uniform way in order to simplify comparisons between studies. Statistical tests have been applied to verify the significance of apparent differences in most cases where the data permit. All statistical probabilities cited were derived by the reviewer unless otherwise stated. When data have been summarized from a paper its citation in the list of references directs to the relevant table or figure.

General

General extra-auditory morbidity tends, categorically, to be elevated among people who have been exposed at work for at least 3-5 years to sound of 85 dBA or greater. Morbidity increases more with advancing age and with increasing years of employment for both men and women if they work under relatively high intensities of sound.

Morbidity tends to be greater under non-periodic intermittent, impulse or impact sound than under periodic, continuous or relatively steady sound.

Noise-associated disorders tend to have a skewed or bimodal rather than a statistically normal population distribution.

People whose work involves mental concentration tend to be more affected than those who do mainly manual work.

Cardiovascular

Cardiovascular morbidity of one kind or another has been found to be greater among people who work for prolonged periods under high intensity sound than among people who work under low intensities of sound in 40 different studies. These studies are summarized in Figures 1-14 and in Tables 1-3. Of these, 21 studies provide data in a form that allows at least approximate statistical confirmation of apparent differences. Increased cardiovascular morbidity has been associated with long-range occupational exposure to high intensity sound in at least 11 different nations and in at least 18 different industries (Table 1). The data, conservatively interpreted, suggest that long-term work under high intensity sound is associated with an increased risk of cardiovascular disease of at least 60 percent, possibly with a doubling or more than doubling. No study involving appropriate measures and statistical analyses has been identified which failed to suggest an adverse cardiovascular effect of long-term employment under high intensity industrial sound.

Blood Pressure

The dominant and best documented concomitant of prolonged routine exposure to intense industrial sound is impaired regulation of blood pressure, the most distinct manifestation of which is an increased prevalence of hypertension. Chronically elevated blood pressure and/or an increased prevalence of hypertension has been associated with long-term employment under high-intensity sound in 27 studies, and the data accessible permit at least approximate statistical validation of apparent differences in 20 of these (Table 1).

In each study reported the definitions of hypertension and hypotension used by the author have been retained and are given in the legend of the figure or table to which they apply. Lower blood pressures are often considered hypertensive by the authors of reviewed papers than are defined as such by the World Health Organization (91). But this may be appropriate since the blood pressures of the general population in these reports is often relatively low (c.f. 2). Hypotension and hypertension are merely definitions of deviation from statistical norms. Clinically significant hypotention is relatively rare. But elevated blood

pressure is common and any sustained elevation of blood pressure above the conservative minimium adequate to sustain physiological function increases the risk of coronary and cerebrovascular disease (71).

Since the many studies that suggest hypertensive effects of long-term exposure to industrial sound are detailed in the figures and tables, they will not be individually discussed here. Details of the few studies that may seem at variance with them, however, will be discussed.

Hypertension is more strongly correlated with long-term work under intermittent, periodic or impulse sound than with work under continuous relatively stable sound (16, 19, 24, 30, 31). The former may be associated exclusively with strong hypertensive change whereas continuious stable sound may be associated concomitantly with both hypertensive and hypotensive change in different individuals within the same acoustical environment. Of the studies that report an apparent hypertensive effect of routine industrial sound exposure, six also report an apparent hypotensive effect on other individuals in the same noise-exposed population (14, 19, 20, 24, 27, 30). The hypotensive response is not necessarily an abiding characteristic of an individual. It is most common in relatively young people who are relatively new to work under industrial sound, and with advancing age and increasing duration of employment under noise the tendency to hypotension commonly diminishes and is replaced by a hypertensive trend (14, 19, 20, 24). This shift with advancing age is particularly evident in Figure 11. When hypotension develops in a noise exposed population it normally is quantitatively much less important than hypertension. It is, of course, much less important in terms of its implications for health.

Two studies have been cited as having shown exclusively hypotensive effects of industrial noise. But inspection of these reports shows that one dealt only with the initial period of work under noise for previously noise-naieve young people (21), and the other dealt mainly with temporal changes during the working day rather than with chronic abiding change (33). Moreover, the data in the latter paper clearly show that the lowering of blood pressure through the working day was superimposed upon a long-term hypertensive trend. Thus,

Volpikina (33) found that 300 weavers and 300 spinners who had been employed in a cord factory averaged a gradual decrease in blood pressure from systolic/diastolic values of 115/70 mm Hg at the start of a routine working day to 100/60 mm Hg at its end. For ages 20-39 years (194 individuals) the blood pressures were strongly hypotensive: 29.4 percent of the measures taken at the end of the work day were $<$90/60 mm Hg and 41. 8 percent $\leq$100/65 mm Hg, whereas only 6.2 percent were $\geq$ 135/70 mm Hg and none attained 140/80 mm Hg. By contrast, for ages 50-59 (186 individuals) blood pressures were more hypertensive: none were $\leq$90/60 mm Hg and only 9.7 percent were $\leq$100/65 mm Hg, but 29.0 percent $\geq$ 140/80 mm Hg, 19.4 percent $\geq$150/100 mm Hg and 4.5 percent $\geq$160/100 mm Hg; among spinners, who were more affected than weavers, fully 63.6 percent of blood pressures were $\geq$ 140/80 mm Hg and 36.4 percent were $\geq$ 150/100 mm Hg. Since these blood pressures were measured at the end of the work day and had declined from pre-work day values 10-15 mm Hg higher, the long-term blood pressure trend for these women was strongly hypertensive. It reflects more than the modest increase in blood pressure that normally occurs with advancing age. Clearly, the daily hypotensive trend was not associated with a long-term hypotensive trend, but the opposite.

Kachny (14, Table 2) studied 591 young Russian women (age 15-27 years) who tended automatic weaving machines under continuous 102-108 dB high frequency sound in two textile factories. Hypotension (defined as systolic and/or diastolic $<$ 100/55 mm Hg) developed in fully 50 percent of these young women in one mill and in 64.7 percent in the other during their first 1-6 months of employment. However, the prevalence of hypotension diminished to just under 10 percent after 5 years on the job and to 8 percent within 10 or more years on the job paralleling the commencement a modest hypertensive trend (hypertension being defined as systolic and/or diastolic $>$ 140/90 mm Hg). This hypertensive trend was strongest in the mill in which the women were exposed to about 102 dB sound and were also under the pressure of concomitantly attending several machines. In the other mill, in which the hypotensive tendency was greatest and the hypertensive tendency weakest, the sound level was 4-6 dB higher but the women tended only 2-3 machines. I constructed a 3 x 3 table

for brachial hypotension, normotension and hypertension versus 1-6 months, 1-5 years and 6-10 years on the job, and I computed Chi square = 47.9: thus, the shift from predominant hypotension towards hypertension with increasing time on the job - even within the narrow and youthful age range of 15-27 years - was highly significant ($p < .0001$). Also, I constructed a 5 x 2 table for temporal arterial pressure of 40, 50, 60, 70 and 80 mm Hg versus work in Mill 1 or Mill 2 and computed Chi square = 20 : thus, temporal arterial pressure was significantly higher in the mill in which the women worked under 102 dB sound and tended a relatively large number of machines ($p<.001$). In both mills the strong hypotensive trend initially evident had largely abated and a modest hypertensive trend had become evident by the relatively young age of 27 years. The possibility that a substantial excess prevalence of hypertension might become evident in these populations during the middle and latter decades of life, as in other textile populations (2, 5, 14, 15, 22, 30, 33) cannot be precluded.

A yet unpublished study by Cohen et al. (40) suggests that people who worked under continuous relatively steady 85-102 dBA sound in a paper mill in the midwestern United States, and who also had severe hearing impairment, had lower blood pressure than men of the same age in the general national population (65). Their sample included only 51 subjects; average age was 47 years, average job experience 22.3 years. For their average age, their systolic blood pressure was 10 mm Hg less than the national average (65), their diastolic pressure about 7 mm Hg less, and their prevalence of hypertension (medicated and unmedicated combined) about one-quarter less (17.6 percent vs. 24.2 percent). Concomitantly, Cohen et al. studied 51 other subjects who shared the same work and noise environment but who were 13 years younger, had 10 years less job experience and possessed normal hearing. Their average systolic and diastolic blood pressures were virtually identical to the national norm for their average age, and were also virtually identical to those of their much older and more experienced cohorts whose hearing was impaired. Their prevalence of hypertension (medicated and unmedicated combined) was approximately 50 percent greater than the national norm for their age (17.6 percent vs. 11 percent). None

of these comparisons to national averages can be said significant because of the small sample sizes employed in this study, and because the national averages selected for comparison are sensitive to sex, age and race (65), and Cohen et al. give no information on sex, age range or race of their subjects. For the estimates made above, I assumed that the range around the stated average age was small (which is unlikely), and I used national average prevalence figures for hypertension for races and both sexes combined. Certainly a systolic blood pressure of 160 mm Hg and/or a diastolic blood pressure of 95 mm Hg, which are the cut-off levels used for defining hypertension by Cohen et al., would be viewed with much greater alarm in a person 34 years of age than for one 47 years of age. Although the prevalence of hypertension, as defined, was the same for the middle-aged subjects with impaired hearing and the young men with normal hearing, it was not functionally the same. Blood pressures of systolic and/or diastolic $\geq$ 140/90 are commonly considered hypertensive clinically for people below age 40 years (94), certainly below age 35 years. If 17.6 percent of Cohen et al.'s subjects of average age 34 years had blood pressures $\geq$ 160/95 mm Hg or were on antihypertensive medication, it is very likely that at least a similar number had blood pressures $\geq$ 140/90 but $<$ 160/95 mm Hg. In short, the possibility cannot be excluded from the limited data available that the prevalence of clinically important hypertension among Cohen et al.'s young subjects with normal hearing was high.

Notably, a study of blood pressure in the specific context of noise exposure in a paper making plant apparently has not previously been reported anywhere in the world. Cohen et al.'s data could be interpreted to suggest that the paper mill environment gave their hearing-impaired subjects some protection against the usual ageing-associated increase in blood pressure. Is so, the protection could conceivably be due to the sound itself, or alternatively to another environmental factor (Figure 6 suggests such a protective effect of some factor in the gas and oil industry). But that hypothesis cannot be accepted unless larger samples are taken and found unequovically to show not only the absence of a trend to excess hypertension in the normal-hearing subjects, but a hypotensive trend for them similar to that suggested for the hearing-impaired. An alternative

hypothesis would be that noise tends to increase the prevalence of hypertension among those who can hear it; that extreme hearing impairment itself has a protective effect against blood pressure elevation by shutting the noise out; and that, once established, deafness may even favor the regression of a previously established hypertensive trend. Other possible explainations, of course, are that paper mill noise had no long-term effect on blood pressure, or that Cohen et al.'s results reflect chance abberation due to the very small sample sizes employed; or technical error. A more substantial extension of Cohen et al.'s investigation and a more complete reporting is needed before it can be judged to have any relevance to the question of whether industrial noise importantly affects cardiovascular health.

Attempts have been made recently to discount a possible causal relationship between long-term industrial noise exposure and hypertension on the grounds of a failure to find hearing impairment and hypertension correlated. That such correlation need not necessarily be expected, and that a failure to find it does not necessarily argue against an association between industrial noise exposure and hypertension is discussed in some detail in Appendix I. Also, studies of middle-aged men in the City of Uppsala (45-47) that have been purported to offer evidence against an association between occupational noise exposure and hypertension, but which I judge irrelevant to that subject, are discussed in Appendix I.

Expressions of population average and median blood pressures are not sensitive indicators of hypertensive trends within noise exposed populations. The population distribution of hypertensive change is not statistically normal, but skewed. Moreover, the bimodal distribution of blood pressures that may be created by concomitant hypertensive and hypotensive pressures upon different sectors of the same population under some noise conditions further minimizes the sensitivity of analytical tools that compare only central tendencies . That major differences in the prevalence of hypertension and/or hypotension may exist where differences in population median or average blood pressures are small or statistically non-significant is vividly illustrated in Figures 2 and 8, in Table 3 and in the data of Jonsson and Hansson, and to a lesser

extent in the data of Takala et al., in Table 2. Such differences in data can be detected only by focusing analysis directly upon the prevalence of distributional extremes of blood pressure, hypertension and hypotension themselves. Since major differences in hypertension and/or hypotension may exist between compared populations although average and median blood pressures show little difference or none at all, studies that report a negligible difference or no difference between the medians or the average blood pressures of noise-exposed and non-noise exposed populations cannot be accepted as evidence that there was, in fact, no difference between the populations compared. A study by Folprechtova-Stenzlova and Janicek is a case in point (70).

Inspection of Figures 1-14 and Tables 2 and 3 reveals a remarkably uniform tendency from one study to the next for an elevation of blood pressure and an increase in the prevalence of hypertension with long-term employment under industrial noise. All of these studies, with several notable exceptions in Table 2, employed large or very large numbers of subjects.

Capellini and Maroni (4), in a study of 1365 people in an Italian chemical plant, did not find hypertension significantly associated with occupational noise exposure; but ischemic heart disease was significantly associated with it, as is noted below. Otherwise, no well-designed and adequately analyzed study has been located which fails to suggest a tendency for blood pressure to be elevated during the middle and later years of life in people who have been employed for long periods of time under intense industrial sound.

Lability of Cardiovascular Responses

Hypertension and hypotension are fundamentally disorders of circulatory regulation. Both are characterized by exaggerated and often inappropriate cardiac and vasomotor responses to changes in body position, physical and psychological stimuli and autonomic nervous system active drugs. Moreover, lability of blood pressure and heart function is often considered one of the first and most definitive indications of an impending loss of circulatory control that may ultimately result in frank hypertension (92). Hence, it is of particular interest that one of the most characteristic con-

comitants of work under high intensity sound appears to be an enhanced cardiovascular lability (Table 1).

Enhanced vascular lability under high intensity sound assures a decreased adequacy and appropriateness of the circulatory adjustments that must normally be made during the course of the working day. For those whose circulation is already compromised, excessive lability favors the acute precipitation of congestive heart failure, cardiac ischemia or cerebrovascular stroke. Pokrovskii (24, Figure 10) found blood pressure fluctuations over twice as great during the course of the working day for hypertensive and hypotensive individuals as for normotensive people when under constant 85 dB sound in machine shops, and this difference was further enhanced under 95 dB impulse sound. Extreme tachycardia or bradycardia was five times more common for people working under the 95 dB impulse sound than under the 85 dB steady sound.

My impression from review of the studies cited here and numerous others which say nothing about chronic pathology is that the dominant tonic vascular change over the course of a day of physically undemanding work under noise is an increase in cutaneous vascular tone and, likely, splanchnic vascular tone. This is commonly reflected in a modestly increased diastolic blood pressure, although diastolic blood pressure may remain unchanged or actually be lowered, particularly if the work involves muscular exertion; related to this, total peripherial resistance may either be raised or lowered, likely depending upon whether or not blood flow to the skeletal muscle mass is increased. Categorically, one of two basic patterns of heart rate and systolic blood pressure change is then commonly superimposed this basic change: either (i) a decrease in heart rate and systolic blood pressure, with consequent drop in pulse pressure and cardiac output; or (ii) an increase in heart rate and systolic blood pressure and a resultant increment in pulse pressure and cardiac output.

When the heart rate and blood pressure decrease this is probably a reflex response to the increased tone in cutaneous and splanchnic vascular beds. But it may involve more. In many instances, cardiac output and minute volume are clearly depressed more than is necessary for mere reflex compensation. A tonic parasympathetic restraint of the heart sufficient to impair the ability for adequate circulatory

regulation during postural change and exercise may occur during work under noise in people who, from long years of experience, have become fully accustomed to the noise (c.f. 26, 30, 34). One may presume that such tonic parasympathetic drive actually benefits some hypertensive persons, at least up to a point. But, for individuals who have established atherosclerosis, it could poise a distinct threat of vascular insufficiency and ischemic change.

The opposite of the hypodynamic response, a hyperdynamic response in which heart rate and systolic blood pressure rise over the course of the working day, seems to be more common under varying intermittent sound than under steady continuous sound and more common in contexts that may be supposed to favor emotional arousal (c.f. 27, 64). The hyperdynamic response is apparently associated with a relatively greater tendency for the eventual development of frank hypertension than the hypodynamic response (c.f. Table 3). Indeed, one might speculate on reasonably well established grounds that a daily hyperdynamic response to noise at work might be more likely to eventually result in fixed hypertension than would a daily hypodynamic response. But the possibility that hypertension may also evolve in association with a diel hypodynamic trend under occupational noise should not be prematurely dismissed. While a long-term chronic hypertensive trend under industrial noise may be presumed exacerbated by a routine work-day hyperdynamic trend, it does not seem to depend upon it. Essential hypertension, in general, is commonly characterized by increased peripherial resistance and normal or reduced cardiac output (92). In aggegrate, the available data suggest that long-term occupational exposure to noise, whether routinely associated with a hypodynamic vascular trend through the working day or a hypodynamic trend, may be associated with an eventual increase in the probability of developing frank hypertension in the middle and later decades of life.

The underlying mechanisms for these dynamic long-term vascular changes cannot be readily postulated. Indeed, the factors that underlie the development of essential hypertension in other contexts than noise exposure are not known, and perhaps even less is understood about the putitive evolution of hypertension from long-

maintained extremes of neurogenic stimulation than from any other apparent cause. Essential hypertension is not a single disorder but the possible end result of many disorders. The changes in the regulated set point for blood pressure control that apparently occur over long periods of time in people who are routinely exposed to occupational noise undoubtedly involve altered nervous and neuro-endocrine activity and metabolic and structural changes of many kinds. Chronic long-term reductions of stroke volume of the heart, such as the 20-35 percent decreases reported by Sanova in compressor operators with $\geq$ 11 years of routine exposure to 87-98 dB sound on the job (26, Table 2), may well reflect more than mere parasympathetic bias, namely, impairing structural change. A detailed discussion of the physiological concomitants of acute and short-term vascular response to sound and their possible etiological singificance for cardiovascular pathology is beyond the scope of this review.

Cardiac morbidity

Remarkably few efforts have been made to carefully assess the potential relationship of long-term industrial noise exposure to chronic cardiac morbidity. However, such studies as have directly addressed the possibility favor an increased risk. Approximately 13 studies suggest this. Six of them have data accessible in a form that permits a rough statistical confirmation of the apparent results (Table 1).

Several studies suggest that routine work under intense sound may be associated with increased risk of cardiac ischemia. Kalicinski et al. (15) found that women who worked under 95-105 dB sound as spinners and weavers in Polish textile mills for longer than 6 years were up to 1.8 times as likely to be hypertensive and up to 6.3 times more likely to show distinct evidence of myocardial ischemia on a resting electrocardiogram than co-workers who had been on the job only 1-6 years (Table 2). Although their data are significant statistically,

justification for generalizing from them is limited by the fact that their subjects were few - only 31 with 6 or fewer years job experience and only 109 with more. However, the potential merit of their findings is enhanced by the facts: (i) that, regardless of job experience, all of the women were within the same narrow age range of 47-51 years; (ii) that the prevalence of ischemic change among the more experienced workers was, by any standard, high; (iii) that the electrocardiographic signs of ischemia were correlated with complaints of pain typical for coronary disease; and (iv) that all women who had left ventricular hypertrophy were a prior excluded from the study.

Capellini and Maroni (4), in a study of 1346 people comprising 98.6 percent of the total workforce of an Italian chemical plant, claimed that clinically confirmed ischemic heart disease was correlated with routine occupational exposure to 85-95 dB sound at a high level of statistical significance, and with no other environmental factor. It was not significantly correlated with chemical exposure, temperature, vibration or illumination, nor with emotional distress, physical exertion, work rhythmn, work schedule, repetitiveness of work or pay level. The expected incidence of ischemic heart disease, according to the authors' derivation, suggests an increased risk of 2.5 or greater of developing coronary heart disease for persons working under this 85-95 dB sound (Figure 12). This intensity of sound, paranthetically, brackets the 90 dBA level that is presently allowed for routine eight-hour per day exposure for a working lifetime in United States industries. Capellini and Maroni's clinical diagnostic procedures seem amply sophisticated. Their study population was large. And they seem to have tried to rigoriously weigh the potential contribution of other variables to the effect observed. Their data, however, are not given in enough detail to permit an independent evaluation of their statistical significance. Interestingly, hypertension was not significantly correlated with noise exposure or with any other variable considered in this study.

Cuesdean et al. (6) conducted a very well designed and technically quite sophisticated study of 160 people exposed to 85-106 dB sound at work in a Roumanian rubber plant and of 160 matched non-noise-exposed controls in the same plant. They screened out persons

having familial risk of cardiovascular disease and amply recognized other standard risk factors. Individuals exposed at work to toxic substances were not included in the study. The observed correlates of noise exposure seem independent of differences in environmental temperature and humidity: for instance, the group having the highest prevalence of hypertension and the most electrocardiographic anomolies was exposed to more intense noise than any other subjects in the study, but not to temperature or humidity extremes, and, as compressor operators, they did not engage in heavy labor and should not have been exposed to inordinate amounts of airborne dust. All subjects, control and noise-exposed alike, engaged in light manual labor. The data, much of which is summarized in Figure 13, indicates various repolarization and conduction anomolies in the resting electrocardiogram of people exposed to 85-95 dB sound which may be considered borderline between normal and pathological; for people working under the higher intensity sound of 95-106 dB there was a significant increase in the prevalence of hypertension and of cardiac hypertrophy and the electrocardiographic anomolies noted under the lower intensities of noise were accentuated. However, no organic lesions were reported. And prolongation of the electrocardiographic QRS interval suggestive of retarded intraventricular conductivity was actually significantly less common among the noise exposed subjects than among controls. The populations were relatively young, 65 percent less than 41 years of age; controls average one years older than noise exposed subjects, 35 years vs. 36 years of age, which biased slightly against the obtained results. The prevalence of electrocardiographic anomolies was about the same above and below the age of 40 years: for instance, the repolarization anomalie identified in Figure 13 as Code 9-2-1 - which was found by Parsie et al. (68), earlier, in only 2.4 percent of 49,512 healthy men - was evident in about 40 percent of noise-exposed subjects both above and below age 40 years, but in only 5 percent of non-noise-exposed controls. Since the average job experience of these people was only 7 years (6 years below age 40, 16 years above age 40), it seems reasonable to expect important differences in the incidence of frank cardiac pathology to emerge between the noise-exposed and control subjects as the years pass on the job and the population achieves the more advanced ages at which overt cardiac pathology is most commonly expressed.

Jansen (12, Table 2) found the relative risk of cardiac dysrhythmias for people employed in 90-115 dB zones of German steel mills to be about 1.6 times that in 65-90 dB zones (24.1 percent vs. 15.8 percent, $X^2 = 11.8$, $p < .001$). His sample was large, 669 men in the more noisy zones and 336 in the less noisy zones. People having chronic disease or psycho-emotional factors in their personal history judged to put them at risk for cardiovascular disorders were excluded from the study. Jansen also found a difference in the prevalence of cardiac dysrhythmias of similar magnitude among men who worked under comparable high and low sound intensities in mines (27 percent vs. 17 percent); this difference, however, was not significant statistically, apparently because of the relatively small sized samples (125 men in high noise, 76 in low noise zones). Dysrhythmias included extrasystoles, tachycardia and other undefined rhythmic anomalies. Extrasystoles and tachycardia may either of relatively benign atrial origin or may result from premature ventricular contractions.

Shatalov et al. (30) found, in 144 people working under 85-95 dB sound in a Russian spinning mill and in 86 people working under 114-120 dB sound in a ballbearing plant, an uncommonly high prevalance of electrocardiograms that evidenced retarded interventracular conductivity or near-retardation (0.10-0.11 sec); this anomalie was significantly more common in the spinning mill than in the more noisy ballbearing plant (38 percent vs. 9 percent, $X^2 = 22.6$, $p < .001$), an intriguing observation, particularly in light of the analagous difference in Cuesdean et al.'s data mentioned above. Retarded intraventracular conductivity does not reflect an immediate direct neurogenic influence on the heart. It may reflect conduction fiber damage but frequently occurs in the absence of organic heart disease. Shatalov et al. noted few cases of overt organic heart disease. The electrocardiographic changes reported were observed in people having no organic disease of the cardiovascular system.
The degree of retardation reported (to 0.10-0.11 sec) is normally associated with partial right bundle branch block but could result from an electrolyte imbalance, particularly hyperpotassemia. This suggests that the people employed in these two noisy factories were at increased risk for premature ventricular contractions, which may cause ventricular tachycardia, fibrillation and sudden death, but that risk was not enhanced most in the most noisy of these two factories.

Shatalov et al. (30) also observed depression of the T-wave in the electrocardiogram, which may suggest enhanced susceptibility to cardiac ischemia, in 11-13 percent of the subjects in their two factories before beginning the working day, and such depression was 2-3 times more common after exercise. Moreover, in the spinning mill this electrocardiographic suggestion of mild cardiac ischemia was twice as common at rest after the end of the work day as it had been before beginning the day, and in the ballbearing plant its frequency quadrupled by the end of the day. Unfortunately these end-of-day electrocardiographic data were taken on only a small portion of the total subjects (Table 2), but they showed highly significant differences that are consistent with data reported by others. This apparent accentuation of the vunerability of the heart to circulatory insufficiency towards the end of the normal workday under noise was paralleled by a distinct bradycardia which was most common in the noisiest plant (27 percent of subjects in the ball-bearing plant, 18 percent in the spinning mill, $X^2 = 12.2$, $p < .001$) and by a declining systolic blood pressure, which was also most common in the noisiest plant (18 percent in ballbearing plant, 7 percent in spinning mill, $X^2 = 8.4$, $p < .01$). These changes were common in combination with a tendency for retarded intraventricular conductivity, and bradyarhythmia occurred in a large proportion of subjects. Blood pressure recovery from exercise tests was slow and the adjustments sometimes erratic and inappropriate. The hearts of people employed in both of these noisy factories had a reduced ability to cope with varying normal functional demands, and this deficiency was significantly more common at the end of the working day in the most noisy of the two plants.

The same basic point was made by Yazbursakis (34) based upon more definitive and much more extensive studies (1790 electrocardiograms and 504 blood pressure measures) made on 36 ostensibly healthy young (21-42 year old) men and women who had worked with 100-160 dB ultrasound devices for 4-5 hours per day for 2-5 years. The subjects wore earplugs at work. Whereas only 2 subjects evidenced rectilinear electrocardiographic S-T segment depression of at least 1 mm during a standard exercise test given before starting the work day, 12 did so in an exercise test given after work; whereas no S-T segment depression exceeded 1 mm before work, 7 did so after work. These

differences are statistically highly significant (Table 2). Just as in Shatalov et al.'s study, these electrocardiographic indications of enhanced myocardial vunerability to ischemia at the end of routine physically undemanding work under noise were paralleled by a distinct progressive bradycardia and lowering of systolic blood pressure and by a marked impairment of the ability to compensate normally for the circulatory demands of exercise. Correlating with objective electrocardiographic signs, 33 percent of the subjects complained of unpleasant sensations in the heart region; in Shatalov et al.'s study fully 43.3 percent of subjects were said to make such complaints, in Cuesdean et al.'s study 35 percent of the noise exposed subjects complained of palpitations and 33 percent of precordial pain judged non-anginal, and Kalicinski's subjects with electrocardiographic signs of cardiac ischemia complained of pain typical of coronary disease.

Observations intriguingly similar to some of Shatalov et al.'s were made by Terentev (32) on 90 air line service engineers and technical personal who were exposed to aircraft engine noise of 100-136 dB for 3-6 hours per day for 2-3 times per week, sometimes daily: common, particularly near the end of the work period were depressed or inverted T-waves in the electrocardiogram, increased diastolic blood pressure, decreased pulse pressure, complaints of heart pain, a dominant trend to bradycardia (interrupted by episodes of tachycardia) and cardiovascular overreactivity to exercise.

Stasiow et al. (36) reported seemingly relevant observations on men operating automatic coal washing machines. Seven of 30 subjects, 23 percent, had resting electrocardiographic changes consistent with myocardial ischemic change and classifiable under the Minnesota Code. Before beginning the working day, the electrocardiogram of 5 of 30 subjects showed repolarization anomalies indicative of mild to severe ischemia (S-T segment and T-wave changes coded 4-1, 4-3 or 5-3). At the end of a routine day of work exposure to 86-102 dB broadband sound plus slight vibration, but with very little physical exertion, 4 of these subjects had such changes (code 4-2 or 4-3) and 3 subjects had S-T segment elevations commonly interpreted to suggest epicardial lesion (code 9-2). Individual vasoregulatory trends over the course of the day were similar to those

elicited more rapidly in the same individuals by a standard cold pressor test. Over the work day under noise, diastolic blood pressure increased somewhat in 71 percent of the subjects and progressive bradycardia was evident in 65 percent, while systolic blood pressure exhibited variable response, resulting in a dominant overall trend for a decline of pulse pressure and, obviously, of cardiac output. Fully 80 percent of these subjects manifest spastic contraction of retinal arterioles at the end of the normal working day.

It should be emphasized that these responses apparently occur daily in people who have become fully accustomed to the noise experienced on their job through repeated exposure over periods of years. Sanova (26) reported that the stroke volume of the heart decreased progressively with increasing years on the job (20 percent in the first year, 35 percent by 20 years), paralleling a distinct hypertensive trend with advancing age (Table 2), in 144 compressor operators exposed to 87-98 dB sound(ranging up to 90-110 dB in the 16 Hz "infrasonic" range); there were frequent complaints of pain in the region of the heart.

Kangelari et al. (17) claimed that the three-year incidence of angina pectoris for 152 locksmiths exposed to 80-90 dB low frequency sound was exceeded by only 10 percent in 135 mechanics whose work entailed exposure to 116-120 dB high frequency sound; it was exceeded by 20-30 percent in foundary workers exposed to 110 dB sound plus vibration. Maugeri and Odescalchi (37) are said to have shown an accentuation of electrocardiographic anomalies after long-term noise exposure in people who had unquestionably diagnosed cardiopathies.

Some of the electrocardiographic responses just discussed can be placed in perspective by noting that Doyle and Kinch (69) found that only 10.2 percent of people had abnormal exercise electrocardiograms in a large survey of 7023 people with normal resting electro-

cardiograms. By contrast, the frequency among Yazaburski's ultrasound device operators, if one accepts S-T segment depression of $\geq$1 mm as abnormal, was 6 percent before work and 33 percent after, or, if one accepts only S-T segment depression of $>$ 1 mm as abnormal, zero before beginning the work day and 19.4 percent at its end - end-of-the-day values that exceed normal expectation by a factor of 2-3. Recall also that Kalicinski, accepting only S-T segement depression of $>$ 1 mm as the criterion for ischemia, found that of the women who had been on the job over 6 years, 17 percent had abnormal electrocardiograms at rest, a frequency higher by 70 percent than Doyle and Kinch found under stress loading in their extensive survey of normal people. In the Framingham study, the resting electrocardiogram was perfectly normal in two-thirds of of subjects who were soon to develop frank coronary heart disease and in half who had document ed angina pectoris (72). On the other hand, false positive electrocardiograms frequently occur in perfectly normal individuals, both during exercise and at rest. But a positive resting electrocardiogram, particularly when it occurs in conjunction with anginal pain, as in Kalicinski's study, is not to be ignored. And a positive exercise stress test is one of the most powerful predictors of coronary heart disease: people who are otherwise asymptomatic but have an abnormal exercise electrocardiogram have a fifteenfold greater risk of developing coronary heart disease than people who show no negative stress test (73).

Meinhart and Renker (20) compared cardiovascular morbidity statistics of all 807 men in the East German district of Halle who had been recognized as having neurosensory hearing loss ensuant to long-term exposure to industrial noise with those of 3948 male patients in ambulatory clinics of the same district. Coronary sclerosis and all myocardial disease except rheumatic and congenital heart diseases were, as a group, all ages considered together, twice as common in the noise injured men as in the clinic patients. The authors claim the difference highly significant statistically. But my analysis suggests that it is not statistically significant, and there was no significant difference in the prevalence of functional cardiovascular disorders or peripherial vascular disease. The noise exposed subjects did, however, greatly exceed the general clinic population, and significantly so, in both hypertension and hypotension, particularly the former (Figure 11).

Graff et al. (11) compared 117 Germans who had worked for about eight years under 95-110 dB middle and high frequency impulse noise of heavy machinery with 50 others matched for age, educational background and experience who were subjected to little noise at work. The proportion of high noise workers with hypertension was three times that of low noise workers (36 percent vs. 12 percent, $X^2 = 8.64$, $p < .01$), and the proportion having "heart and other blood pressure problems" was 2.8 times as great (33.3 percent vs. 12 percent, $X^2 = 8.1$, $p < .01$). Persons with a family history of hypertension were excluded from the study (Table 2).

Dokukina et al. (7) reported "abnormalities of cardiac function" to be 2-4 times more frequent, and "abnormalities of blood pressure regulation" 5-8 times more frequent, among 144 women who worked under 112-122 dB high frequency sound in electrical stations of metallurgical factories, and also among 390 women who experienced similar intensities of low frequency sound plus vibration on platforms in cement factories, than among 446 female grocery produce store and warehouse workers who had no uncommon occupational exposure to sound. For 89 other women on these noisy jobs who were considered to have "noise disease", these differences were further increased by a factor of 2-4. Most of these women were in their mid-thirties and had 7-14 years of job experience. The controls averaged 1-4 years older than the noise-exposed women, thus biasing against the results obtained. The data were retrospective from three-year medical records.

No other study offering evidence either for or against an effect of long-term industrial noise upon chronic cardiac morbidity has been found.

Cerebrovascular

Grusha (62) claimed that blood pressures in the temporal and retinal arteries, respectively, derivatives of the external and internal carotid arteries, were commonly elevated in persons suffering from a "neurosis like state" ostensibly induced by long-term exposure to industrial noise, even though their blood pressure as conventionally measured in the brachial artery of the arm was normal or low. Antonova (63) found a more pronounced tendency for

blood pressure to rise in the temporal artery than in the brachial artery in mill operators subjected to intense noise plus vibration during their shift. Stasiow et al.(36), as noted above, claimed that 80 percent of the operators of mechanical coal washing machines examined evidenced a marked narrowing of the retinal arterioles, particularly at the end of the work day. A mechanism for the suggested tendency for a relatively greater cerebral than peripherial hypertension is not readily apparent. Nevertheless, if such a tendency were confirmed, it would imply an increased risk of cerebrovascular stroke for persons who work under noise greater than would be otherwise expected. The possible effect of long-term noise exposure upon chronic cerebrovascular pathology, for all practical purposes, has not been studied. Epidemiological and physiological studies are needed to evaluate potential influences of long-term occupational sound exposure upon cerebrovascular pathology.

Peripherial vascular

Poor peripherial circulation is often evident in people working under intense sound and is manifest by spastic capillary constriction, pallor of the skin, dryness and pallor of the oral and nasal mucosa and in coolness, mottling and paraesthesias of the extremities (7, 12, 16, 17, 26, 32, 35, 36, 41). Although this is particularly likely where noise is combined with vibration, it also occurs without vibration (12, 16, 26). It is likely that noise, like smoking or cold, exacerbates vibratory vasospastic disease and other Raynaud-like phenomena. Meinhart and Renker, however, found hypertension and hypotension strongly associated with noise exposure in one industrial population without evidence of a difference in peripherial vascular disease.

Although poor circulation may be due in part to the maintenance of a high cutaneous vasomotor tone and a reduced cardiac output , it may also reflect arteriosclerotic and atherosclerotic change. The evidence for ischemic heart disease cited in the section above leads also to the expectation that atherosclerosis will be manifest elsewhere in the body than in the heart.

The possibility that atherosclerosis may be exacerbated by long-term occupational noise exposure has received little systematic atten-

tion. Shatalov (27, Table 3) compared the pulse wave velocity of people who worked under 85-111 dB periodic sound with that of people of the same age who routinely worked under 85-122 dB continuous sound. The pulse wave velocity of people who worked under continuous sound did not differ from that of non-noise-exposed controls. However, pulse wave velocity was 23 percent greater under periodic sound than in the other conditions ($p < .01$). An accelerated pulse wave velocity denotes the loss of normal distensibility of arterial walls, primarily as a result of atherosclerosis and other degenerative change. Arterial distensibility has been calculated from pulse wave velocity (38) and found 30 percent less for people working under periodic than under continuous sound in Shatalov's populations (Table 3). This, in effect, is equivalent to aging the arteries about 15 years.

Elevated cholosterol favors atherosclerosis and increases the probability of thrombotic or embolic occlusion of small vessels. Khomulo et al. (39) found plasma cholosterol significantly higher in 103 people working under 95-117 dB sound than in 51 people working under 60-95 dB industrial sound, averaging about 20 percent greater after 5 years on the job; total fatty acids and beta-lipoprotein did not differ. Rumyantsev and Mekhel'son (42) found beta-lipoprotein, but not cholosterol, elevated in men who worked under the intense noise and low frequency vibration of engine rooms in ships, as compared with crewmen on deck.

Research on peripherial vascular change, on cholosterol and lipoprotein metabolism and on thrombocyte adhesiveness in humans working under intense noise is needed.

General

Several studies that were not referred to above in relation to specific cardiovascular disorders have reported an enhanced morbidity with long-term industrial noise exposure only in broad categorical terms such as "abnormalities of blood pressure regulation" (7), "circulatory disorders" (3, 8), "functional cardiovascular disorders" (31) or "cardiovascular/circulatory disorders" (25). The only study of the potential correlation between long-term industrial noise exposure and extra-auditory health impairment that has been conducted and published in the United States is of the latter kind (25); its data on disorders in a broad cardiovascular/circulatory category - which is defined to include "tachycardia, angina pectoris, hypercholesterolemia, hemotrypsia, coronary sclerosis, hypertension and hypotension" - are summarized in Figure 14.

Comparing noise exposed and non-noise-exposed populations merely on the basis of the aggregate frequency of disorders diagnosed within such broad categories as this tends to obscure and trivialize important chronic disorders with the acute. Nevertheless, the results of this United States study, conducted by the Raytheon Corporation, are of particular interest for their categorical confirmation of the major elements of evidence found in the foreign literature. The 5-year medical records of 520 people who were routinely exposed at work to sound of 95 dBA or greater were retrospectively compared with those of 514 people exposed to 80 dBA or less in the same two factories. About 87 percent of the records were from a factory manufacturing nuclear reactor pressure vessels, the others from a manufacturer of electronic components for missiles. The records had been maintained in a reasonably comprehensive manner by professionals. Excluded from the study were records of people found on pre-employment medical examination to have active health disorders. Double-blind techniques were used. Environmental factors other than noise were said to be well under control and not at variance with the NIOSH Act of 1970; bias due to such factors or to socioeconomic differences was said to be minimized by rigorious sampling and matching. Records were matched by job description, length of employment, age, sex, race, martial status and work shift, but inherent task differences, of course, made perfect job-for-job matching impossible.

Lower morbidity was actually favored for the high noise workers: (i) their median age was about 6.5 years less than that of the low noise workers, and (ii) their median time on the job was 1.1-3.4 years less. Yet, the prevalence of clinically designated "cardiovascular/circulatory" disorders was significantly greater in high noise than in low noise areas of each factory. It was 70 percent greater under high noise than under low noise in the nuclear vessel fabrication facility ($p<.01$) and 100 percent greater in the electronics factory ($p<.01$). Figure 14, in which the data from both factories are pooled and plotted by age group, shows the prevalence of cardiovascular/circulatory disorders to be greater among high noise workers in each age group and to increase with advancing age only under high intensity noise. The only one of the eight general diagnostic categories considered in this study for which significant differences were found in both factories was cardiovascular/circulatory.

For each of the other seven extra-auditory diagnostic categories evaluated (respiratory, digestive, allergenic/dermatological, muscular/skeletal, glandular, neurological and urological) a significantly higher prevalence was recorded among the high noise workers only in the vessel fabrication facility. There, the prevalence of disorders within these various categories was 2.1-3.9 times greater under high than low noise, differences that were all highly significant statistically. The failure to find such differences in the electronics factory could mean that the differences observed in the nuclear vessel fabrication facility were not actually caused by noise; it could reflect the relatively small sized sample in the electronics factory; or it could be due to the fact that the electronics facrory had average and peak high noise levels 13-15 dBA lower than the vessel fabrication facility. In any event, it is notable not only that cardiovascular/circulatory was the only diagnostic category to significantly differentiate high noise from low noise areas in both factories, but that an elevated prevalence of cardiovascular/circulatory disorders was evident under the high noise conditions of the electronics factory at a time when significant increases were not evident in any other kind of extra-auditory disease.

Even when compared with hearing impairment, cardiovascular/circulatory disorders seem strong competitors for the first order concern of these workers. Thus, audiometric measures made on 385 high noise workers and 186 low noise workers in the vessel fabrication facility found average hearing thresholds elevated in the worst ear of the high noise group by only 3 dB at 1 kHz and 7 dB at 2 kHz, which are commonly considered two of the major frequences for speech communication. Hearing loss is not formally considered handicapping until it exceeds 25 dB at these key frequencies, and threshold must be elevated about 30 dB before one is unable to hear a whisper at a distance of one foot. These measures of average hearing levels do not demonstrate that there was no serious hearing loss in the exposed population; they merely demonstrate the inadequacy of mean values for describing hearing loss in noisy environments. One may assume that there was, in fact, a significant percentage handicapping hearing loss although it was not reported; the population distribution of hearing loss is not normative but skewed, and measures of central tendency, as is also the case for blood pressure, are inadequate to describe hearing loss. Nevertheless, while a percentage of the population may have hearing loss in the Raytheon study, the significance for over-all health seems small compared with the cardiovascular/circulatory disorders implied in Figure 14.

The Raytheon study data also indicate that the eight categories of extra-auditory disorders reported had skewed distributions. The investigators used only the median test as a criterion of difference between noise-exposed and non-noise exposed populations, and this clearly contributes to the modesty of their reported results. Based upon median frequencies, they reported significant effects in the nuclear vessel fabrication facility only for the diagnostic categories of respiratory and allergenic/dermatological ailments; analysis based upon the prevalence of diagnosed disorders shows highly significant differences in each diagnostic category for high vs. low noise areas of the vessel fabrication facility. Work under noise is associated primarily with an increased number of people for whom disorders are diagnosed, not necessarily with a significantly increased frequency of disorder diagnoses for the median individual. Indeed, one might expect this to be the case for chronic disease: coronary sclerosis, for any individual, need only be diagnosed once.

The largest difference between high noise and low noise workers was found for administrators in the Raytheon study. Administrators working under high noise in the vessel fabrication facility had about 5.6 times as many diagnosed disorders, 16.5 times as many subjective complaints, 25.3 times as many accidents, 15.9 times as many discrete illness-related absences and 11.4 times as many total days of illness-related absence as their counterparts in quieter parts of the plant. For most of these comparisons in high noise areas, but not in low noise areas, administrators actually tended to be more affected than non-administrators in their own age class. The adverse changes noted more broadly in this study, therefore, cannot be attributed to factors associated solely with the physical tasks performed at work.

Other surveys of general morbidity of people working long periods under industrial noise generally agree with the Raytheon data in suggesting cardiovascular disorder as the most sensitive indicator of extra-auditory effect (3, 7, 8, 12, 13).

Several points suggest that the evidence cited here for impairment of cardiovascular health by long-term industrial noise exposure is actually conservative: (i) Subjects who were identified as being genetically or otherwise predisposed to cardiovascular disorders when beginning work at noisy jobs were a priori explicitly excluded from eight studies (6, 9, 11, 12, 13, 22, 25, 27), and one study excluded persons with left ventricular hypertrophy (15). (ii) In five studies that reported an association of cardiovascular disorders with long-term employment under high intensity noise the population of high-noise workers was actually younger and less experienced than the low-noise workers to which they were compared (4, 5, 6, 7, 25). (iii) In some studies early retirement and health-related transfer to other jobs was specifically noted to minimize the differences reported (c.f. 1, 5), and in the Raytheon study,at least, some workers actually transferred from noisy work for health related reasons and were later counted as controls. (iv) Several studies that correlate cardiovascular disorders with noise exposure were not actually comparing high noise versus low noise or quiet, but were instead comparing high intensity noise and very high intensity noise (5, 12, 24, 30). (v) One large-scale study reported

highly significant increases in hypertension and hypotension among people exposed for prolonged periods to industrial noise although neither healthy people nor the general population were used for baseline "control" reference, but ambulatory clinic patients of the same geographical district (20).

On the other hand, noise-exposed people were on the average 2-3 years younger than reference controls presumed to have less noise exposure in the studies of Jonsson and Hansson (43) and of Takala (44). But those studies had very small sized samples and were also rather weak on other grounds. They are not so crucial to the central conclusions of this review as the many studies mentioned in the paragraph above which had very large sized samples. Ear protectors were said to have been worn by the ultrasound operators studied by Yazabuskis (34) and by the weavers studied by Cieslewicz (5). The latter may indicate that the observed effects were not due to sound. But sound is not totally excluded by ear protectors; in actual practice, protectors are often improperly worn or not worn at all (74, 80). Moreover, at least four studies document acute vasomotor and heart rate effects of sound in people equipped with ear protectors properly worn (75-78), although Dega and Klajman (79) found that ear protectors appreciably diminished the increase in heart rate that otherwise occurred over the course of the working day in shipyard grinders working under intermittent impulse sound.

Gastrointestinal

The possible association of long-term work under industrial with clinical signs of gastrointestinal pathology has not been extensively studied. Seven of the studies for which cardiovascular morbidity has already been discussed also reported morbidity data for gastrointestinal problems. One of these studies, that of Jansen, found no difference: clinical examination suggested virtually the same frequency of gastrointestinal problems for men working in high and low noise areas of German steel mills, and his sample was quite large (12; Table 2). The other six of these studies suggested that more gastrointestinal disorders occurred under the higher intensity of the compared noise conditions. Their

[1] There is no page 26.

findings are briefly summarized as follows:

Kangelari et al.,(17, Table 2) reported the three-year incidence of gastritis and all kinds of gastrointestinal ulcers combined about 2.3 times greater for 135 mechanics exposed to 116-120 dB sound at work than for 152 locksmiths who worked under sound of 88-90 dB. The frequency was low in each case, and my estimate is that the difference is not statistically significant. Morbidity, however, was said to be 2-3 times greater yet for foundary workers exposed to 110 dB sound plus vibration.

Pilawaska (23, Table 2) reported about 5 times the frequency of peptic ulcer, gastric or intestinal, for 1826 Polish shipyard workers exposed to the noisiest portion of the yard than for 5225 co-workers exposed to 20 dB less (3.7 percent vs.0.8 percent, $X^2 = 81$, $p < .0001$).

Jirkova and Kremarova (13, Figure 9) found peptic ulcers generally more common among 969 Polish industrial machine operators exposed to 85-115 dB sound than among 687 other in non-noisy work-sites. The difference was greater for men than women, reaching a factor of 8 for men below 40 years of age who had over ten years of job service.

Dokukina claimed chronic gastritis and gastrointestinal ulcers to be 2-3 times as common for 144 women working under 112-122 dB high frequency sound in electrical stations of metallurgical factories, and also for 390 who experienced similar intensities of low frequency sound plus periodic low amplitude vibration on platforms in cement factories, than among 446 "controls" who worked in grocery produce stores and warehouses. For 89 other women said to have "noise disease" these differences were further increased. Most were in their mid-thirties and had 7-14 years on the job. The controls were 1-4 years younger than the noise-exposed (7, Table 2).

Cieslewicz (5, Figure 3) reported gastrointestinal ulcers significantly more prevalent at all ages for male weavers exposed to 90-116 dB medium frequency sound in Polish weaving mills than for male spinners routinely exposed to 84-90 dB, the overall difference being 17 percent and 3.4 percent, respectively ($p < .001$). The difference between the two groups of men increased steadily with

advancing age, increasing from 5.8 percent for the weavers at ages 18-29 to 30 percent for ages 50 and above; at the latter age the frequency for spinners was zero. By way of comparison, Cieslewicz noted that the ulcer frequency for men in the general population at large was 5 percent. There was no difference in the ulcer frequency for females spinning vs. weaving, and their gastrointestinal ulcer rate was about one-fourth that of men.

The Raytheon study (25, Figure 14) found no difference in the general diagnostic category "digestive" for morbidity in the more noisy and less noisy areas of the electronics factory studied. The sample size in each area, however, was small, e.g. 66 and 65 men. A highly significant excess morbidity in the noisy area of the nuclear vessel fabrication plant was found (11 percent vs. 4.7 percent, $p < .001$). The category of disorders thus labelled included "ulcer (duodenal & peptic, etc.), gastritis, diverticulitis, duodenitis, appendicitis, acute gastroentitis."

Tarantola et al. (81) followed the evolution of gastrointestinal pathology in 73 men engaged in the trimming of malleable cast-iron with fixed grinders for a period of ten years. Radiographically verified pathology emerged in 65 percent of the men, becoming evident within the first 2-3 years on the job. The authors attributed the pathology to "the associated action of vibrations, noise and psycho-psychic stress connected with the type of work."

Shitskova (82) has reviewed inaccessible Russian papers purported to demonstrate the induction by sound of excessive parasympathetic tone in the gastrointestinal system of ultrasound device operators.

The data presently available are insufficient to justify any judgement about the possible relevance of long-term high-intensity noise exposure to gastrointestinal pathology. My guess is that future research will confirm a differential susceptibility for the sexes and identify certain types of high intensity acoustical exposures as being conducive to gastrointestinal pathology, and others not.

Infectious Disease

Remarkably few reports address the subject of the possibility that occupational noise may influence the body's resistance to infectious disease. Four have been identified, all superficial.

Kangelari et al. (17) claimed that the three-year incidence of influenza, catharra and other respiratory infections considered as a group was 2.3 times greater for 135 mechanics working under 116-120 dB sound than for 152 locksmiths working under 88-90 dB sound . The difference is significant ($X^2 = 8.1$, $p < .01$).

Barhard et al. (3) reported 90 percent more respiratory disorders for 132 boilersmiths working under 98-127 dB high frequency sound plus vibration than for lift drivers who experienced less noise and vibration.

Dokukina (7) claimed about twice the incidence of severe infections "viral respiratory, cholangio hepititis," etc., and suggested a generally lowered resistance to infection, for 144 women working under 112-122 dB high frequency sound in electrical stations of metallurgical factories, and for 390 women who experienced similar intensities of sound plus periodic low amplitude vibration on platforms in cement factories, than among 446 women whose work in grocery produce stores and warehouses did not subject them to noise. Morbidity for 89 women from the noisy enviroments said to have "noise disease" was higher.

The Raytheon study (25) used the general diagnostic category of "respiratory" to include "pneumonia, pleurisy, bronchitis, broncho-pneumonia, pharyngitis, laryngitis, cold and sore throat." In the electronics factory there was no difference between high noise and low noise areas (85 percent and 91 percent, respectively). In the nuclear vessel fabrication facility morbidity was higher in the more noisy area (73 percent vs. 32 percent, $p < .001$).

While there has been much speculation about the possible association of high intensity occupational noise exposure with altered resistance to infective disease, substantive research to provide relevant data apparently has not been done.

Reproductive

There is substantial reason from preclinical studies to expect pervasive reproductive system effects of long-term noise exposure. However, the subject is virtually unstudied with respect to occupational noise.

Carosi and Calabro (83) compared 330 families in Italy in which either husband or wife worked in noisy industrial jobs (metalworking or weaving) with 200 non-noise exposed families matched for age, and

they reported a significantly smaller number of sons born to the noise-exposed.

Dokukina (7) found 2-3 times the frequency of disruption of ovulatory or menstural cycles in women working under 112-122 dB noise, or such noise plus vibration, than in non-noise-exposed women (see Table 2 for details on exposure). In jobs clearly involving not only noise but strong segmental or whole body vibration reports, for which only abstracts are available, are said to have found work at such jobs associated with prostitis (84); impaired spermatogenesis and unspecified female reproductive change (85); and a high incidence of menstural problems, fallopian tube inflammation, late toxemia and at childbirth a high frequency of atonic delivery (86). Men working as drillers and sinkers in mines and exposed to considerable noise and vibration had erectile difficulties with prevalence 14 times expectation for age group 31-40 years and 5.5 times expectation for age group 41-50 years, the problem increasing with length of service irrespective of age.

Neurological

Numerous authors report functional neurological change associated with long-term occupational noise exposure. Categorically, the main functional signs include: (i)aberrant vasomotor regulation and other signs of autonomic imbalance such as dermatographism, hyper-(and occasionally hypo-)reflexia, hyperhydrosis, tremor of extended hands and, occasionally, of the eyelids; (ii) altered sense of balance; (iii) decreased tactile sensitivity of hands and feet, as observed in standardized pain and vibratory stimulation tests; (iv). decreased reaction time; and (v) electroencephalographic hyposynchrony with underreactivity to photic stimuli and a tendency for normal regional boundaries of electrical activity within the brain to be obscured.

Vasomotor instability, as discussed in an earlier section, is perhaps the most ubiquitious functional neurological disorder found in people exposed for long periods to occupational noise. Cuesdean (6, Figure 13) reported vasomotor dysregulatory phenomena in almost a third of his noise exposed subjects. Impaired neuroregulatory control of the circulation appears to be the central neurophysiological effect of long-term work under noise. Extreme vasomotor instability has obvious adverse implications for those whose vascular system is otherwise impaired. The central question is if, and if so, how

it acts to eventually result in fixed hypertension and/or organic heart disease. Shatalov et al.'s (30) electrocardiographic changes were "functional" in that they occurred in people having no known organic heart disease. The dramatic differences in the prevalence of hypertension that are evident in Table 3 were observed in populations from which all individuals having organic heart disease had been a priori excluded. Hypertension is "functional" until it results in demonstrable organic change. The study of populations that are exposed to occupational noise may provide much needed insight into the yet poorly understood role of neurogenic stimuli in the production of chronic cardiovascular disease.

Kachny (14, Table 2) reported a remarkable study of 591 young (ages 15-27 years) women who tended automatic weaving machines under continuous high frequency sound of 102-108 dB in two Russian textile factories. Brachial blood pressure asymmetry (defined as a difference between the two arms of $\geq$ 10 mm Hg) increased with passing time on the job, and after ten years or more systolic asymmetry was evident in 8-11 percent of subjects and diastolic asymmetry in 2-5 percent. This phenomenon was more common in women who tended a larger number of machines, fully 3.6 percent developing systolic asymmetry of 20 mm Hg or more after five or more years of service, and 5.2 percent after 6-10 years of service. (As mentioned earlier, systolic hypotension developed in over 50 percent of these young women during their first six months on the job, diminished in frequency to just under 10 percent by five years and began to give way to a modest hypertensive trend thereafter.) Blood pressure asymmetry is commonly though of as being indicative of structural change, particularly supravalvular aortic stenosis (primarily in children) or major occlusive disease of the proximal subclavian artery such as is associated with the subclavian steal syndrome (95). However, the context of this study suggests that neurological disorder should also be considered as a potential cause.

Zvereva has recently reported two studies that highlight neurological change. In the first, which involved 334 men aged 30-40 years who worked under 95-100 dB sound plus vibration at various automated jobs in the quarrying of dolomite, he also reported blood pressure asymmetry - 25 percent of subjects (35). He also reported abnormal temperature responses in this study, and, in a companion study (41)

of 340 men working in a pipe and sheet metal rolling mill under percussive sound reaching 122 dB (occasionally 142 dB), he reported thermoasymmetry. Anisocora (inequality of pupillary diameter) was noted in 8 percent of the dolomite miners. Tremor of extended hands was evident in 30 percent of subjects in the first study and was common, along with tremor of the eyelids, in the second. Sensitivity to pain and vibratory stimuli experimentally administered was impaired in both studies. Of particular interest is the fact that whereas vibration as well as sound was important in the first study it appears not to have been a significant factor in the second; but similar neurological signs developed in each. Likewise, in 144 compressor operators who routinely experienced 87-98 dB sound, increasing to 90-110 dB in the 16 Hz infrasonic range, but who would have experienced little other than airborne vibration, Sanova (26, Table 2) observed eyelid and hand tremor in 73 percent, along with such signs as persistent red dermatographism, low skin temperature and hyperhydrosis.

Romberg instability was common in Zvereva's rolling mill, and it was reported in several studies reviewed by Gulian (88). In Jansen's study of steel mills (12, Table 2), an impaired sense of balance approached but did not quite achieve statistical significance as a differentiating factor for subjects in high noise and low noise areas. An impaired sense of balance was listed by Enderlein (8, Table 2), along with circulatory disorders and hearing impairment, as contributing to the quantitatively greater association of health impairment in the East German workforce with noise exposure than with other environmental factors, including chemical agents, mineral dusts and vibration.

The technically most sophisticated and most adequately designed study of functional neurological change encountered is that of Kanevskaya et al. (19, Table 2). It is of particular interest in that the subjects were said to have experienced essentially no vibration. There were three compared conditions: (i) 256 people working under stable continuous broadband predominantly high frequency 90-100 dB sound of Leq about 95 dB as fitters, lathe turners and sanders; (ii) 284 people who, in addition to essentially the same continuous noise, also experienced pulses of 107-117 dB, occasionally to 122-125 dB, also of high frequency and with overall Leq of about

95 dB; and (iii) 100 controls performing comparable work as fitters, repairmen and instrument adjusters, but who were not exposed to uncommon sound. The groups were comparable in that they were comprised about one-third of females and in that they were mostly (about 60 percent) 25-40 years of age and had been on the job over ten years. Here, as in Zvereva's rolling mill workers, sense of balance was impaired: labyrinth excitability was abnormally decreased as evidenced by weak, short-lasting and slowly-developing experimentally-induced nystagmus in 26 percent and 21 percent, respectively, of those exposed to stable and complex noise, as compared with only 8.3 percent of controls ($p < .01$).

In addition, those exposed to the two most noisy conditions manifest, in common, 2-8 percent hyperreflexia, 11 percent impaired distal sensitivity for pain, and a 14-16 percent frequency of elevated thresholds to cutaneous vibration, all significantly different from controls. Extreme dermatographic responses were evident in 9.5 percent of those who worked under the most complex noise. Kanevskaya et al.'s noise-exposed subjects manifest several electroencephalographic anomalies that were not evident in controls, all more common under the noise having superimposed pulses than under steady noise of about the same Leq: irregular slow abnormally low amplitude activity, 53 percent vs. 26 percent ($X^2 = 39$, $p < .001$); absence of normal differences in regional activity of the brain due to either disorganized polyrhythmic activity or generalized alpha, 52 percent vs. 37 percent ($X^2 = 13$, $p < .001$); below normal reactivity for photostimulatory desynchronization, 42 percent vs. 21 percent ($X^2 = 43$, $p < .001$); total absence of electroencephalographic reaction to photic stimulation, 16 percent vs, 5 percent ($X^2 = 15$, $p < .001$). The average alpha amplitude was 30.4 mV under the complex sound and 36.9 mV under steady sound, as compared with 50.2 mV in controls. Very low amplitude tracings (< 30mV) were present in 68 percent of subjects under complex sound and in 54 percent of subjects under steady sound, compared with only 11 percent of controls. Such hyperactivated electroencephalograms with decreased reactivity to photic stimuli and a tehdency for normal zonal differences to be obscured were reported by Gulian for other more limited studies (88).

The Raytheon study (25, Figure 14) reported about 3.1 times the frequency of disorders classified in a general "neurological" category in the high as opposed to the low noise areas in the nuclear vessel fabrication facility (7.5 percent vs. 2.4 percent ($X^2 = 12.2$, $p < .001$). Unfortunately, this broad category included neuropsychiatric disorders (such as "manic depressive, acute anxiety and neurosis") in addition to disorders more conventionally considered as neurological, and accessible data do not permit individual disorders or groups of disorders to be parceled out. Tavtin (31) recently reported an prevalence of 1.7 percent for "functional nervous disorders" described as an "asthenoneurotic syndrome" in 120 people working under $\leq$ 70 dB in a machine construction plant. The prevalence was significantly higher for 267 people working under continuous sound $\geq$ 110 dB (12.5 percent) and for 166 people working under impulse sound of about 114 dB (16.2 percent). Symptoms typical of anxiety neurosis or anxiety state are commonly reported by people who are exposed routinely over long periods of time to occupational noise of high intensity. Phenomena that are clearly psychiatric or neuropsychological, however, are out of the scope of this review.

Limitations of the Evidence

Prolonged exposure to any real life acoustical environment involves variables that cannot be fully controlled. The factor of contrast in most of the environments compared in this review is the intensity of audible sound, in some intermittency. But aspects of sound other than intensity and intermittency are obviously important in determining whether or not chronic exposure will have adverse extra-auditory effects. In no instance have the long-term effects of sound been fully analyzed with respect to its physical and temporal characteristics, its meaning for the hearer and the hearers' stimulus history. Infrasound and ultrasound often accompany audible sound, and while evidence suggests that their effects are modest and mainly due to the audible components of their spectra, neither is often measured and both may prove to have effects yet poorly defined.

All sound is detected as vibration. The cochlea of the ear is stimulated by vibration transmitted through the middle ear from the eardrum and by vibration transmitted through the body. Mechanoreceptors in the skin, joints, viscera and muscles and also the

vestibular apparatus detect high intensity sound, especially at low intensities, as vibration; flutter, a special type of vibration, is detected by specialized mechanoreceptors in the skin. Sound vibrates the body not only directly but also indirectly by vibrating structures with which it is in contact; sound and vibration often have common origin in an object or surface that touches the body. It is often moot - and, indeed, somewhat academic - to ask to what extent the effects of an acoustical environment are due to auditory perception per se as opposed to more general vibration. This confounding factor, of course, is of concern not only in studies of the extra-auditory effects of sound: vibration also contributes to hearing impairment, and it interacts with sound in its impairment of hearing (35, 96-99). Nevertheless, it was apparent in quite a few of the studies reviewed here that vibration other than airborne vibration could not have been an important environmental influence.

Extremes of temperature and humidity, chemical dusts and fumes and non-noise-related emotional distress undoubtedly confound in some situations, but they have been rather clearly precluded as the cause of observed extra-auditory changes in some studies. Moreover, none of these extranaeous influences is common to all of the many diverse situations in which high intensity audible sound is associated with increased morbidity. In studies of hearing impairment associated with industrial noise exposure the possible ototoxic effects of airborne industrial chemicals, to my knowledge, has never been clearly separated from the effects of sound.

The self-selection of relatively unhealthy people into noisy environments and/or of relatively healthy people out of noisy environments may in some cases diminish the importance of correlations between noise and impaired health. But this is no more likely for extra-auditory health disorders - and, indeed, may be less likely - than for hearing impairment.

In perspective, the evidence that long-term noise exposure may increase extra-auditory morbidity in humans is of the same kind, of essentially the same quality and subject to the same criticisms as the evidence which supports the generally accepted belief that long-term exposure to noise that does not physically disrupt the tympanic membrane impairs hearing, namely, epidemiological correlations and controlled experiments with animals. In the most

strict sense, such evidence can never provide absolute proof of cause and effect in human disease. Even in the most sophisticated epidemiological surveys, correlation remains forever correlation; despite fundamental similarities between humans and other higher animals, species difference remain. But for ethical reasons, these are the only kinds of evidence that we are ever likely to have. There are penalties both for falsely indicting an environmental agent and for failing to arraign a guilty one. Yet, judgements must be made. In a practical sense, the available evidence now demands that prolonged exposure to high intensity sound be viewed in a much broader sense than heretofore as a serious threat to general human health. The evidence for associating long-term sound exposure with cardiovascular disease, in particular, is comparable to that for associating it with loss of hearing. Indeed, some studies suggest a greater prevalence of cardiovascular disorders than of hearing impairment among people who have been employed under noise for long periods of time (c.f. 13, 16, 25).

Implications for Standards

Data presently available are inadequate to suggest whether chronic occupational sound exposures shorter than 3-5 years or less intense than about 85 dBA - 90 dBA is associated with an increased extra-auditory morbidity.

However, the fact that there does appear to be a substantial increase in cardiovascular morbidity within the range of long-term noise exposures commonly considered permissible for the conservation of hearing emphasizes the importance of enforcing existing standards and emphasizes the importance of conducting aggressive research to determine whether these standards provide as much protection as we should afford. Cardiovascular disorders not only diminish enjoyment and productivity but threaten life itself.

The indications that varying and discontinuous sound more adversely affect cardiovascular function than relatively unvarying continuous sound makes it questionable that the equal energy assumptions that guide judgements of acceptable exposures for the protection of hearing are adequate for the protection of cardiovascular health.

Table 1

Industrial Distribution of Reviewed Studies Suggesting Cardiovascular And Related Effects of Long Term Work Under Intense Sound*

Industry	General Cardiovascular Disorder Category	Hypertension	Hypotension	Cardiac Disorders	Vascular Lability	Peripherial Vascular
Aircraft engine testing		28				
Aircraft line crew	32				32	
Chemical industry				4		
Compressor shops	26	26		26		26
Cement factory	7			7		7
Electrical station in Metallurgical Industry	7			7		7
Electronic component mfg.	25					
Grain silo		18				
Industrial Machine Operate		1, 11, 16, 28		11	1	16
Metal fabrication & heavy machine building	3, 25	30, 24, 31	24		3, 24 30	
Mining		35		12	35	35
Oil industry		10				
Rubber plant		6		6	6	
Textiles		2, 5, 14 15, 22, 30	14, 33	15	14, 30 33	
Ultrasound operators				34	34	
General industrial	8, 17, 27 36, 37	44, 62 13, 19, 20 27, 29, 43,	19, 20, 21, 27, 30	17, 20, 30 36, 37	19, 27 36, 62	17, 19
Steel mill		41		12	41	12

*Numbers refer to citations in the list of references. Underlining indicates that data are available in a form that permits approximate statistical confirmation of apparent diffeeences.

Table 2

Miscellaneous Industrial Studies Correlating Cardiovascular Morbidity With Long-Term Exposure To High Intensity Noise

Barhard, et al., 1969 (3). Circulatory disorders claimed increased by a significant 30 percent among 132 boilermakers working under 98-127 dB high frequency noise plus vibration, as compared with lift drivers who experienced less noise and vibration.

Dokukina et al., 1972 (7). Abnormalities of cardiac function said to be 2-4 times more frequent, abnormalities of blood pressure regulation 5-8 times more frequent and extreme tonic peripherial vasoconstriction evidenced by undue dryness and pallor of the mucous membrans of the throat and mouth 1.5-2.5 times more frequent among 144 women who worked under 112-122 dB high frequency sound in electrical stations of metallurgical factories, and also in 390 who experienced similar intensities of low frequency noise plus periodic low amplitude vibration on platforms in cement factories - than among 446 "controls" who did work in grocery produce stores and warehouses. For 89 other women considerd to have "noise disease" these differences were further increased by a factor of 2-4. Most in their mid-thirties and had 7-14 years job experience. Controls averaged 1-4 years older than noise-exposed workers, thus biasing against the results obtained. Data retrospective from 3-year medical records.

Enderlein et al., 1975 (8). Two computer programs developed for periodic overall evaluation of the entire East German work force found the frequency of health impairment higher (46.1%) among people working under high intensity industrial noise than with substances that sensitize the skin and respiratory tract (45.7%), with tools that cause segmental vibration (40.6%) or who were exposed to mineral dusts (37.3%). The major problems among people working under noise were circulatory, hearing and sense of balance.

Graff et al., 1968 (11). Hypertension, and also heart and other blood pressure problems, among 117 Germans who worked under 95-110 dB middle and high-frequency impulse sound of heavy machinery than among 50 others matched for age, educational background and experience who were exposed to relatively little noise. Persons with a family history of hypertension were excluded a priori.

	Number Subjects	Hypertension (%)	Other heart and blood pressure problems (%)
High noise	117	35.9	33.3
Low noise	50	12.0	12.0
X^2 =		8.64	8.10
$p <$		.01	.01

Table 2 (Continued)

Jansen, 1959 (12). Cardiac dysrhythmias (tachycardia, extrasystoles and other rhythmic anomalies) and peripherial circulatory problems more prevalent in steel mill workers and miners who work under 90-115 dB sound than in those who work under 65-90 dB. Ages ranged from 20-60 years, mean 42 years. All had been on the job at least 3 years, most over 10 years. Peripherial signs were mainly paraesthesias and extreme vasomotor tone as indicated by persisten/pallor of the skin and of the mucous membrans of the mouth and throat. Raynaud-like symptoms were claimed to be significantly more common among those who worked under the highest intensity noise even though they did not use vibrating tools. From rather detailed medical examination, the only statistically significant differences found were cardiovascular.

	Steel mills			Mines		
	Number	Cardiac Dysrhythmia(%)	Peripherial Vascular(%)	Number	Cardiac Dysrhythmia(%)	Peripherial Vascular
90-115 dB	669	24.1	61.2	125	27.0	62.0
65-90 dB	336	15.8	49.1	76	17.0	49.0
X^2 =		11.8	13.6		2.7	3.6
p <		.001	.001		-	-

People with chronic ailments a prior excluded from study.

Jonsson and Hansson, 1977 (43). Hypertension more prevalent, diastolic and systolic blood pressures higher in Swedish industrial workers who suffered hearing impairment and had experienced considerable noise exposure during past employment in shipyards, mechanical workshops, etc. than in cohorts in the same industrial population and within the same age range, but averaging three years younger. Implies that current employment not considered to involve consequential noise. Noise history not adequately known for either experimentals or "controls". Experimentals average three years older than controls, thus biasing towards the obtained results. Not a strong study.

	No.	Blood pressure (mm Hg, mean ± s.e.m)		Hypertension (%)
		Systolic	Diastolic	
Impaired hearing	44	145±1.3	89±1.7	22.7
Normal hearing	74	133±2.6	81±0.8	8.1
p <		.001	.001	.05

Kachny, 1977 (14). Hypotensive change strongly dominated the initial period of employment for 591 young (age 15-27 years) Russian women beginning as weavers in textile factories without prior experience of industrial noise. Over half showed a marked tendency to systolic hypotension during the first 3-6 months on the job under 102-108 dB sound, a response that was most apparent near the end of the working day. With increasing time of employment there was an increasing tendency to hypertension. Hypotension defined as systolic and/or diastolic of $\leq$ 100/55 mm Hg, hypertension as $\geq$140/90 mm Hg.

Table 2 (Continued)

Kanevskaya et al, 1977 (16). Hypertension more prevalent among 284 turrent lathe operators, leveling machine operators, guillotine operators, pressers and other automatic machine operators subjected to high frequency 90-100 dB steady sound with superimposed pulsed sound of 107-125 dB, than among 256 fitters, lathe turners and sanders subjected to only the 90-100 dB high frequency stable sound. Exposure for both groups had equivalent levels of 95 dB. Most 25-40 years of age, employed over 10 years.

	Subjects	Hypertension (%)
Steady	256	22.2
Impulse	284	34.7

$X^2 = 10.4$, $p < .01$

Kangelari et al., 1966 (17). The three-year incidence of angina pectoris for 135 mechanics whose work involved exposure to 116-120 dB high frequency sound was a non-significant ten percent higher than that for 153 locksmiths of similar age who were exposed to 88-90 dB low frequency sound at work. It was claimed that these morbidity differences increased 2-3-fold for foundary workers exposed to vibration plus noise of 110 dB.

Kalicinski et al., 1975 (15). Hypertension prevalence and electrocardiographic signs of cardiac ischemia increased with years on the job for female weavers and spinners who experienced 95-105 dB, 32-16,000 Hz sound throughout each work day in Polish textile factories. Hypertension defined as systolic and/or diastolic $\geq$ 140/90 mm Hg at rest. Ischemia defined as S-T segment depression $\geq$ .1mV in a standard 12-lead electrocardiogram taken at rest, the depression being rectilinear or downward sloping. Ischemic change was 6.2 times as common in hypertensives as in normotensives. Of particular interest, all subjects were within the narrow age range of 47-51 years.

Employment (years)	Number Subjects	Hypertension (%)	ECG signs of cardiac ischemia (%)
1-6	31	26	3
7-12	48	38	19*
13	61	47*	16**

*$p < .04$ from 1-6 years on job (Fisher exact probability).

**$p < .06$ from 1-6 years on the job (Fisher exact probability).
Subjects with cardiac hypertrophy were a prior excluded.

Subjects complained of pain typical of coronary heart disease.

Table 2 (Continued)

Maksimova et al., 1974 (19). Hypertensive effect of 95 dBA impulse sound (equivalent 95 dB) for 145 machine shop workers said to be greater than for 141 in the same shop exposed to 95 dBA steady state high frequency sound. Ages 20-45, job experience 5-20 years. When initially starting work under steady state sound the dominant effect is hypotensive, under impulsive noise hypertensive. With passing time and job experience, a hypertensive effect claimed under both steady state and impulse sound - although the hypertensive effect of impulse sound remains greater. No data.

Pilawaska et al, 1977 (23). Hypertension more prevalent among persons working in the most noisy part of a Polish shipyard than in areas having a sound level about 20 dB lower. Subjects matched for age and period of service. Data from medical records.

	Subjects	Hypertension (%)
Noise	1826	1.3
No (less) noise	5825	0.5

Chi square = 9.83, $p < .01$

Sanova, 1975 (26). Systolic blood pressure increased more with advancing age among 144 compressor operators routinely exposed to 87-98 dB low frequency sound (with 90-110 dB in the 16 Hz range) than among 30 people in the same plant not exposed to noise. Under noise, the frequency of hypertension increased with years on the job and the stroke volume of the heart decreased. Statistical evaluation not possible.

Age Years	Systolic b.p.(mm Hg) Noise	Quiet	Years on Job	Hypertension (%)	Stroke Volume (ml) /
20-29	122.6	123.7	Control	?	63.0±1.1
30-39	133.1	125.0	1	10	50.2±2.9
40-49	133.7	127.2	1-5	23.9	49.6±1.3
			6-10	23.4	46.2±1.1
			11-20	30.5	44.5±1.6
			20	?	41.2±3.0

/ Minute volume also decreased.

Shatalov et al., 1969 (29). Elevation of blood pressure, systolic and diastolic, both under and over age 40 years, for male and female working in mechanical engineering and synthetic fiber plants where routinely exposed to 90-120 dB broadband high frequency sound. Values following are mean ± s.e.m. adapted from authors' data.

	Age 40 or under			Over age 40		
	Number	Systolic	Diastolic	Number	Systolic	Diastolic
Noise	521	121.5±.06	76.7±.06	285	130.2±.13	78.5±.09
Quiet	161	113.8±.16	72.0±.12	49	117.6±.75	75.9±.27
Student's t		45.1 ($p < .001$)	35.0 ($p < .001$)		16.6($p < .001$)	9.1($p < .001$)

(Adapted from the author's data).

Table 2 (Continued)

Shatalov et al., 1962 (30). Increased susceptibility to myocardial ischemic towards the end of working day under noise, particularly under intense noise or during exercise.

	Electrocardiographic mild ischemic response			
	Spinning mill		Ball bearing plant	
	No. Measure	t-wave depress(%)	No. measure	t-wave depressed (%)
Before work	144	11.1	86	12.7
After exercise	144	33.3*	85	27.0*
After work	33	21.2/	9	55.0**

* $p < .02$ from before work

** $p < .01$ from before work

/ not differ from before work. $p < .0517$ from after work ballbearing plant by Fisher exact probability test

These are both high noise situations: spinning mill 85-95 dB
ballbearing plant 114-120 dB

In ball-bearing plant as contrasted spinning mill, greater tendency:

- to bradycardia, particularly near end of day (27% vs 17.9%, $X^2 = 12.2$, $p < .001$
- to lower systolic pressure, especially near days end (18% vs 7%, $X^2 = 8.4$, $p < .01$

In spinning mill greater retarded intraventricular conductivity at upper limit of the norm (.1-.11 sec)....$X^2 = 22.6$, $p < .001$.

Mean arterial pressure elevated within the 101-120 mm Hg range in 20.3 % of people from the two plants. Normal is about 85 mm Hg, range 75-105 mm Hg.

Takala et al., 1977 (44). Hypertension more prevalent among men "whose work exposed them to noise and who had impaired hearing" than among men averaging two years younger in the same municipal population in southwest Finland.

	No.	Blood pressure (mm Hg, mean ± s.e.m.)		Hypertension (%)
		Systolic	Diastolic	
Impaired hearing	32	155±3.9	99±1.9	34.4
Normal hearing	67	151±2.7	95±1.8	20.8
$p <$		-	-	.045 by one-tailed hypothesis, otherwise not significant.

Table 2 (Continued)

Taytin, 1976 (31). Functional cardiovascular disorders more prevalent among 267 people working under 110 dB continuious sound or 166 working under 114 dB impulse sound in machine construction factories than among 121 "controls" exposed to only 70 dB or less in their work. There was no significant difference in this study between those exposed to steady and impulse sound.

	Subjects	Functional Cardiovascular disorders (%) ++
Continuious 110 dB	267	6.3*
Impulse 114 dB	166	8.4**
Control 70 dB	121	1.7

++ Also called "neurocirculatory dystonia"

* Chi square from control = 8.3, $p < .01$
** Chi square from control = 16.5, $p < .001$

Yazbursakis, 1971 (34). Cardiac ischemia, as indicated by electrocardiogram, more easily induced by an exercise test at the end of the work day for people who routinely work with 100-160 dB ultrasound devices. This is accompanied by a bradycardia, a lowering of systolic pressure, a reduced ability to regulate blood pressure appropriately under physical load and complaints of fatigue and malaise. For 36 ostensibly healthy young (aged 21-42) men and women who had worked with ultrasound for 2-5 years the before-work and after-work S-T segment depression with rectilinear or downward slope during exercise stress testing was as follows.

	S-T depression ≥ 1 mm	
	no	yes
Before work	34	2
After work	24	12

$X^2 = 7.18$ (Yates), $p < .01$

	S-T depression		
	none	1 mm	1-2 mm
Before work	34	2	0
After work	24	5	7

X^2 (2 degrees freedom) = 79
$p < .0001$

Zvereva et al, 1975 (35). Hypertensive tendency claimed in 20 percent of workers subjected to 95-100 dB low and mid-frequency sound, plus vibration, in dolomite flux production facilities, despite their young age (30-40 years). Job experience ≥ 3 years.

Table 3. Hypertension more prevalent, hypotension less prevalent and velocity of pulse wave propagation in arteries greater for people routinely working under intense periodic sound than for people working under intense continuious sound in Moscow factories. 24 percent had been on the job about 5 years, 24 percent 5-10 years, all others over 10 years. Individuals with organic heart disease a priori excluded from study. Hypertension defined as systolic and/or diastolic $>$130/90 mm Hg under 40 years of age, $>$ 140/90 above 40 years of age; hypotension $<$100/60 mm Hg; pulse pressure norm 40-60 mm Hg. Measures made after 10 min rest at beginning of work day. Sound broad-band predominantly high frequency. Arterial distensibility (38) and statistical probabilities derived by the reviewer. The course of a work day under periodic sound was characterized by rising blood pressure, tachycardia and capillary spasm, a day under continuious sound by extreme arterial and venous pressure lability, lowering of venous pressure and peripherial resistance, bradychardia and reduced stroke volume.Despite the marked differences in hypotensive and hypertensive extremes, the average blood pressures of people under continuous and under periodic sound differed by only 6-8 mm Hg. In non-noisy Moscow factory jobs the general prevalence of hypotension and hypertension, all types combined, was said to be, respectively, 1.3 percent and 5.2 percent. Adapted from Shatalov, 1965 (27).

Sound Exposure	Number Subjects	Hypotension (Percent)			Hypertension (Percent)		
		Systolic	Diastolic	Pulse Pressure	Systolic	Diastolic	Pulse Pressure
Periodic 85-111 dB	338	o.2	0.2	4.7	25.4	11.2	8.5
Continuiout 85-122 dB	1019	6.1	3.1	30.5	9.0	4.9	4.8
X^2 (one degree freedom)		17.9	7.5	89.2	60.0	16.8	6.7
$p <$ (two tailed)		.001	.01	.001	.001	.001	.01

Sound exposure	Number Age-Matched Subjects	Pulse wave velocity m/sec (mean ± s.e.m.)	Arterial Distensibility≠
Periodic 85-111 dB	25	7.0 ± 0.34*	.259
Continuious 85-122 dB	15	5.7 ± 0.31*	.371
Quiet controls	14	5.8 ± 0.15	.378

*$p <$.01 (t = 2.83)

≠ The percent of change in volume per 1 mm Hg rise in pressure.

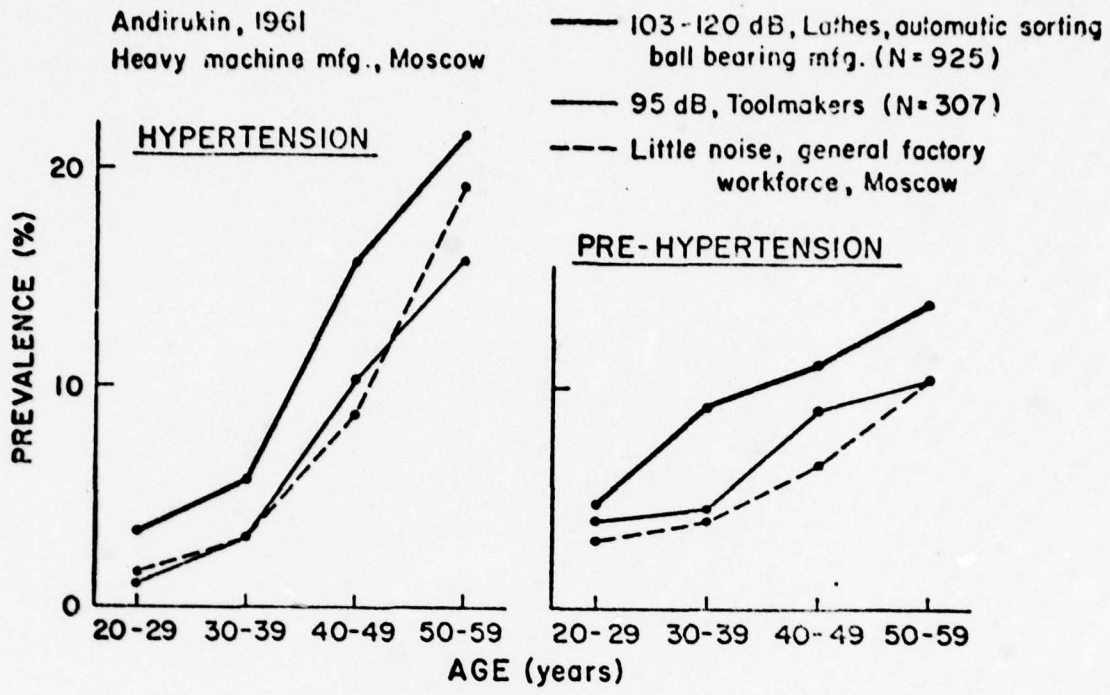


Figure 1. Hypertension and prehypertension (labile and borderline hypertension) prevalence greater among 925 Moscow workers routinely exposed to 103-120 dB broadband predominantly high frequency sound in automatic sorting, lathe and ball bearing shops (solid heavy line) than among 307 toolmakers routinely exposed to about 93 dB (light solid line) or among the general work force in Moscow factories exposed to a "relatively small amount of noise"(dashed line). Comparing the prevalence of hypertension and prehypertension combined for the 103-120 dB machine shop workers vs. the 95 dB toolmakers, $X^2 = 7.02$, $p < .01$; comparing the 103-120 dB machine shop workers vs. an equal number of general factory workers in less noisy jobs, $X^2 = 78.8$, $p < .0001$. Hypertension defined as systolic and/or diastolic $>$130/90 mm Hg under age 40 years and $>$ 140/90 mm Hg over age 40 years; blood pressure measured during work after 10 min rest, repeated until consistent. Over 50 percent of the workers had been on the job over 10 years. Sample distribution among age groups not given. For statistical tests I assumed samples equally distributed among age groups. This biased against significant results since the largest proportion of subjects may be expected in younger age groups, and the difference within the oldest age group was minimized by transfer from noisy work and to early retirement. Adapted from Andirukin 1961 (1).

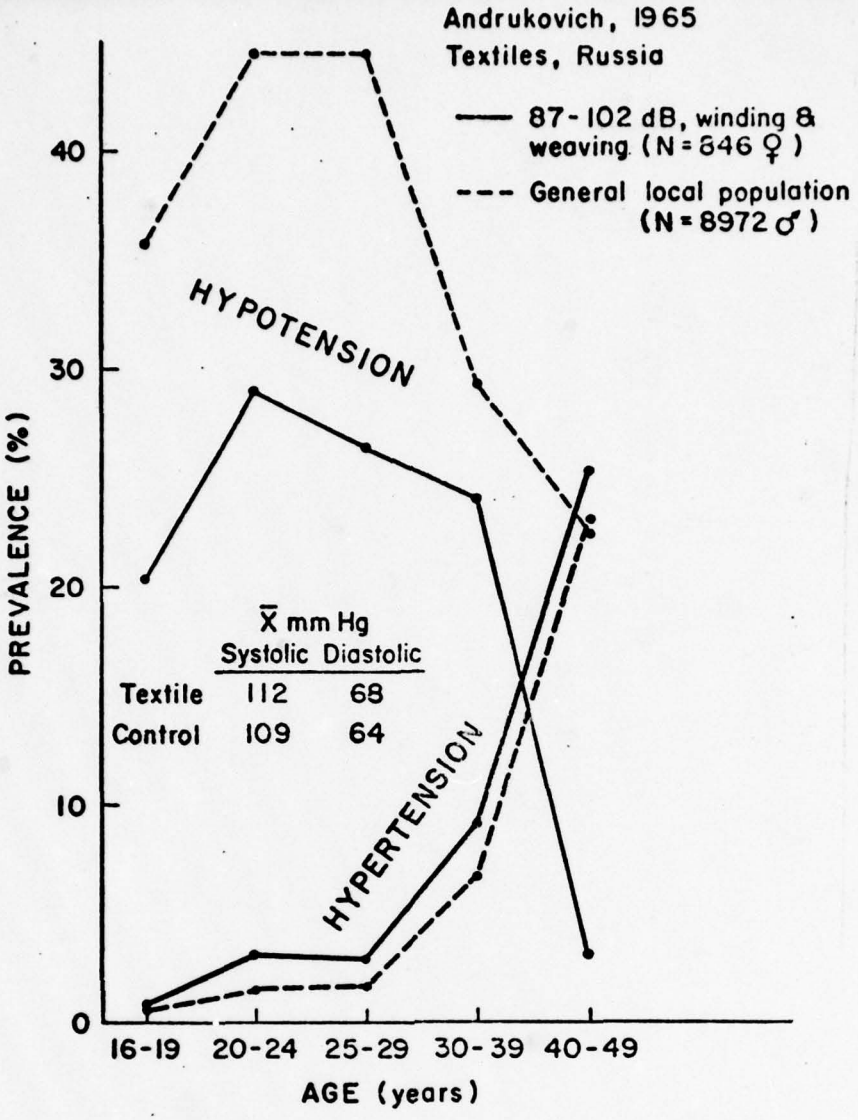


Figure 2. Hypotension prevalence less (descending curves) and hypertension prevalence greater (ascending curves) among 846 female winding and weaving machine operators exposed at work for 1-5 years in Russian textile mills to 87-102 dB high frequency impulse noise (solid lines) than among 8972 females of the same general local population (dashed lines). Blood pressures measured 4-8 times over a three year period. Chi square (one degree of freedom) highly significant for the overall difference in prevalence of hypotension, either systolic ($X^2 = 19.4$, $p < .001$), or diastolic ($X^2 = 30.1$, $p < .001$) or both systolic and diastolic hypotension in the same individual ($X^2 = 12.8$, $p < .001$). The difference in prevalence of hypertension is not significant for people having either systolic or diastolic hypertension alone but it is highly significant for people having both systolic and diastolic hypertension ($X^2 = 10.8$, $p < .001$). In the figure all forms of hypertension and hypotension (systolic, diastolic both systolic and diastolic) are combined. In this uniquely hypotensive population, the large decrease in hypotension and small increase in hypertension increased normotension from 57.9 percent in the local population to 68.8 percent among the textile workers ($X^2 = 37.9$, $p < .001$). Hypertension defined as systolic and/or diastolic $\geq$ 140/85 mm Hg, hypotension as systolic and/or diastolic $<$ 90/50 mm Hg for ages 16-19 years or $<$ 100/60 mm Hg for ages 20-49. Population average blood pressures were higher for the textile workers but the differences small: systolic 112.7 ± 0.42 vs. 108.9 ± 0.14 mm Hg ($t = 8.7$, $p < .01$), diastolic 68.3 ± 0.3 vs. 64.2 ± 0.12 mm Hg ($t = 12.7$, $p < .001$). Adapted from Andrukovich, 1965 (2).

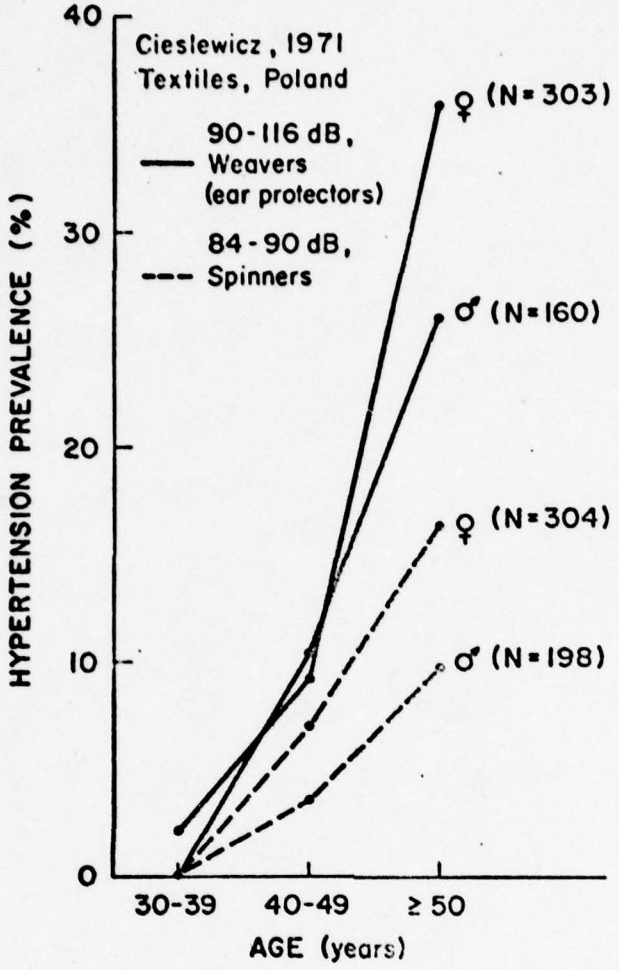


Figure 3. Hypertension more prevalent among 160 males and 303 females routinely exposed at work to 90-116 dB medium frequency sound in Polish weaving mill (solid lines) than among 198 males and 304 females routinely exposed to 84-90 dB in spinning mill (dashed line). For ages $\geq$ 40 years, Chi square (one degree of freedom) = 5.48 ($p < .02$); for ages $\geq$ 50 years, $X^2 = 6.46$ ($p < .02$). Blood pressure not measured under 30 years of age. Hypertension defined as systolic and/or diastolic blood pressures > 150/99 mm Hg. Ear protectors worn in weaving mill. In weaving mill, 85 percent of subjects had been on the job > 5 years, 43 percent > 15 years. In the weaving mill 64 percent were under 40 years of age, but only 40 percent in the spinning mill. Hypertension in weaving mill not significantly correlated with job classification, living conditions, smoking or distance of residence from work. Claim morbidity differences observed minimal since people who transfer to more desirable jobs, are relieved for disability or quit were not counted in weaving mill morbidity; in the spinning mill those who change jobs normally stayed within the same work environment and were included. Says that hypertensive medication that is effective in spinning mill was not effective in the weaving mill unless persons are also removed from the work environment, but no data. Adapted from Cieslewicz, 1971 (5).

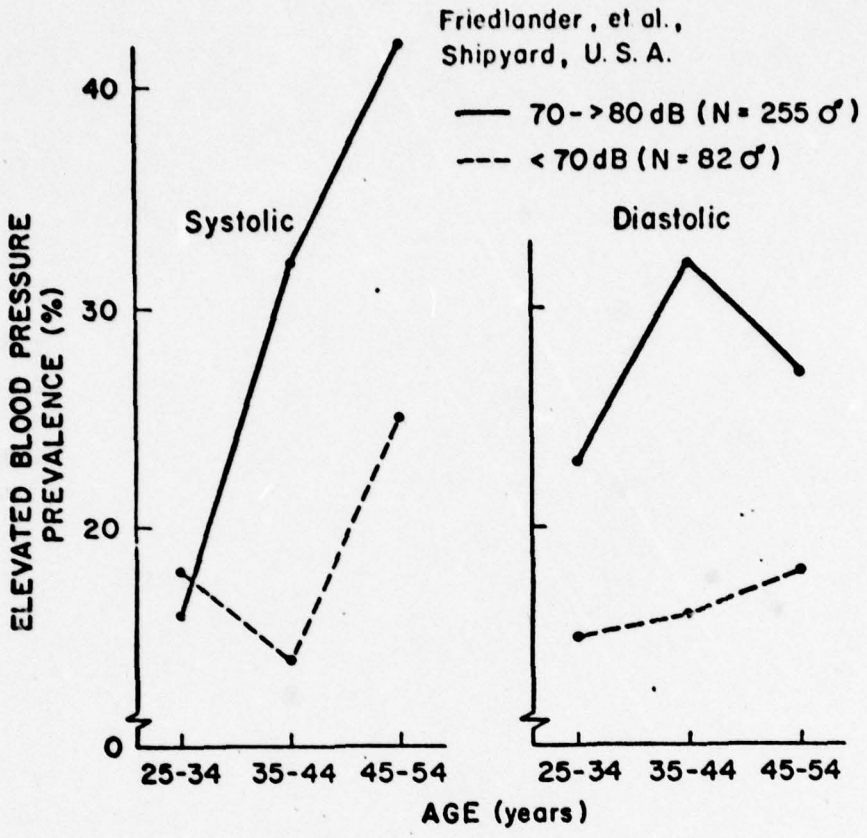


Figure 4. Elevated blood pressure, systolic and diastolic, more prevalent among 255 men working for at least 5 years under intermittent 70 dB to > 80 dB sound (solid lines) than among 82 men working under < 70db sound (dashed lines) in an American shipyard. Data from medical records. Elevated blood pressure defined as systolic and/or diastolic > 140/90 mm Hg. Subjects screened if hypertensive on first physical examination after employment. For ages 35-54 combined, overall Chi square (one degree of freedom) for comparing systolic blood pressures = 8.79 ($<.01$); for comparing diastolic pressure, $X^2 = 9.85$, $p < .01$). Adapted with permission from Friedlander et al., undated (9).

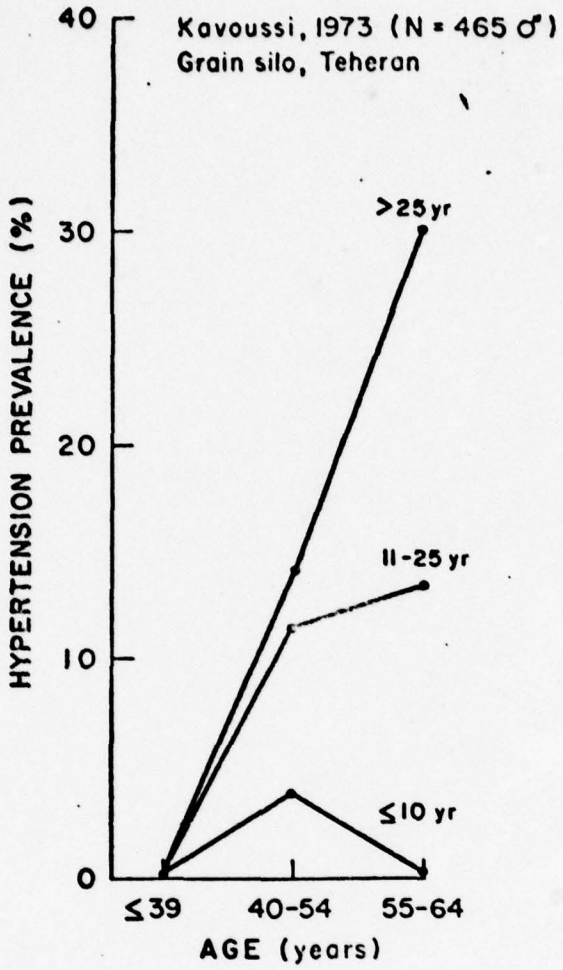


Figure 5. Hypertension prevalence increased with increasing years on the job for 465 men working in a grain silo in Teheran. Hypertension is significantly more prevalent among people aged 55-64 with $>$ 25 years job experience than with $\leq$ 25 years job experience ($p < .043$, Fisher exact probability). Years of job experience indicated on the curves. Sound said too loud for speech comprehension, but not measured. Hypertension defined as systolic and/or diastolic $>$ 140/90 mm Hg. Adapted from Kavoussi, 1973 (18).

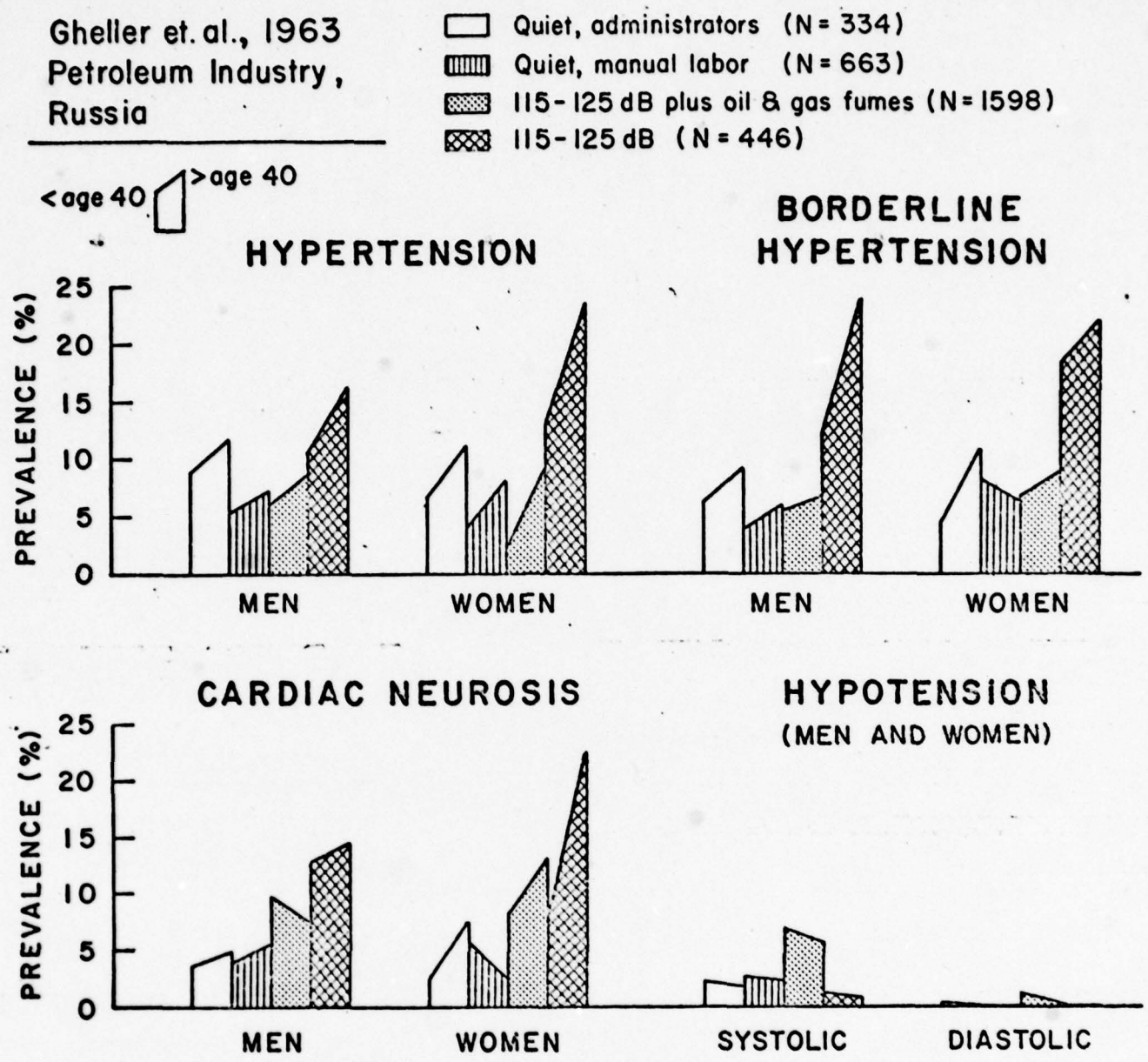


Figure 6. Hypertension, borderline hypertension and cardiac neurosis more prevalent, hypotension less prevalent, among 446 petroleum industry employees who had routine work exposure to continuious 115-125 dB, 2.4-6.0 kHz sound (solid column) than among 663 manual laborers (vertically striped columns) or 334 administrators (horizontally striped columns) not exposed to noise, or 1598 workers exposed to noise plus oil and gas fumes (stipled columns), a combination that was somewhat hypotensive. Left side each columnm under 40 years of age; right side over 40 years of age. All major comparisons claimed statistically significant. Hypotension taken as systolic and/or diastolic $\leq$100/60 mm Hg. Hypertension and borderline hypertension undefined. Adapted from Gheller et al., 1963 (10).

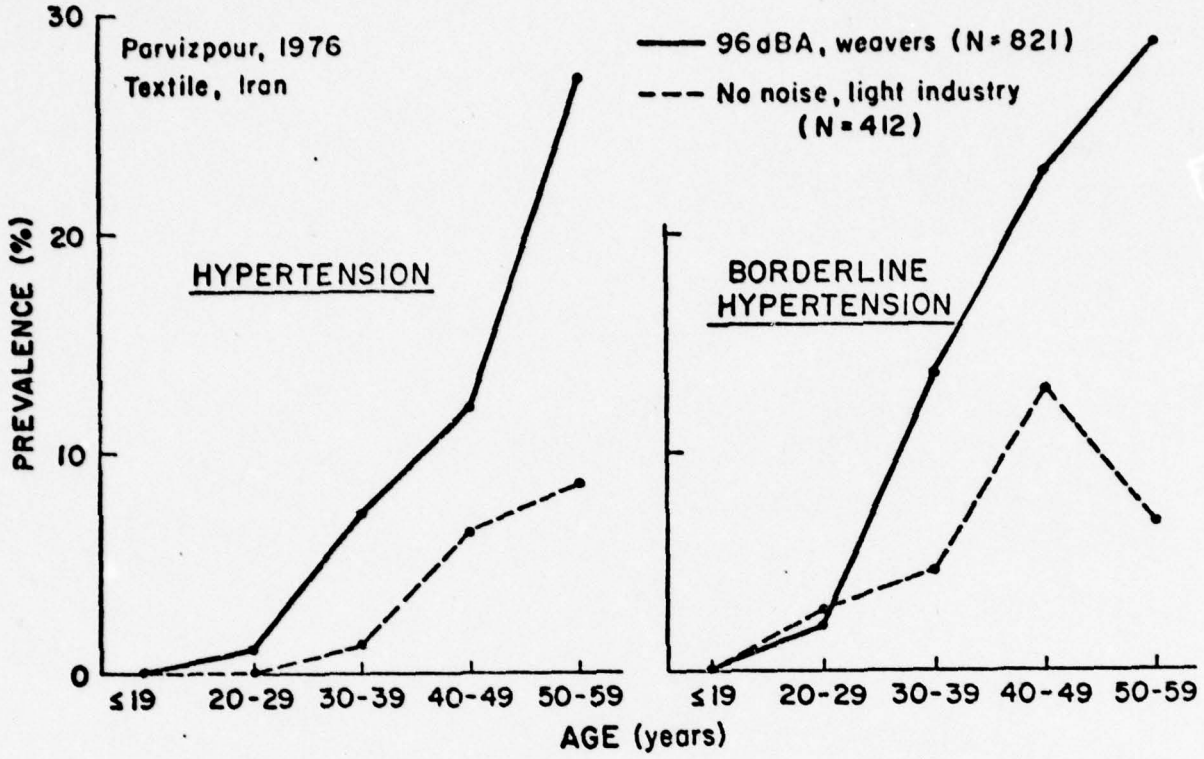


Figure 7. Hypertension and borderline hypertension more prevelant among 821 male Iranian weavers who worked under an average sound level of 96 dBA (solid lines) than among 412 controls of similar socio-economic background who worked in light industry in the same general population area without intensive noise exposure (dashed lines). For hypertension, Chi square (one degree of freedom) = 16.8 ($p < .001$); for borderline hypertension, Chi square = 18.9 ($p < .001$). Hypertension defined as systolic/diastolic $\geq$ 160/95 mm Hg, borderline hypertension 140-159/90-94 mm Hg. Workers whose family history suggested high risk for hypertension were excluded from the study. Adapted from Parvizpoor, 1976 (22).

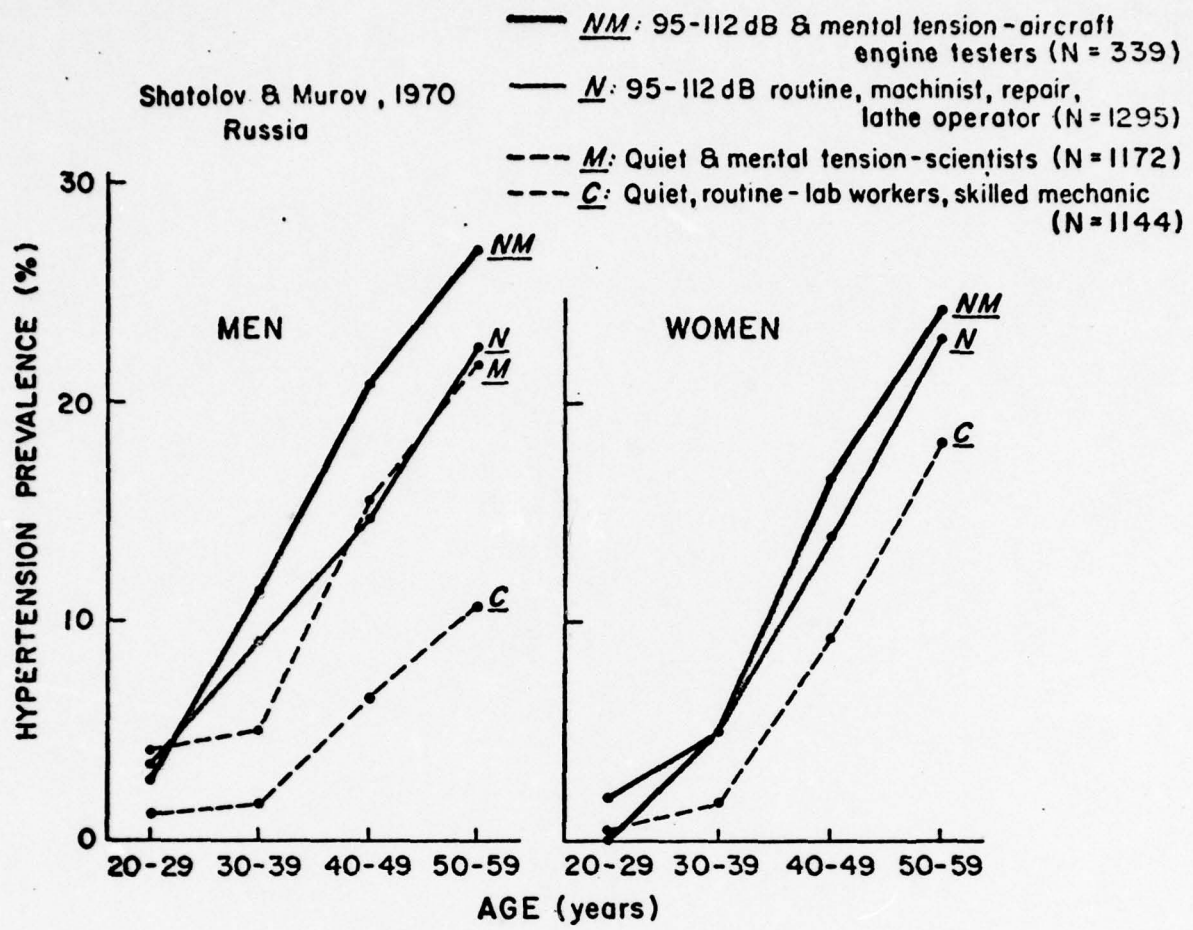


Figure 8. Hypertension prevelance less among 1144 Russian controls whose work involved neither noise nor mental tension (C, routine laboratory workers and skilled mechanics) than among 1275 people doing light routine work involving exposure to 95-112 dB broad band high frequency sound but no mental strain (N, machinists, lathe operators, repair men, etc.) or 339 aircraft engine testers whose work involved similar noise plus intense mental concentration (NM). Prevelance among 1172 scientists whose work was said to involve mental tension but no noise (M) was similar to N. Chi square (one degree of freedom) for overall difference in prevelance is significant for men for N vs. C (13.1, $p < .001$), NM vs. C (27.1, $p < .001$), and NM vs. N (4.3, $p < .05$); for women for N vs. C (5.4, $p < .05$). Population mean blood pressures also differed significantly but were quite small (7-9 mm Hg systolic, 4-6 mm Hg diastolic). Criteria for hypertension undefined. Adapted from Shatalov and Murov, 1970 (28).

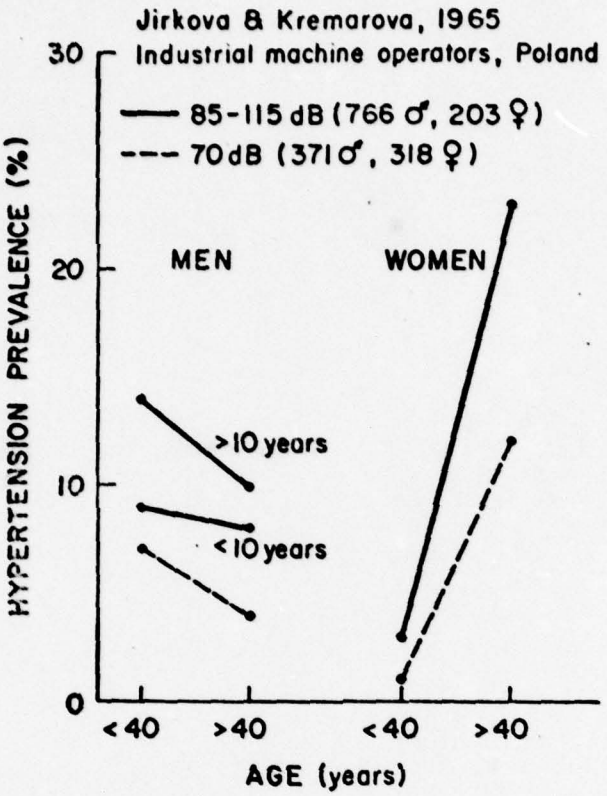


Figure 9. Hypertension more prevalent among 969 Polish industrial machine operators in 34 work sites exposed to 85-115 dB sound, dominant frequency about 1.0 kHz (solid lines), than in 687 people in non-noisy worksites (dashed lines). Attempt made at all sites to exclude effects of other physical factors, toxic substances and emotional distress. Sample allocation in age and experience categories not given, but assuming realistic allocations in Chi square analyses differences seem significant. Thus, for men $<$ 40 years of age, a comparison of controls vs. subjects with $>$ 10 years of occupational noise attains significance ($p < .05$) by a two-tailed test assuming sample sizes of 150, or by a one-tailed test assuming 110 per treatment. For men $>$ 40 years of age this comparison attains two-tailed significance with sample sizes of 140 and one-tailed significance with 100 men per treatment. For women over age 40, sample sizes of 95 achieve statistical significance for a two-tailed test, samples of 65 with a one-tailed test. Adapted from Jirkova and Kremarova, 1965 (13).

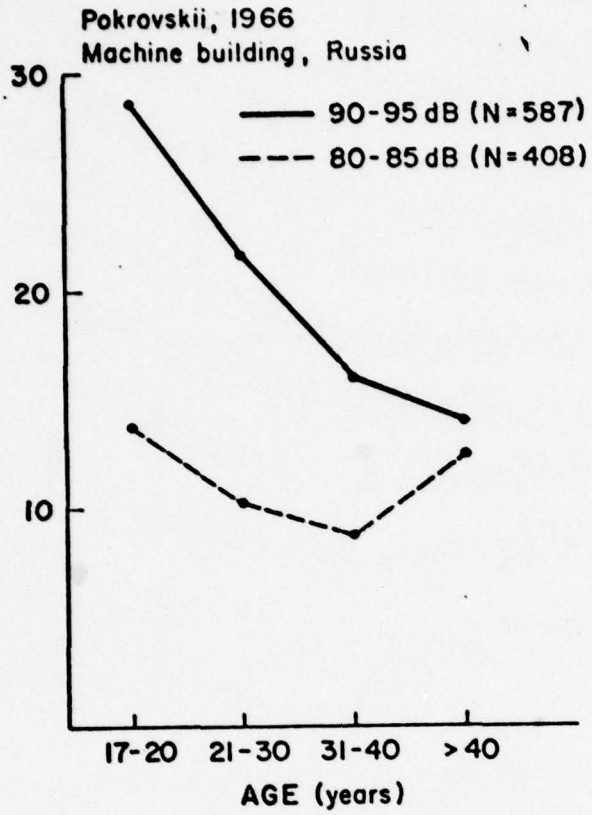


Figure 10. Blood pressure abnormalities more prevalent in Russian machine building plants among people routinely exposed to 90-95 dB high frequency impulse sound (solid line) than among 408 people routinely exposed to 80-85 dB stable mid-frequency sound (dashed line). The dominant abnormality was hypotension in the 17-20 year age class, hypertension in older age classes. For the difference in abnormality prevalence over all age classes, Chi square (one degree of freedom) = 11.21, $p < .001$; for ages $\geq$ 21 years, $X^2 = 9.51$, $p < .01$. People working under the higher level noise were about five times as likely to have either marked tachycardia of marked bradycardia as controls and their blood pressure demonstrated much greater fluctuations during the course of the day. Adapted from Pokrovskii, 1966 (24).

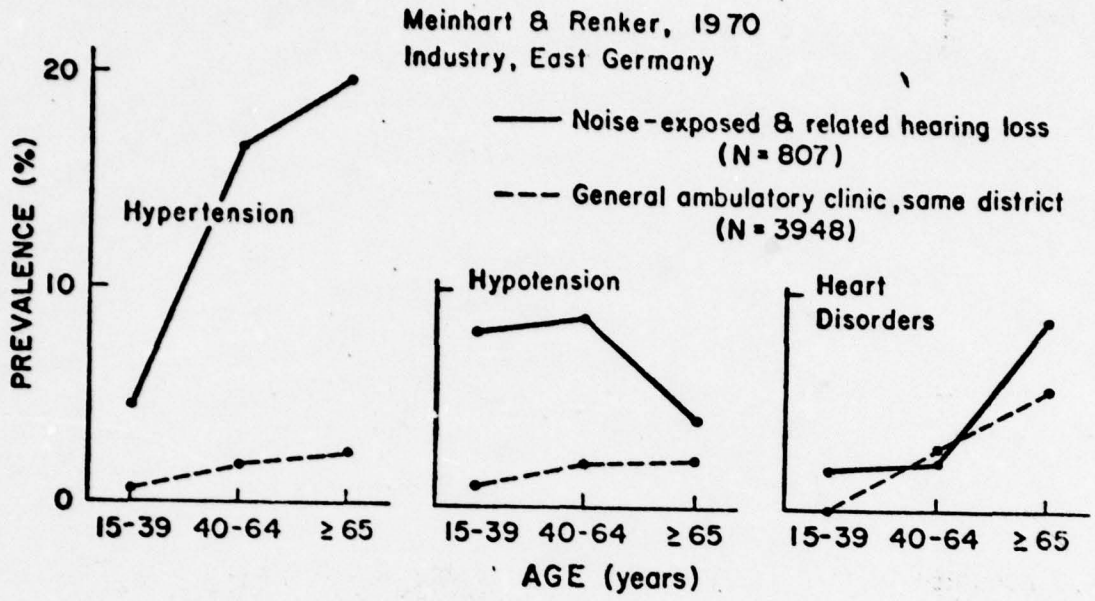


Figure 11. Hypertension and hypotension prevalence for all 807 men in the District of Halle, East Germany, who were recognized as having developed neurosensory deafness ensuant to long-term employment under industrial noise significantly greater than among 3948 male patients in communal ambulatory clinics of the same district. For all age classes general cardiovascular morbidity began rising after about 5 years of work under noise and rose precipitiously after about 20 years. Hypertension among the hearing impaired men exceeded that of ambulatory clinic patients at a very high level of statistical significance in each age class, and hypotension was also significantly more prevalent at all ages except the most advanced. Heart disease (coronary sclerosis and myocardial disease of all kinds except congenital and rheumatic considered as a group) was, overall, about twice as common in the hearing impaired men as in the controls, a difference the authors claim highly significant statistically; my analysis suggests no statistical significance. There was no difference in the prevalence of peripherial circulatory disease or functional cardiovascular disease. Adapted from Meinhart and Renker, 1970 (20).

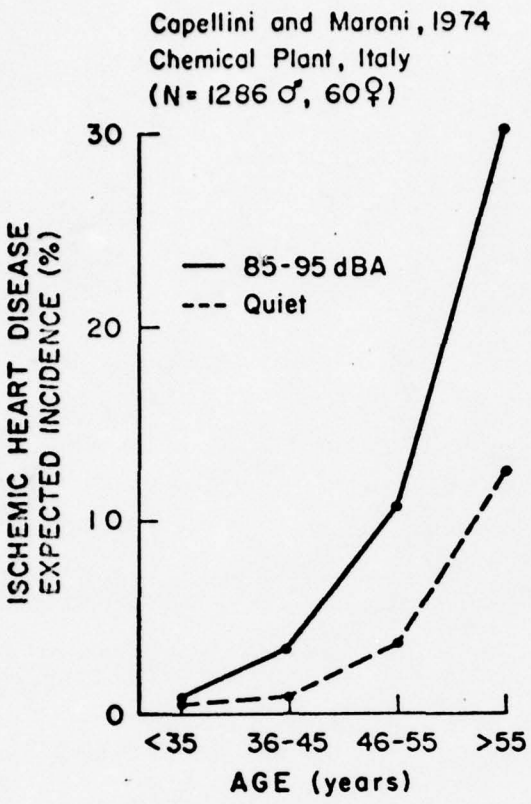


Figure 12. Ischemic heart disease among 1365 people comprising 98.6 percent of the workforce of an Italian chemical plant said significantly correlated with only one environmental factor, namely, 85-95 dB sound. Not correlated with emotional distress, chemical exposure, physical exertion, temperature, work rythmn, repetitiveness of work, vibration, illumination, work schedule or pay level. Diagnosis confirmed by history of prior infarct and/or electrocardiographic or vectorcardiographic signs of myocardial necrosis; active angina pectoris; or exercise stress test precipitating an anginal attack and/or a rectilinear or descending S-T segment depression $\geq$ .1 mV in a standard 12-lead electrocardiogram. Graphed is expected incidence of ischemic heart disease derived by author from log transformation and weighted regression of the data. It suggests that people working under noise had an expected incidence of ischemic heart disease roughly equivalent to that of a person a decade older who was not working under noise. Hypertension was not significantly correlated with noise or with any other factor. Reproduced with permission from Capellini and Maroni, 1974 (4).

Figure 13. Anomalies in standard 12-lead electrocardiogram taken at rest and classified according to the Minnesota Code (67), more prevalent among 160 people (93 percent male) who had done light manual labor for an average of 7 years under 85-106 dB broadband (63-8000 Hz) sound in a Roumanian rubber plant (13 percent air compressor operators, 12 percent stokers, 34 percent mechanics, 29 percent electricians and 12 percent laboratory assistants) than among 160 non-noise-exposed controls (93 percent male) also doing light manual labor in the same plant.

Cardiac hypertrophy and repolarization anomalies borderline between normal and pathological which suggest evolution towards lesions, but no organic lesions detected. Retarded conductivity of one kind greater in noise subjects and, to a lesser degree, another kind in controls.

Four codes more common under 85-95 dB than under control conditions (code 4-4, $X^2 = 34.4$, $p < .001$; code 9-2, $X^2 = 13.1$, $p < .001$; code 9-2-1, $X^2 = 30.8$, $P < .001$; code 9-4-1, $X^2 = 23.3$, $p < .001$) but show no significant further increase under 95-106 dB sound. One, code 3, not significantly more common under 85-95 dB sound than for controls, bit its risk under 85-95 dB sig-ificantly exceeded by a factor of 2.1 under 95-106 dB sound ($X^2 = 4.1$, $p < .05$). Code 7-5 more common in controls ($X^2 = 9.0$, $p < .01$). Uncoded change suggesting "minor" right bundle branch block in 3.8 percent of noise subjects but in no controls ($X^2 = 4.2$, $p < .05$).

Labile hypertension (systolic and/or diastolic > 140/90 mm Hg during work but subsiding to lower levels after work) 5.6 percent in noise, none in controls ($X^2 = 7.3$, $p < .01$). Hypertension Stage I greater prevalence for the most extreme noise exposure, 21 air compressor operators working under 95-106 dB continuious sound, than for controls (14.7 percent vs. 2.6 percent, $X^2 = 4.1$, $p < .05$) and greater than for people working under 85-95 dB sound ($X^2 = 8.8$, $p < .01$). These operators did not experience temperature or humidity extremes.

Individuals with known exposure to toxic substances or family history of cardiovascular disease excluded a priori. Smokers, obese people and those on high fat diets equally represented in noise and control groups and the association of ECG anomalies with these risk factors, and with combinations thereof, similar in noise-exposed and control subjects. Noise exposed subjects 66 percent $\leq$ 40 years of age, average age 35 years: average 29 years $\leq$ age 40, 46 years $>$ age 40 years. Controls 64 percent $\leq$ 40 years of age, average age 36 years: average 30 years $\leq$ age 40, 46 years $>$ age 40 years.

Code 3: High amplitude R wave suggesting left ventricular hypertrophy. Here, 86 percent clinically significant, not normal variant.

Code 4-4: S-T j-junction depression 0.1 mV with ascending orientation or "U" shape. May be a normal exercise response, but these subjects at rest. May precede true ischemic S-T segment depression during exercise in patients having a normal resting ECG but significant coronary artery disease. Among 32 subjects in this study on whom radio-ECGs were taken while actually at work under noise was one 52-year-old woman with resting code 4-4 who was observed experiencing an angina pectoris attack with upward deflection of the S-T segment indicative of subepicardial ischemia.

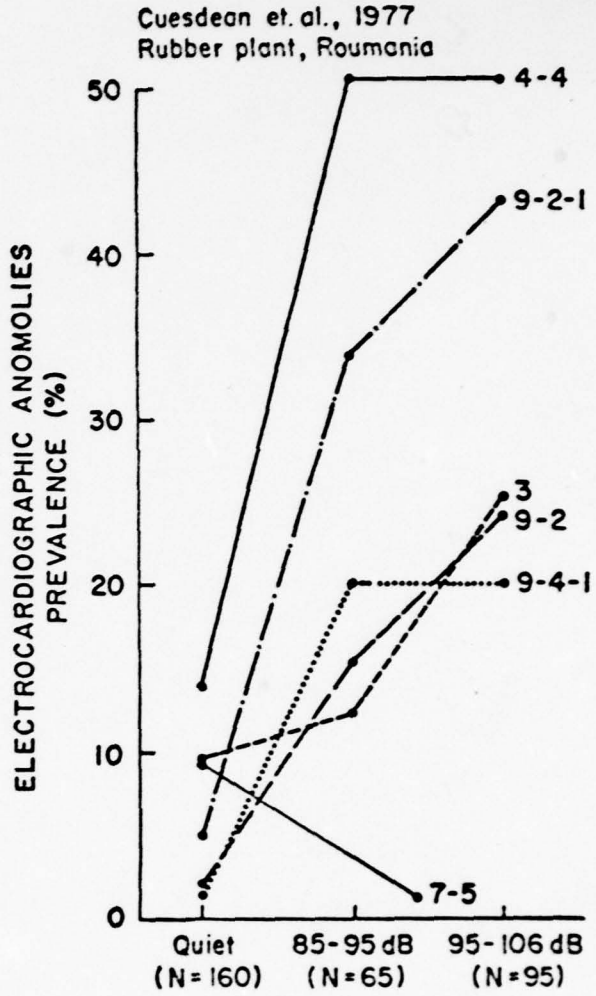
Code 9-2: Maximium upward deflection of the S-T segment, commonly reflecting post-infarction cardiac ischemia but may be functionally related to exercise or hyperventilation. Here, anteriolateral or inferior localization suggested the "benign" type.

Code 9-2-1: Maximium upward S-T deflection on at least two leads and at least 0.5 mm in I, II, III, aVL, V5, V6 or at least 1 mm in V1, V2, Vs, V4. Common incidence about 2.4 percent of normal healthy men (68), a strong contrast to the 40 percent of n-ise exposed men both above and below age 40 years here.

Code 9-4-1: Poor R-wave progression across precordium, shifting QRS transition or null zone to the right of lead V3 on the cheast. Suggests prior anterio-septal infarct, but not always diagnostic unless R-wave actually disappears. Here, half of subjects with labile hypertension had this code.

Code 7-5: Prolonged QRS suggesting retarded intraventricular conductivitty.

Adapted from Cuesdean et al., 1977 (6).



Welch 58

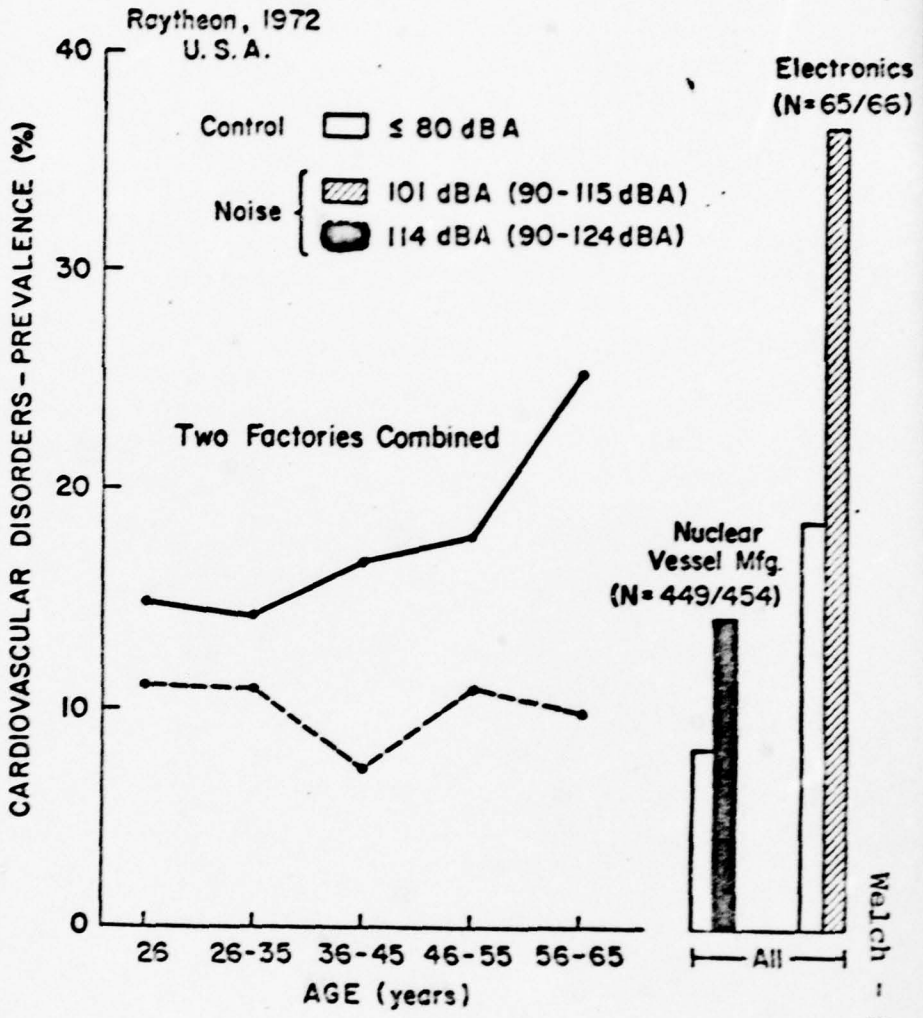


Figure 14. Cardiovascular/circulatory disorders more prevalent among people exposed at work for at least 5 years, median time about a decade, to 95-130 dBA industrial sound than among controls exposed to $\leq$ 80 dBA in two factories in the United States, as judged by retrospective analysis of five-year medical records by the Raytheon Corporation. At right, the overall prevalance of disorders is 70 percent greater under high than low noise in a nuclear vessel fabrication facility ($p < .01$), and twice as great in an electronics factory ($p < .01$). At left, data are pooled and plotted for all 515 high-noise exposed people and all 519 low-noise-exposed people in both factories, showing the prevalence of cardiovascular/circulatory disorders to be greater among high noise workers within each age group and the overall difference to be highly significant (for one degree of freedom, $\chi^2 = 12.3$, $p < .001$). Records had been maintained in a reasonably comprehensive and consistent manner by professionals. Records of people found on pre-employment medical examination to have active health orders were excluded. Double blind techniques were used. Environmental factors other than noise were said to be well under control and not at variance with the NIOSH Act of 1970, and bias by such factors or by socioeconomic differences was said to be minimized by rigorious sampling and matching. Records were matched by job description, length of employment, age, sex, race, martial status and work shift; but inherent task differences made perfect job-for-job matching impossible. "Cardiovascular/circulatory" disorders were broadly defined to include "tachycardia, angina pectoris, hypercholesterolemia, 'hemotrypsia', coronary sclerosis, hypertension and hypotension," and breakdowns were not given for individual factors. Cardiovascular/circulatory was the only one of eight broad diagnostic categories evaluated in which a significant difference between high and low noise exposure was found in both factories. The largest differences were found between administrators working under high and low intensity noise, thus demonstrating that the observed effects were not likely due solely to non-noise factors associated with the performance of physical work. Adapted from Raytheon, 1972 (25).

Appendix I

Hypertension Not Predicted By Hearing Loss

Although hypertension and hearing impairment both tend to increase during long-term occupational noise exposure a correlation between them cannot necessarily be assumed. Consequently, a lack of correlation between hearing impairment and hypertension is not evidence, in and of itself, for a lack of relation between noise exposure and hypertension.

Three recent Scandinavian studies of the epidemiological correlates of hypertension attempted to use hearing impairment either wholly or partially as an indicator of past noise exposure (43-45). All three used the same criteria for hypertension (systolic and/or diastolic $\geq$ 160/100 mm Hg); for severe hearing impairment ($\geq$ 65 dB at 3, 4 or 6 kHz); and for normal hearing ($<$20 dB at all tested frequencies). All three studies were *a priori* biased against detecting significant blood pressure differences due to noise exposure by the assumption that any subject whose hearing loss was less than 20 dB at all tested frequencies therefore had no prior noise exposure that could have significantly elevated blood pressure. A much smaller hearing loss than 20 dB, even at the most sensitive frequency of 4 kHz will be sustained by the vast majority of people who work for 40 years under 90 dBA sound for eight hours daily, the maximium exposure that United States law for industrial noise control permits. A lifetime of work under 85 dBA, eight hours per day, is expected to lower hearing by 19 dB at 4 kHz in only 10 percent of an exposed population (66). Clearly, a hearing loss less than 20 dB does not necessarily imply a lack of significant prior exposure to noise, and in none of these studies were the controls shown to lack significant prior noise exposure.

Jonsson and Hansson (43) identified 44 male Swedish industrial workers aged 41-66 years, mean 57 years, who suffered hearing impairment and who had experienced considerable noise exposure during past employment in shipyards, mechanical workshops, etc. These men had significantly higher systolic and diastolic blood pressures (by 12 mm Hg and 8 mm Hg, respectively), and a significantly higher prevalence of hypertension (22.7 percent vs. 8.1 percent) than 74 men with

normal hearing in the same industrial population and within the same age range, but averaging three years younger. This study was biased against finding this difference in blood pressure by the failure to demonstrate lack of significant noise exposure for controls and biased in favor of finding the observed difference by the relative youth of the controls.

Commenting on this study, Takala et al. (44) compared 32 men of average age 47 years "whose work exposed them to noise and who had impaired hearing" with 67 normal hearing men averaging 45 years of age in the same municipal population in southwest Finland. These subjects averaged 10 years younger than Jonsson and Hansson's, making it less likely that any potential difference in blood pressure due to noise would be as fully developed. Mean systolic and diastolic blood pressures of men with impaired hearing averaged only a non-significant 4 mm Hg greater than those of men with normal hearing. The prevalence of hypertension was considerably higher for those with impaired than normal hearing (34.4 percent vs. 20.9 percent), a difference that lacks significance by a conventional two-tailed hypothesis ($X^2 = 2.08$), but which is significant ($p < .045$) by the not wholly unjustified one-tailed hypothesis that men with occupational noise exposure are more likely to be hypertensive. If the same difference were to obtain for samples twice as large it would be significant by a two-tailed test. This study, like Jonsson and Hansson's deserves criticism for using relatively small samples, for giving inadequate information on the noise history of the subjects with impaired hearing and none on that of the normal hearing subjects, and for using sunjects with normal hearing who were 2-3 years younger then the subjects whose hearing was impaired.

Also commenting on Jonsson and Hansson's study, Hedstrand et al. (45) compared 376 men who had "normal" hearing and 383 with impaired hearing, all 49-50 years of age and all from the general population of Uppsala. They reported that the average systolic and diastolic blood pressures for the two groups differed by less than 1 mm Hg and that the prevalence of hypertension was also virtually identical (6.4 vs. 6.6 percent). But, unlike in the preceding studies, the hearing impaired subjects were not actually known to have a history of noise exposure: this was simply assumed on the basis of hearing

loss of the "noise exposure type" - and that, it is widely recognized, is not actually specific for noise (48-53); Glorig labels the belief that it is specific a "myth" (53). This audiometric profile of hearing loss can be caused by many other things than noise, and hearing loss associated with long-term noise exposure does not necessarily have this profile (54). That many of Hedstrand et al.'s (45) hearing impaired subjects did not in fact have appreciable noise exposure is suggested by the fact that the ratio of persons with impaired hearing to that of persons with normal hearing was twice as large in their study as in the two aforementioned studies (43, 44) in which prior occupational noise exposure for hearing impaired subjects was known.

In an earlier report based upon the same general health survey of men aged 49-50 years in the population of Uppsala, Hedstrand and collaborators (46) compared 388 subjects who claimed "a history of civil and/or military noise exposure" with 374 subjects who "denied such exposure." No significant differences were found in average values for systolic or diastolic blood pressures (or serum cholosterol, triglycerides, uric acid, hematocrit or glucose tolerance). But the statement later made by the authors that "no correlation between blood pressure and noise trauma was found" (45) seems unjustified if used to imply - as it was - that no such correlation existed in their study population. No actual data were given on blood pressures or on the prevalence of hypertension, and if the two groups were compared on the basis of distributional extremes as well as average blood pressures this was not stated. An element of bias against the possible detection of a difference in blood pressure between the compared groups was also inherent in the facts: that the subjects were self-selected into the study by responding positively to an invitation sent by mail; that it was established that individuals who did not respond were more likely to be unmarried and of low socioeconomic status; and that unmarried persons and those of low socioeconomic status have a high probability of having hypertension or other cardiovascular disorders (89, 90). The actual nature or duration of the claimed noise exposure was not given, nor was it stated to what extent it was occupational. Most important, an adequate differentiation apparently was not made between people

who had actually sustained significant past noise exposure and those who had not: the authors acknowledged that "most of the subjects who denied noise exposure had nevertheless had a period of military service"; also, left-sided inferiority of hearing, commonly attributed to noise, especially the use of firearms, was common to both people who claimed past noise exposure and those who denied it. The difference in median threshold of hearing between 160 subjects who claimed noise exposure and 197 who claimed none (the only subjects for whom data were actually given), averaged over both ears, was small: the maximium difference, 9.3 dB, was at 4000 Hz and was said statistically significant, but the difference was only 3.8 dB at 3000 Hz and only 0.2 Hz at 2000 Hz; at the latter, a key frequency for speech reception, an elevation of threshold $\geq$ 25 dB is commonly considered necessary to warrent the judgement of handicapping hearing impairment. The actual difference in past noise exposure of the two groups compared in this study was apparently only nominal, and was in no sense comparable to that attained in studies of people who work for prolonged periods of time under high intensity noise.

Paranthetically, these authors also found no difference in the hearing ability of men classified at "high risk" and "low risk" for cardiovascular disease (46). But while this may reflect reality, again, their data need not necessarily be interpreted to have discounted the possibility that such difference may actually have existed in the study population. The actual differences in the risk factors of the compared groups were too small, except for smoking ($\geq$ 10/day for at least 10 years vs. no smoking), for much of a difference to have been expected (systolic blood pressure 10 mm Hg, diastolic blood pressure 5 mm Hg, serum cholosterol 20 mg/100 ml), and the samples were small, namely, only 28 men and 35 men, respectively, in the two extreme "risk" categories compared. That the comparison was rendered unduly insensitive by the small sample sizes employed is suggested by the fact that when more ample samples of 92 smokers and 105 non-smokers in the "non-noise-exposed" population were compared the smokers had significantly poorer hearing. These observations, inaccurate reference citations and other apparent discrepancies render this study (45-47) difficult to interpret and discourage giving it heavy weight in

judgements about the potential relationship between hypertension, hearing impairment and noise. In perspective, this Uppsala study offers no data relevant to the hypothesis that long-term exposure to industrial noise tends to increase the prevalence of hypertension, while Takala et al. (44) and Jonsson and Hansson (43) offer tentative, very limited, support.

That hearing impairment and elevated blood pressure are not necessarily correlated in these three Scandinavian studies is particularly apparent from the facts that: (i) although Takala et al's (44) normal hearing subjects averaged 12 years younger than Jonsson and Hansson's (43) hearing impaired subjects, their average blood pressures and prevalence of hypertension were comparable; and (ii) although Hedstrand et al.'s (45) hearing impaired subjects averaged 4.5 years older than Takala et al.'s normal hearing subjects, the latter had systolic and diastolic blood pressures that were higher by 19 mm Hg and 11 mm Hg, respectively, and their prevalence of hypertension was 3.2 times as great.

Other studies similarly show elevated blood pressure and hearing loss correlated with long-term noise exposure but not necessarily correlated with one another. In an earlier but much more adequate East German study, Meinhart and Renker (20) compared all 807 men in the District of Halle who had been recognized as having neurosensory hearing loss ensuant to long-term exposure to industrial noise with morbidity statistics for 3948 male patients in ambulatory clinics of the same district. Occupational noise history was known. For all age classes, cardiovascular morbidity rose after about 5 years of employment in a noisy job and rose precipitiously after about 20 years. The prevalence of hypertension among the hearing impaired men strikingly exceeded that of ambulatory clinic patients in every age class: by 7.7 times for ages 15-39, by 9.7 times for ages 40-64 and by 8.2 times for ages 65 years and older. But <u>hypotension</u> was also increased, its prevalence in hearing impaired subjects exceeding that in ambulatory clinic patients by a factor of 8.2 for ages 15-29, 4.4 for ages 40-64 and 1.9 for ages 65 years and older. Except for the last, all of these differences were statistically highly significant. (Average blood pressures were not given, but they would have averaged somewhat higher in the

hearing impaired subjects since hypertension was about twice as common as hypotension; nevertheless, the difference may well have been very small and statistically insignificant due to the bimodality of the blood pressure distribution.) One may assume that the elevated blood pressures reflected the noise exposure and were only incidently correlated with impaired hearing.

Shatalov et al. (29) compared 521 men and 285 women who worked under 90-120 dB high frequency sound in Russian mechanical engineering and synthetic fiber plants with 161 men and 49 women who worked in quiet areas of the same plants. Although systolic and diastolic blood pressures and the prevalence of hearing impairment were all significantly elevated for subjects working under noise, both below and above 40 years of age, blood pressure was elevated to about the same degree whether hearing was impaired or not and its degree of elevation bore no relation to the severity of hearing impairment. The possibility has not been excluded that hypertension and hearing impairment occur, at least in part, in different sectors of a noise exposed population.

Cohen et al. (40), in the midwestern United States, recently compared 51 people of average age 47 years who had worked for an average of 22.3 years in a paper-making plant under 85-102 dBA sound with 34 others who were doing similar work in the same plant, but who averaged 13 years younger, who had been on the job 10 years less and whose hearing was not impaired. Despite the greater age and time on the job of the hearing-impaired subjects, blood pressures were virtually identical in the two groups. This study reinforces the expectation that no relationship need necessarily be expected between hearing loss and elevated blood pressure in industrial populations. It also appears, superficially, to contradict the expectation of a hypertensive effect of long-term occupational noise exposure and, indeed, could even be interpreted to suggest a protective effect against the increase in blood pressure that normally occurs with advancing age. This has been discussed in greater detail on pages 5-7.

In studies in which noise was not considered, hearing loss and hypertension commonly have been found unassociated. Hugson and Thompson (56) found 285 patients with impaired hearing to have blood pressures generally in the low-normal range for their age. Richter-Heinrich et al. (57) reported lower auditory thresholds in hypertensives than in normotensives. Hensen (58) found no systematic differences in hearing loss of 263 normotensive patients and 79 hypertensives, all over 45 years of age. Bunch (59) found age-related hearing loss similar in 468 patients whether they had hypertension, arteriosclerosis, chronic heart conditions, cancer or syphilis. Crowe et al. (60), in their landmark studies correlating high tone hearing loss with histological change in the cochlea, often found normal-looking blood vessels to co-exist with total atrophy of the organ of Corti and concluded that this atrophy was not caused by arterio- or atherosclerosis. As Fowler (61) emphasized, there are many people in their 80's and above who in spite of severe generalized arteriosclerosis have essentially normal hearing in all clinical audiometric frequencies. In industralized societies hearing loss, hypertension and systemic vascular sclerotic change all tend to be concomitants of advancing age, but they need not be associated (48, 55, 61). Similarly, they may be unassociated concomitants of long-term exposure to industrial noise.

There is evidence that the circulation of the inner ear is inherently inadequate to support the intense metabolism induced in sensory cells by loud noise for prolonged periods of time; very loud noise, therefore, may rapidly impair hearing although no abiding systemic hypertensive or other vascular change may have occured. On the other hand, systemic vascular disease may cause hearing loss and may also increase vunerability to the impairment of hearing by noise. Detailed discussion of the evidence for these points is beyond the scope of this review.

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