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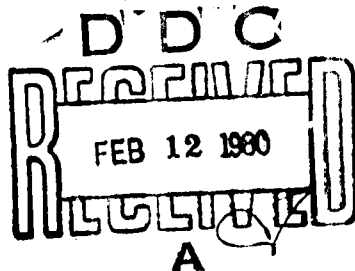
## BEHAVIORAL AND PHYSIOLOGICAL EFFECTS OF NOISE ON PEOPLE

A review of the literature

DR Lambert  
NOSC  
FS Hafner  
San Diego State University

30 April 1979

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US Coast Guard  
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## FOREWORD

This document has been prepared for the US Coast Guard, Office of Research and Development, and for Naval Sea Systems Command (NAVSEA 05H) for general guidance in the development of noise standards for US merchant ships. It is one of several dealing with various aspects of noise as related to habitability and the safety of personnel aboard merchant ships.

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## 1 EFFECTS OF NOISE ON HEARING

### INTRODUCTION

The Environmental Protection Agency (EPA) estimates that 20 million people in the United States are exposed to noise levels that are hazardous to hearing (ref 1). Noise can permanently impair various aspects of the hearing mechanism, and the term "hearing conservation" could refer to the complete preservation of all hearing. But the relation between noise exposure and hearing damage is complex and not fully understood, and parameters representing "all hearing capability" are neither well established nor commonly measured. Furthermore, hearing normally deteriorates with age in our society.

For an individual in today's world, the most important practical effect of hearing damage occurs when the ability to hear speech is impaired. Consequently, hearing conservation has focused on this aspect, and present damage claims are evaluated on the basis of loss of the ability to hear speech.

The sound pressure level of everyday speech for people 3 to 6 feet apart is roughly 55 to 65 dB(A), which corresponds to a hearing level (arithmetic average of 500 Hz, 1 kHz, and 2 kHz) of about 45 dB. Particular syllables may vary about this level by  $\pm 15$  dB. For practical purposes, a person begins to have difficulty with speech when he has a hearing loss (arithmetic average of loss at 500 Hz, 1 kHz, and 2 kHz) of greater than 25 dB. His hearing is then considered to be handicapped (ref 2).

Hearing conservation frequently refers only to the preservation of the ability of a stated percentage of the population to hear speech in quiet. But it is possible to lose a considerable amount of hearing capability before one is noticeably handicapped in this regard. Indeed, the conservation of all hearing is impractical, partly because one cannot segregate losses due to nonoccupational causes. In attempting to set criteria, it is important to recognize that the amount of noise exposure that can be tolerated depends on the susceptibility of the individual to hearing damage and the amount of hearing loss deemed acceptable. The differences between various criteria—eg, for 8 hours the OSHA limit of 90 dB(A), the DoD recommendation of 84 dB(A) (ref 3), the ISO R1999 recommendation of 80 dB(A) (ref 4), and the EPA recommendation of 75 dB(A)—are due largely to the amount of hearing loss considered acceptable and the percentage of the population one is willing to affect. OSHA considers some hearing loss outside the speech range to be acceptable and accepts a noise-induced permanent threshold shift (NIPTS) at 500 Hz, 1 kHz, and 2 kHz up to 25 dB, an amount which is not expected to interfere with speech at normal levels.

### HEARING DAMAGE RISK

Table 1 (from ref 2) shows the percentage of people expected to show a hearing handicap as a function of years of exposure at work to various levels of continuous noise. Age is

1. EPA-550/9-78-100, The Ability of Mildly Hearing Impaired Individuals to Discriminate Speech in Noise, Environmental Protection Agency, Washington DC, 1978.
2. Damage-Risk Criteria for Hearing, by A Glorig, in Noise and Vibration Control, LL Beranek ed, McGraw-Hill, New York, 1971.
3. DoD Instruction 6055.3, Hearing Conservation, 8 June 1978.
4. ISO R1999, Assessment of Occupational Noise Exposure for Hearing Conservation Purposes, International Organization for Standardization (ISO), Geneva, 1971.

Table 1. Percentage of people expected to show a hearing handicap as a function of years of exposure at work to various levels of continuous noise. Age is assumed equal to exposure years plus 20 years. In each level, the lower row takes out the effects of aging (presbycusis). (From Glorig (ref 2).)

Age, years		20	25	30	35	40	45	50	55	60	65
Exposure, years ( <i>or</i> age 20)		0	5	10	15	20	25	30	35	40	45
Exposure level in dBA	80 Total Due to noise	0.7	1.0	1.3	2.0	3.1	4.9	7.7	13.5	24.0	40.0
		No increase in risk at this level of exposure									
	85 Total Due to noise	0.7	2.0	3.9	6.0	8.1	11.0	14.2	21.5	32.0	46.5
		0.0	1.0	2.6	4.0	5.0	6.1	6.5	8.0	8.0	6.5
	90 Total Due to noise	0.7	4.0	7.9	12.0	15.0	18.3	23.3	31.0	42.0	54.5
		0.0	3.0	6.6	10.0	11.9	13.4	15.6	17.5	18.0	14.5
	95 Total Due to noise	0.7	6.7	13.6	20.2	24.5	29.0	34.4	41.8	52.0	64.0
		0.0	5.7	12.3	18.2	21.4	24.1	26.7	28.3	28.0	24.0
Exposure level in dBA	100 Total Due to noise	0.7	10.0	22.0	32.0	39.0	43.0	48.5	55.0	64.0	75.0
		0.0	9.0	20.7	30.0	35.9	38.1	40.8	41.5	40.0	35.0
	105 Total Due to noise	0.7	14.2	33.0	46.0	53.0	59.0	65.5	71.0	78.0	84.5
		0.0	13.2	31.7	44.0	49.9	54.1	57.8	57.5	54.0	44.5
	110 Total Due to noise	0.7	20.0	47.5	63.0	71.5	78.0	81.5	85.0	88.0	91.5
		0.0	19.0	46.2	61.0	68.4	73.1	73.8	71.5	64.0	51.5
	115 Total Due to noise	0.7	27.0	62.5	81.0	87.0	91.0	92.0	93.0	94.0	95.0
		0.0	26.0	61.2	79.0	83.9	86.1	84.3	89.5	70.0	55.0

assumed equal to exposure years plus 20 years. In the lower row of figures in each exposure level, the effects of aging (presbycusis) have been taken out by removing the fraction of persons expected to experience the hearing loss because of age alone, independent of noise exposure.

#### NOISE-INDUCED PERMANENT THRESHOLD SHIFT (NIPTS)

The relationship between long-term noise exposure and noise-induced permanent threshold shift (NIPTS) is well established, but simplifying assumptions are made when these parameters are defined and measured. In practice, it is impossible to control noise exposure closely, and considerable variability of noise level with time is to be expected, along with a wide variety of noise spectra. In many cases, however, noise exposure is treated as though it were continuous at some specified level for 8 hours per day, 5 days per week. Its intensity is frequently expressed as dB(A), the A-weighted sound pressure level in dB.

Table 4. Summary of the permanent hearing damage effects expected for continuous noise exposure at various values of the A-weighted average sound level.

Entries are the difference in decibels between pure-tone thresholds of noise-exposed and control populations matched for age.

(From EPA "levels document" (ref 12).)

75 dB for 8 hrs

	<u>av.0.5,1,2 kHz</u>	<u>av.0.5,1,2,4 kHz</u>	<u>4 kHz</u>
Max NIPTS 90th percentile	1 dB	2 dB	6 dB
NIPTS at 10 yrs. 90th percentile	0	1	5
Average NIPTS	0	0	5
Max NIPTS 10th percentile	0	0	0

80 dB for 8 hrs

	<u>av.0.5,1,2 kHz</u>	<u>av.0.5,1,2,4 kHz</u>	<u>4 kHz</u>
Max NIPTS 90th percentile	1 dB	4 dB	11 dB
NIPTS at 10 yrs. 90th percentile	1	3	9
Average NIPTS	0	1	4
Max NIPTS 10th percentile	0	0	2

85 dB for 8 hrs

	<u>av.0.5,1,2 kHz</u>	<u>av.0.5,1,2,4 kHz</u>	<u>4 kHz</u>
Max NIPTS 90th percentile	4 dB	7 dB	19 dB
NIPTS at 10 yrs. 90th percentile	2	6	16
Average NIPTS	1	3	9
Max NIPTS 10th percentile	1	2	5

90 dB for 8 hrs

	<u>av.0.5,1,2 kHz</u>	<u>av.0.5,1,2,4 kHz</u>	<u>4 kHz</u>
Max NIPTS 90th percentile	7 dB	12 dB	28 dB
NIPTS at 10 yrs. 90th percentile	4	9	24
Average NIPTS	3	6	15
Max NIPTS 10th percentile	2	4	11

would be the same individuals as the 4% who would normally develop a hearing loss with no significant exposure to noise.

The Inter-Industry Noise Study (Yerg et al, ref 11) investigated hearing damage caused by industrial noise exposure within the 82-92 dB(A) range. Minimum exposure duration was 3 years; median was 15 years. The noise exposure of the control population did not exceed 75 dB(A). When the hearing levels of the male population were compared with those of the control, differences were significant at 3, 4, and 6 kHz. To compare the results of Yerg et al

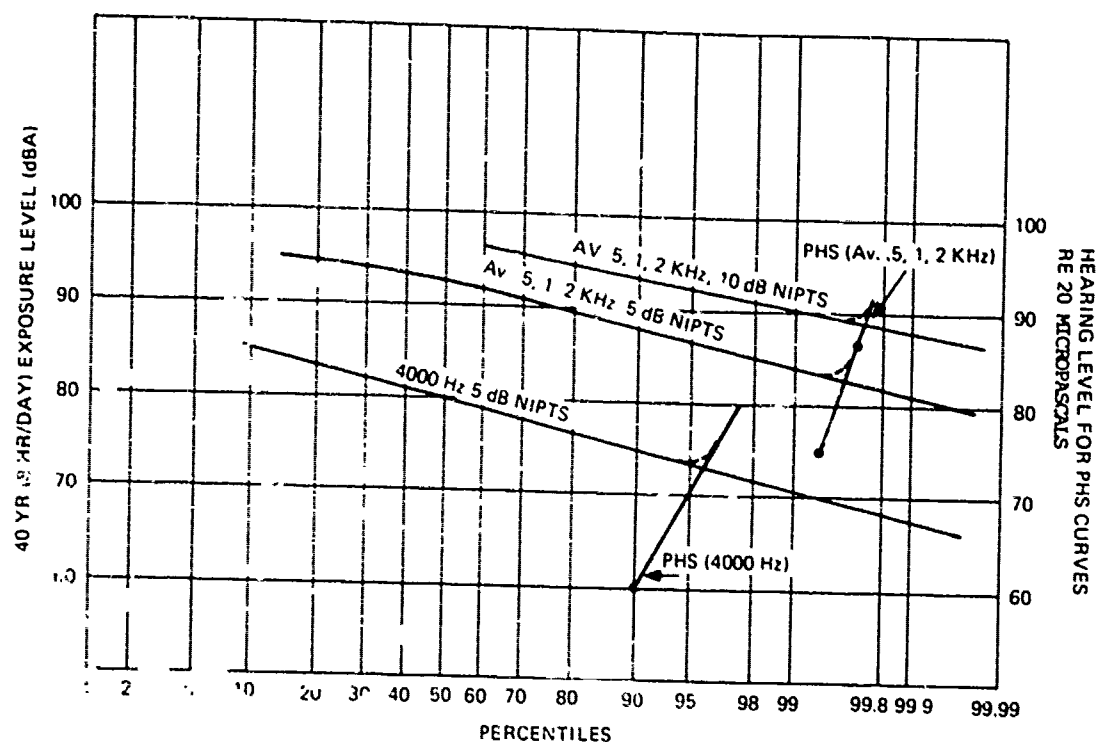


Figure 1. exposure level and hearing level as a function of population percent, showing merging of different NIPTS curves with PHS curves. (From the EPA "levels document" (ref 12).)

with those of the EPA "levels document" for this review, the mean difference in hearing level at 4 kHz between the experimental and control populations was calculated from table 5 in Yerg et al. These hearing levels agree well with the average hearing levels for all ages and percentiles (unreported male/female composition) exposed to 80, 85, and 90 dB(A), as reported in the EPA "levels document" (table 5).

Table 5. Permanent noise-induced threshold shift (NIPTS): Comparison of selected results from the Inter-Industry Noise Study (ref 11) and the EPA "levels document" (ref 12).

	Noise Levels of Exposure, dB(A)	Average NIPTS, dB		
		at 0.5, 1, 2 kHz	at 0.5, 1, 2, 4 kHz	at 4 kHz
Inter-Industry Noise Study (median 15 years exposure)	82-92	Male: 1.9 Female: 1.4	4.5 2.0	12.1 3.8
"levels document." (all ages, all percentiles)	80	0	1	4
	85	1	3	9
	90	3	6	15

Yerg et al concluded that hearing levels of workers at the upper end of the noise intensity range were not different from the hearing levels of those at the lower end. However, one should note that this failure to show a difference in hearing level due to noise intensity may be a result of difficulties in defining precisely the actual noise exposures. The authors themselves stated:

A worker exposed to noise levels ranging from 82 to 88 dB(A) on the first test could well have been exposed to noise levels ranging from 86 to 92 dB(A) on the second visit. Such variations over time demonstrated that it is unrealistic to assume that a measured noise level can be applied retrospectively with precision. . . .

### TIME/INTENSITY TRADE-OFF

The most direct measurements of hearing damage have been obtained empirically by measuring permanent threshold shift in people who have been exposed to continuous noise almost daily for many years. But noise is often not constant, and hearing damage risk varies with the spectrum, level, and duration of the noise.

The concept of noise exposure, as contrasted to noise level, is important because, as table 1 shows, the duration for which one is exposed to noise is as important as the level. Noise exposure may be expressed in terms of a noise exposure rating, or noise dose, which is considered acceptable when its value does not exceed unity. It is given by the formula

$$\text{Noise exposure rating} = \frac{C_1}{T_1} + \frac{C_2}{T_2} + \frac{C_3}{T_3} + \dots + \frac{C_i}{T_i},$$

where

$C_i$  = total duration of exposure at a particular level

$T_i$  = maximum acceptable duration of exposure at that level

It is generally accepted that, within limits, one can compensate for higher levels by reducing exposure time. But the exact intensity/duration trade-off relationship to be used is an area of current debate. Rules of 5-, 4-, and 3-dB per time-halving are presently being used. For example, OSHA specifies a 5-dB rule, the DoD specifies a 4-dB rule, and both the International Standards Organization (ISO) Recommendation R1999 and the EPA "levels document" specify a 3-dB (equal energy) rule.

Two theories have been advanced for determining the relation between intensity and duration: the equal temporary effect theory and the equal energy theory (ref 15). Botsford (ref 16) states:

The equal temporary effect theory postulates that the hazard of noise exposure increases with the average temporary loss of hearing it produces in

15. Temporary Threshold Shift from Octave Band Noise: Applications to Damage Risk Criteria, by A Glorig and DL Sklar; J Acoust Soc Am, vol 31, 1959, p 522-528.
16. Current Trends in Hearing Damage Risk Criteria, by JH Botsford; J Sound and Vib, vol 4 no 4, April 1970, p 16-19.

young normal ears. . . . This theory arises out of the observation that those noise exposures that ultimately produce permanent hearing loss also produce temporary hearing loss in young normal ears, and, conversely, . . . [w]hile a causal relation between temporary hearing loss and permanent hearing loss has not been established, it is reasonable to assume that those noise exposures that do not cause much temporary effect will not cause much permanent effect either. On the basis of this assumption, results of temporary threshold shift (TTS) studies have been used to define safe limits for all-day exposures to steady noise which agree with those established by permanent threshold shift studies. Studies of TTS also lead to reasonable limits for very short or indefinitely long exposures.

The equal energy theory, on the other hand, is based on the hypothesis that hearing damage risk is determined by the total amount of energy reaching the ear each day. It yields a 3-dB per time-halving rule.

The 5-dB rule is supported by results of TTS studies using intermittent noise. Intermittent noise induces less TTS than continuous noise with the same total energy. Therefore, assuming that the temporary effect of a given noise predicts the permanent effect, more total energy is acceptable if the noise is intermittent (ref 17).

The equal temporary effects theory ascribes great value to rest periods, ie periods of "effective quiet" during which recovery from TTS can occur. Ward, Cushing, and Burns (ref 18) determined the highest noise level that will neither produce a significant TTS nor retard recovery from a TTS produced by prior high-level noise exposure. The value is about 76 dB for the 250- and 500-Hz bands, and 68 dB for 1, 2, and 4 kHz. Applying the A-weighting response to each gives respectively 67, 73, 68, 69 and 69 dB(A) for each of the above bands. Thus 70 dB(A) is considered a reasonable though not conservative estimate of the level of effective quiet.

Another school of thought on TTS studies supports the equal energy theory. There appears to be general agreement that rest periods long enough to provide full recovery from TTS play an important role in reducing damage risk.

Johnson, Nixon, and Stephenson (ref 19) investigated TTS induced by 24 hours of intermittent noise exposure. They found that (1) the growth of TTS clearly reached an asymptote for all interrupted exposure conditions, even when the TTS was as small as 5 dB; (2) interrupted exposures produced lower asymptotic levels than continuous exposure with the same amount of energy; and (3) the TTS recovery patterns were essentially the same at 1 hour and beyond for all conditions - ie exposure to equal energies required the same amount of time for recovery. The authors stated that the results lend support to the use of the equal energy rule for estimating effects of acoustic energy on people.

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17. A Comparison of the Effects of Continuous, Intermittent, and Impulse Noise, by WD Ward; in Effects of Noise on Man, D Henderson et al, ed, Raven Press, New York, 1976.
  18. Effective Quiet and Moderate TTS: Implications for Noise Exposure Standards, by EM Cushing and EM Burns; J Acoust Soc Am, vol 59 no 1, 1976, p 160-165.
  19. Long Duration Exposure to Intermittent Noises, by DL Johnson, CW Nixon, and MR Stephenson; Aviation, Space and Environmental Medicine, September 1976, p 987-990.

Nixon, Johnson, and Stephenson (ref 20) showed that TTS from long-duration noise exposure reaches an asymptote after 8 to 16 hours and does not increase further during continued exposure for durations of at least 48 hours. They compared TTS growth and recovery patterns during 24- and 48-hour exposures of humans to continuous pink noise at an A-weighted level of 85 dB. Results indicate similar patterns of acquisition and relatively equal amounts of TTS for the two exposure durations. At 4 kHz, however, recovery of pre-exposure hearing levels following termination of the noise differed: twice as much time was required following 48-hour exposure as following 24-hour exposure. This indicates that the amount of TTS is not a good indicator of hearing damage risk for long-duration exposure. But it also shows that TTS recovery time is predicted by the equal energy rule, since recovery from the 48-hour exposure (which had twice the energy of the 24-hour exposure) took twice as long.

Martin (ref 21) reviewed studies relating the equal energy rule to NIPTS and TTS and concluded that the equal energy rule does accurately predict NIPTS for intermittent and impulse noise at least to sound pressure level peaks of 150 dB. These findings also suggest that the equal energy rule may in fact not be too conservative for predicting damage risk.

#### **VALIDITY OF A-WEIGHTED SOUND PRESSURE LEVEL AS A MEASURE OF HEARING DAMAGE RISK**

A-weighted sound pressure level has been shown to correlate well with hearing damage risk for noise spectra typical of most industrial settings.

When broad interest in noise induced hearing loss first began, noise level measurements were usually made in octave bands. With more research, it became apparent that the average of the sound-pressure levels in the higher bands only correlated well with noise induced hearing loss. The conclusion was reached that a single number taken with a meter that discriminates against energy content at low frequencies is adequate. Such a number is the A-weighted sound pressure level in dB (sound level A; dB(A)).

Robinson concludes that the magnitude of error in hearing-loss calculations is on the order of  $\pm 2$  dB when sound level A is used to specify the energy even when rising and falling spectra are compared. Similar findings have been reported by Baughn in a study where over 600 spectra have been analyzed. Consequently, sound level A has been accepted for measurements concerning conservation criteria. (Glorig, ref 2.)

It seems advisable to use simple A-weighted sound level, with no further corrections, to measure shipboard noise. It greatly simplifies the data taking and data analysis process, thereby reducing costs. In doing so, however, it should be recognized that shipboard noise spectra generally have much greater low-frequency content than industrial spectra, and that

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20. Asymptotic Behavior of Temporary Threshold Shift and Recovery from 24- and 48-hour Noise Exposures, by CW Nixon, DL Johnson, and MR Stephenson: *Aviation, Space, and Environmental Medicine*, April 1977, p 311-315.
  21. The Equal Energy Concept Applied to Impulse Noise, by AM Martin; in *Effects of Noise on Hearing*, D Henderson et al, ed, Raven Press, New York, 1976.

as a result one occasionally may be applying A-weighting outside the domain it can handle well. The following discussion may aid in identifying such situations.

One school of thought holds that the effects of low-frequency noise on people, though still not well defined, may not be faithfully represented by A-level, especially when low frequencies are present at high levels. Botsford (ref 16) proposed that the C-weighted sound level be used to adjust the A-level in such cases. He concluded that the quantity C-A is a good descriptor of the low-frequency content of noise, and that if C-A exceeds 5 dB, the A-level to be used should be lower than the measured A-level by the amount given in tables 6 and 7.

Table 6. Acceptable exposures to dangerous noise. This table may be used to find the total acceptable exposure duration for repeated noises of known A-level. To use the table, select the column headed by the number of times the dangerous noise occurs per day, read down to the average A-weighted sound level of the noise, and locate directly to the left in the first column the total duration of dangerous noise for any 24-hour period. It is permissible to interpolate if necessary. (From Botsford (ref 16).)

Total Noise Duration Per Day (24 hours)	Number of Times Noise Occurs Per Day						
	1	3	7	15	35	75	160 up
8 hrs.	89	89	89	89	89	89	89
6	90	92	95	97	96	94	93
4	91	94	98	101	103	101	99
2	93	98	102	105	108	113	117
1	96	102	106	109	114	125	125 (1½h)
30 min.	100	105	109	114	125		
15	104	109	115	124			
8	108	114	125				
4	113	125					
2	123						

A-weighted  
Sound Levels, dBA

Table 7. Deductions for noise types. The A-weighted sound pressure level may be corrected for low-frequency content by using the difference between the C- and A-levels, and this table. To use the table, find the A-weighted sound level of the noise in the first column, and read horizontally, in the column headed by the difference between C- and A-weighted sound levels of the noise, the number of decibels to be subtracted from the A-weighted sound level before determining the exposure limit from table 6. It is permissible to interpolate if necessary. (From Botsford (ref 16).)

A-weighted Sound Level, dBA	C-A					
	0	5	10	15	20	25
90	0	0	1	1	1	1
95	0	1	2	3	3	3
100	0	2	4	4	5	6
105	0	3	5	6	7	8
110	0	4	6	8	9	10
115	0	5	8	9	9	
120	0	6	9	12		
125	0	6	10			
130	0	7				

Deductions

A recent experiment, although dealing with noise with a spectrum unlike either ship-board or industrial spectra, has produced an effect in the opposite direction from Botsford's findings. This may rekindle discussion of the effect of low frequencies on hearing damage risk. Because animals were used, caution is in order in applying the results to humans. Burdick, Patterson, Mozo, and Camp (ref 22) exposed two groups of chinchillas to octave bands of noise of equal A-levels but unequal C-levels and noise rating (NR). One group heard low-frequency noise (63 Hz); the other, high-frequency noise (1 kHz). The permanent hearing loss which resulted is shown in table 8. Two main points should be noted. First, low frequencies produced 7 dB more hearing damage than did high frequencies - 16 dB vs 9 dB NIPTS at 2 kHz. Second, A-level did not faithfully predict hearing damage.

Note that when annoyance rather than hearing damage is the issue, some feel A-weighting gives too little influence to low frequencies. Buiten (ref 23) reports that, in his experience, the bridges of ships represent a worst case in this regard. He reports an engine exhaust case on a bridge wing in which noise treatment substantially reduced the annoyance subjectively, the NR dropped from 86 to 76, but the A-level increased from 79 to 80 dB. Above about 50 dB NR, NR is more sensitive to low frequencies than A because the NR curves are flatter.

22. Threshold Shift in Chinchillas Exposed to Octave Bands of Noise Centered at 63 and 1000 Hz for Three Days, by CK Burdick, JH Patterson, BT Mozo, and RT Camp; J Acoust Soc Am, vol 64 no 2, 1978, p 458-464.
23. Report 125S, A Proposal on Noise Criteria for Seagoing Ships, by J Buiten; Netherland Ship Research Center TNO, 1969.

Table 8. The results of exposure to octave bands of low- or high-frequency noise of equal A-level. (Derived from Burdick et al, ref 22.)

Group Number	Octave Band (Center Freq)	Levels of Three Consecutive 72-hour Exposures				Hearing Loss (NIPTS), dB	
						at 1.4 kHz	at 2 kHz
1	Low (63 Hz)	100	110	120	dB SPL	11	16
		74	84	94	dB(A)		
		82	95	107	NR		
2	High (1 kHz)	75	85	95	dB SPL	6	9
		75	85	95	dB(A)		
		75	85	95	NR		

## VALIDITY OF THRESHOLD SHIFT AS A MEASURE OF HEARING DAMAGE

Threshold shift (NIPTS and TTS) may not be an accurate measure of noise-induced hearing damage. The human ear contains about 9000 outer and 3000 inner hair cells (ref 24). The outer hair cells are the elements of the hearing mechanism most susceptible to noise-induced damage. Spoendlin (ref 25) stated that damage of the metabolic type induced by noise in the zone below 130 dB affects the outer hair cells rather than the inner hair cells, which frequently remain intact. Damage to the outer hair cells does not necessarily manifest itself as a shift in threshold. According to Spoendlin, the threshold of hearing in experimental animals does not seem to be appreciably affected even by the selective complete loss of outer hair cells. Henderson and Hamernik (ref 26, p vii) state: "Preliminary analysis of cellular integrity has demonstrated that large losses of outer hair cells may not be manifested in the results of pure-tone audiometry; therefore, it appears that more powerful diagnostic procedures are necessary."

What is the role of the outer hair cells? Bienvenue, Michael, and Violon-Singer (ref 27) hypothesize that they inhibit the output of the inner hair cells in a way which sharpens the critical bands. In other words, they sharpen the tuning characteristics of the ear. Bienvenue et al predict that damage to the outer hair cells will increase the sensitivity of the ear to loudness changes, it will cause recruitment, and will increase the width of the critical bands. They demonstrated that the effects of noise on the ability to discriminate changes in intensity last much longer than does temporary threshold shift. An increase in critical bandwidth could impair speech discrimination in noise. The EPA, in ref 1, shows that such impairments exist in people with normal hearing according to threshold measurements (25-dB fence).

## SELECTED ANNOTATED BIBLIOGRAPHY: EFFECTS OF NOISE ON HEARING

RH Martin, MD Gibson, and BS Lockington, Occupational hearing loss between 85 and 90 dBA, *J Occ Med*, vol 17 no 1, 1975, p 13-18

This study (ref 28) investigated the difference in hearing risk between 85 and 90 dB(A). Three areas of a steel mill were investigated. The results are given in table 9. The results of the 50- to 65-year age group were most striking, and they correlate better with linear sound pressure level than with A-level. They also indicate that 90 dB(A) is more hazardous than 85 dB(A).

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24. Noise and Man, by W Burns; JB Lippincott, Philadelphia, 1973, p 65.

25. Anatomical Changes Following Various Noise Exposures, by H Spoendlin, in *Effects of Noise on Hearing*, D Henderson et al, ed, Raven Press, New York, 1976.

26. *Effects of Noise on Hearing*, D Henderson, RP Hamernik, DS Dosanjh, and JH Mills, ed, Raven Press, New York.

27. The Effect of High Level Sound Exposure on the Loudness Difference Limen, by GR Bienvenue, PL Michael, and JR Violon-Singer; *Am Indust Hygiene J*, vol 37, November 1976, p 628-635.

28. Occupational Hearing Loss Between 85 and 90 dBA, by RH Martin, MD Gibson, and BS Lockington; *J Occupat Med*, vol 17 no 1, 1975, p 13-18.

Table 9. Hearing loss in a steel mill.

Work Areas	Sound Pressure Level		Percent Impaired (normalized)			
	dB(A)	dB linear	Age 18-29	Age 30-39	Age 40-49	Age 50-65
Cold mills	86	90	1.7	4.0*	5.0*	14.0
Slinger floor	86	95	4.6*	1.8	2.5	26.5
Electric furnaces	89	102	4.0	2.3	15.5	32.5*

\*Average hearing level at 0.5 and 2 kHz was significantly different from that of the control population.

All persons working in the above areas at the time of initiation of the study were considered eligible except those in any of the following three categories:

1. Those who had worked in some other noise for more than 3 years.
2. Those who had severe unilateral loss (greater than 40 dB difference) in ears at two or more frequencies.
3. Those who had previously diagnosed bilateral nonsensorineural loss.

All other variables such as nonoccupational noise effects were assumed to randomize out.

The value of the results of this study is limited, for several reasons. More impact noise was associated with electric furnaces than with other locations, but this was not quantified. The duration of exposure was apparently not quantified. The sample size was so small that statistically significant differences between the sample and control populations occurred for only 33% of the exposure groups tested. Hearing loss at 4 kHz, the frequency most sensitive to noise damage, was not considered.

**D Henderson, RP Hamernik, DS Dosanjh, and JH Mills, Effects of Noise on Hearing, Raven Press, New York, 1976**

This book (ref 26), compiled by distinguished experts in their respective fields, is an excellent summary of the effects of noise on people. It is the result of a symposium on the effects of noise on hearing, sponsored by the National Institute for Occupational Safety and Health (NIOSH) in 1975. Forty experts from the fields of acoustics, anatomy, physiology, audiology, epidemiology, otolaryngology, and biochemistry were invited to write critical essays on the specific issues underlying the effects of noise on hearing. The essays discuss the following issues:

- **Noise and Hearing: A Perspective on the Problem**

A general overview of the problems involved in determining the relationship between noise and hearing loss.

- **Cochlear Anatomy and Biochemistry**

Mechanisms of noise damage to the inner ear. It discusses the anatomical and biochemical changes caused by noise damage.

- **Mechanical and Electrophysiological Characteristics of the Ear**

Outer ear and middle ear mechanisms that protect against noise, and the effects of noise on the cochlear potentials, eighth nerve responses, and temporary threshold shift.

- **Experimental Studies of Noise-Induced Hearing Loss**

Experimental studies dealing with temporary threshold shift in humans and permanent and temporary threshold shift in animals.

- **Epidemiological and Analytical Studies of Noise-Induced Hearing Loss**

Models for noise-induced hearing loss, characteristics of noise-induced hearing loss, and the relations between continuous, intermittent, and impulse noise.

- **Scientific, Medical, and Legal Considerations for Establishing Damage Risk Criteria.**

The following three essays from ref 26 are particularly pertinent to this survey and are separately annotated.

### **BA Bohne, Mechanisms of Noise Damage in the Inner Ear**

In this essay, the four hypothesized mechanisms of noise damage to the inner ear are as follows:

- **Mechanical**

The cells in the inner ear are directly damaged by mechanical action.

- **Metabolic exhaustion**

Key enzymes and/or metabolites in the cells are damaged by prolonged noise exposure.

- **Vascular changes**

Blood circulation in the cochlear vessels is impaired by prolonged noise exposure, causing a lack of oxygen and nutrients for cells in the inner ear.

- **Ionic changes**

The continuity of the reticular lamina may be interrupted, allowing endolymph containing a high concentration of potassium ions to enter the fluid space of the organ of corti. This damages the cell membranes, which are not normally in contact with such ionic concentrations.

### **HE Von Gierke and DL Johnson, Summary of Present Damage Risk Criteria**

The authors state in this essay that in spite of uncertainties and open scientific questions, the available data base is consistent enough to predict for preventive and protective purposes the amount of noise-induced permanent threshold shift (NIPTS) to be expected in a population as a result of habitual noise exposure. They discuss the compilation of the EPA "levels document" and the levels of NIPTS to be expected from different noise exposure levels and for various percentiles of the population.

The exposure time-intensity trade-off is then discussed. The authors state that TTS data accumulated in laboratory experiments clearly indicate that no simply TTS-vs-time relationship fits the data perfectly. However, the TTS-vs-exposure time data for 4 kHz are fitted better by the equal energy rule than by the 5-dB increase of level per halving of time relationship. However, the data for frequencies in the conventional speech range might be better fitted by the 5-dB rule. After describing arguments for each rule, the authors conclude that the 3-dB (equal energy) rule is the best.

The authors state that the establishment of noise exposure limits short of levels completely safe for 100% of the population is a social, economical, legal, and, in short, administrative decision. They point out that the damage risk of noise is constant: it does not change with legal interpretation, the economic situation, social changes, and so on. However, the criteria for noise exposure set forth by various institutions may vary on the basis of how much hearing damage is acceptable in view of the above considerations.

#### AM Martin, The Equal Energy Concept Applied to Impulse Noise

Martin reviews, in the light of the equal energy concept, studies that examine the effects of impulse noise on noise-induced permanent threshold shift (NIPTS) and noise-induced temporary threshold shift (NITTS). He concluded that the equal energy concept relates well to NIPTS, but that it does not adequately predict NITTS.

Martin summarizes the equal energy concept in three points: (1) Equal amounts of A-weighted sound energy damage hearing equally. (2) The damage is a function (not necessarily linear) of the acoustical energy received. (3) A trading relationship exists between exposure time and noise level, the product of the two being a measure of the total acoustical energy received.

In support of the equal energy concept as applied to steady-state noise, Martin cites the study by Burns and Robinson (ref 29) as the most convincing evidence. They found empirically that hearing loss is related to  $L_A + 10 \log T/T_0$ , an expression of total sound energy, where  $L_A$  is the steady-state A-weighted sound pressure level over exposure time  $T$ , and  $T_0$  is a reference duration.

For dealing with fluctuating levels, one may define the quantity  $E_A$  as follows:

$$E_A = L_{eq} + 10 \log T/T_0,$$

in which

$$L_{eq} = 10 \log \frac{1}{T_R} \int_0^{t_s} \frac{[P_A(t)]^2}{P_0^2} dt,$$

29. Hearing and Noise in Industry, by W Burns and DW Robinson, Her Majesty's Stationery Office, London England, 1970.

where  $T_R$  is the duration of the nominal working day,  $t_s$  is the total daily exposure time,  $P_A$  is the instantaneous A-weighted sound pressure in pascals ( $N/m^2$ ), and  $P_0$  is the reference rms sound pressure of  $20 \mu Pa$ .

Martin derives and plots  $E_A$  for impulse noises of 85 dB(A) and 95 dB(A) and compares them with various damage risk criteria for impulse noise. He then examines experimental evidence and concludes, "... the equal energy concept should be extended from steady-state noise exposure to include industrial impulse noise, at least up to peak sound levels of 150 dB(P). Circumstantial evidence exists to show that it may also be applied to higher peak sound levels, gunfire, and explosive noises."

That equal energy accurately predicts NIPTS caused by impact noise is shown by studies by AM Martin (ref 30), Guberan et al (ref 31), Ceypek et al (ref 32), and others.

**EPA-550/9-76-007, Some Considerations in Choosing an Occupational Noise Exposure Regulation, February 1976**

This document (ref 33) reviews the effects of noise on people and discusses the considerations to be made when choosing an occupational noise exposure regulation.

It discusses how much hearing damage is to be expected from various noise exposures and how much a population will benefit from various noise exposure criteria. The differences in hearing damage expected from noise exposures of 85 and 90 dB are shown in table 10 and are based on data from Baughn (ref 10) and the National Institute for Occupational Safety and Health (NIOSH) (ref 34). It appears that for any given hearing threshold fence, about twice as many people exceed that fence when exposed to 90 dB(A) as when exposed to 85 dB(A).

The authors discuss the data and calculations used to estimate the damage risk of various noise criteria. They present tables that give the number of workers impaired due to noise 10 and 40 years after compliance with given criteria. They state that no matter what definition of material impairment is used, none of the presently proposed standards would assure that no employee suffers impairment.

Information is presented on other health effects of noise, including cardiovascular effects (reviewed elsewhere in this report).

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30. Industrial Impact Noise and Hearing, by AM Martin; Ph D thesis, Department of Pure and Applied Physics, University of Salford, England, 1970.
  31. Hazardous Exposure to Industrial Impact Noise: Persistent Effect on Hearing, by E Guberan, J Fernandez, J Gardiner, and G Terrier; Ann Occup Hyg, vol 14, 1971, p 345-350.
  32. US EPA Report, Hearing Loss Due to Impulse Noise: A Field Study, by T Ceypek, JJ Kuzniarz, and A Lipowczan; Proc of the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
  33. EPA-550/9-76-007, Some Considerations in Choosing an Occupational Noise Exposure Regulation, Environmental Protection Agency, Washington DC, February 1976.
  34. NIOSH Report TR008-72, Criteria for a Recommended Standard; Occupational Exposure to Noise, National Institute for Occupational Safety and Health; Superintendent of Documents, US Government Printing Office, Washington DC, 1972.

Table 10. Percent of people exceeding various hearing threshold fences, according to data from Baughn (ref 10) and NIOSH (ref 34). Note that exposure to 90 dB(A) causes about twice as many people to be hearing impaired as does exposure to 85 dB(A), regardless of the fence selected.  
(From EPA 550/9-76-007 (ref 33).)

	Baughn Data		NIOSH Data	
Age:	46-54*		46-54*	
Exposure Level:	85	90	85	90
Fence:				
15 dB (.5, 1, 2 kHz)				
Total	83	89		
Presbycusis	75	75		
Net	8	14		
20 dB (.5, 1, 2 kHz)				
Total	50	61		
Presbycusis	39	39		
Net	11	22		
25 dB (.5, 1, 2 kHz)				
Total	26	36	19	31
Presbycusis	17	17	10	10
Net	9	19	9	21
25 dB (1, 2, 3 kHz)				
Total			30	43
Presbycusis			18	18
Net			12	25
50 dB (.5, 1, 2 kHz)				
Total	1.5	2		
Presbycusis	1	1		
Net	0.5	1		

\*Between 31 and 32 years average exposure.

The authors discuss the immunological effects of noise and conclude that available information is far from adequate to assess their magnitude. They also discuss the effect of noise on fetal abnormalities and conclude that more research is necessary in this area.

The remainder of the report covers benefits and costs of protective standards, which include hearing conservation, savings in workers' compensation costs, social costs of absenteeism, annoyance as a social cost, innovation and regulation, and quantification of net costs.

It also covers regulatory alternatives, including industry specific standards, compliance scenarios, new plant standards, administrative controls, personal protective equipment, variances, and abatement agreements.

## 2 OTHER EFFECTS OF NOISE

### INTRODUCTION

The most significant effect of noise on people is that it causes hearing damage, as reviewed in section 1. This section reviews available literature on other effects of noise on people. Noise causes annoyance, stress, and other health-related phenomena. It interferes with the hearing of speech, warning signals, and other desirable sounds. It interferes with work performance and safety, relaxation, and sleep.

The "nonauditory" effects of noise (those effects on body functions other than those involving hearing directly) are generally considered to be of less import than hearing damage. People can successfully adapt to noise in many instances. However, many of these effects have not yet been adequately explored, and some may be important to health under appropriate circumstances. It has been suggested, for example, that the cardiovascular system may never adapt to noise.

### HEALTH (EXCLUDING HEARING DAMAGE)

According to EPA 550/9-76-007 (ref 33), the scientific evidence for nonauditory effects of noise is far from conclusive for daily exposures of 8 hours to 85 to 90 dBA, "... although there is a substantial body of data that suggests a wide variety of noise-induced health effects of potentially great significance in social terms."

Other than hearing loss, noise is not suspected of producing any single health problem unique to itself. The major concern over nonauditory health effects from noise arises from the ability of noise under some circumstances to act as a general, nonspecific biological stressor. Biological stress is the nonspecific response of the body which prepares it for physical activity, eg, fight or flight. Sudden, unexpected, or annoying noise historically has often been a signal of danger or other condition requiring the body to prepare itself for activity. Because of man's genetically determined characteristics, such noises automatically produce stress. In today's industrial societies, noise is often present, and it still produces stress even though the "fight or flight" reaction is usually not necessary.

Kryter (ref 35) discusses the effects of noise on several systems of the body which are controlled by the autonomic nervous system. These include the cardiovascular, vegetative, glandular, and certain muscular systems. He also discusses the effects of noise on mental and motor behavior, and concludes:

In spite of the very large gaps in our knowledge and the existence of some apparently conflicting research results, the following conclusions are put forth, with, of course, the usual admonition that more research is needed before they can be accepted with great confidence.

1. There is no likely damage risk to a person from the possible unconditioned stress responses to noise that are mediated by the autonomic system.

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35. Extra-Auditory Effects of Noise, by KD Kryter; in *Effects of Noise on Hearing*, D Henderson et al, ed, Raven Press, New York, 1976.

2. Noise may often be concomitant with danger and adverse social-environmental factors that are more important than the noise itself as a cause of apparent greater incidences of various physical and psychological disease and accidents in industry.

3. Autonomic system stress responses could conceivably be a contributing factor to ill health in some persons as the result of noise in their living environment directly interfering with auditory communications and sleep, and, thereby, creating the feelings of annoyance and anger that serve as the direct cause of the stress responses.

4. It would appear that controlling meaningless noise to levels that permit auditory communication and sleep behavior adequate for a given work or living environment would obviate the occurrence of any extraauditory responses in the body of a stressful nature.

## CARDIOVASCULAR SYSTEM

Noise-induced stress can affect the cardiovascular system. Figure 2 shows possible pathways of cardiovascular damage from noise. No single study documents this entire series of events. Synopses of the papers felt by the authors of EPA 550/9-76-007 to be the most important to consider when making judgments on the significance of figure 2 are given in table 11. The authors state, "In summary, one might say that although a great deal more scientific work will be needed before it can be said that workplace noise definitely contributes to cardiovascular disease, a relationship between the two is entirely plausible."

The document also concludes that no adequate assessment is yet available of the effects of noise on the immunological system or on fetal abnormalities.

## SLEEP

It is well known that when sleep, especially rapid eye movement (REM) sleep, is disturbed by noise, work efficiency and health may suffer (EPA (ref 36)), although the effects of loss of REM sleep are not yet completely known. Kryter (ref 35) describes interference with sleep and speech communication as major factors contributing to the non-auditory health effects of noise. The effects of noise on sleep are discussed in Williams (ref 37). This review covers the subject well, and is recommended reading. Translations of a very comprehensive review in German (de Camp (ref 38)) and a review in French (Muzet and Naitoh (ref 39)) were not available in time to be reviewed in this document.

36. EPA booklet, Noise: A Health Problem, Environmental Protection Agency, Washington DC, August 1978.
37. Effects of Noise on Sleep: A Review, by JL Williams; in Proc of the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
38. Schlafbeeinflussung durch Geräusche: Eine Literaturübersicht (The Effects of Noise on Sleep: A Literature Review), by U de Camp; Applied Acoustics, vol 10 no 4, 1977 (German), p 263.
39. AD-A 052343/IWP, Sommeil et Bruit (Sleep and Noise), by A Muzet and P Naitoh, Naval Health Research Center, San Diego, 1975, 23 p.

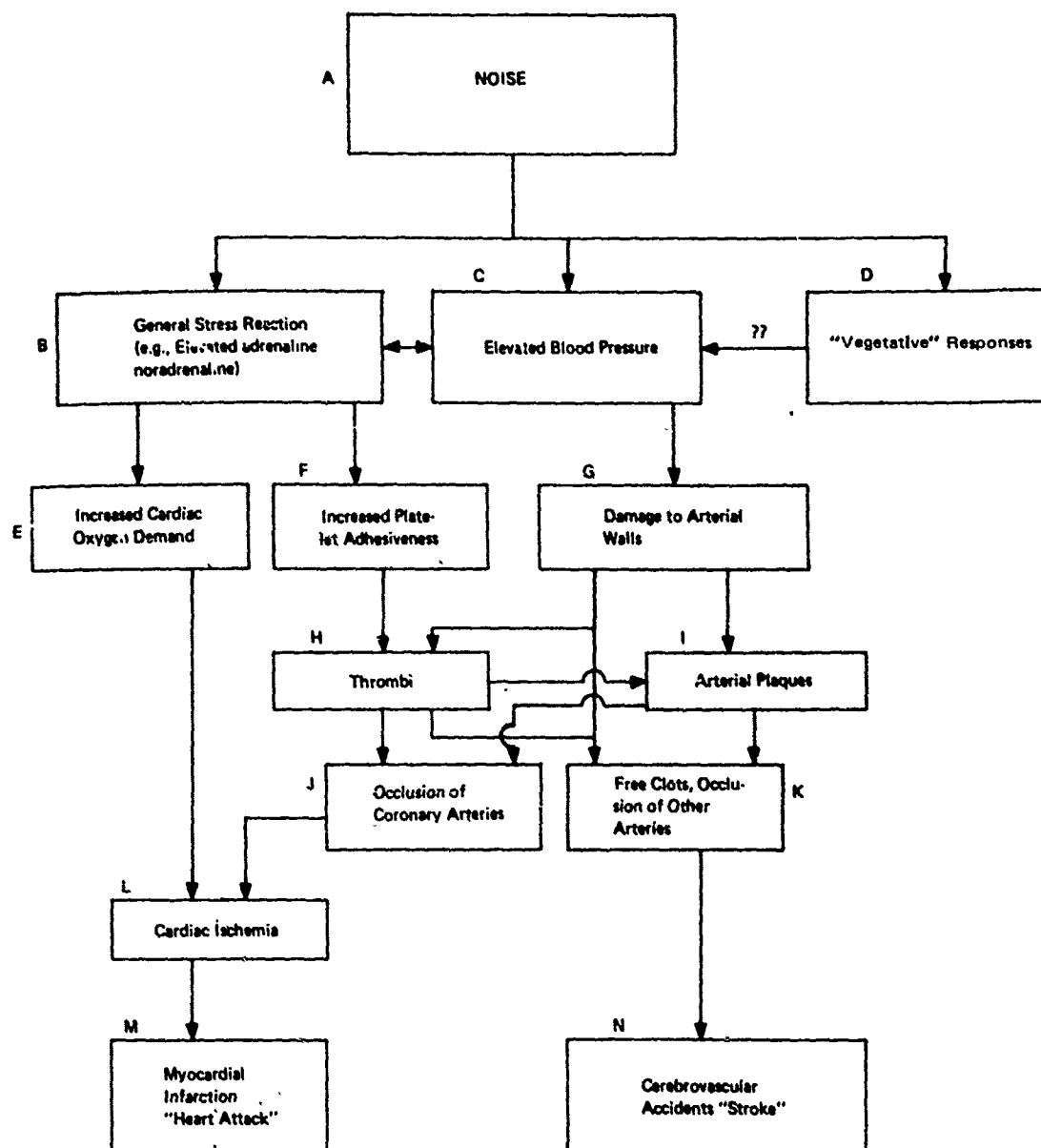


Figure 2. Possible pathways of cardiovascular damage from noise. (From EPA 550/9-76-007 (ref 33)).

Table 11. Documentation suggesting steps in damage pathways indicated in figure 2. EPA references are included at the end of the figure. Secondary references have been deleted.  
(Adapted from EPA 550/9-76-007 (ref 33))

Steps in Figure		Reference number	Author, year, title	Summary conclusions*
From	To			
A	B, C	a	Ortiz, et al. (1974). Modifications of epinephrine, norepinephrine, blood lipid fractions and the cardiovascular system produced by noise in an industrial medium.	Marked elevations of catecholamine excretion and more modest, but statistically significant increases in systolic and diastolic blood pressure in the majority of a group of aircraft turbine testers after 3 hours of normal exposure in their work to noise which "varies between 105 and 115 dB (sic)" in intensity. <i>CAVEAT</i> Other potential noxious agents in the workplace not discussed or controlled.
A	B	b	Arguelles, A. E., et al. (1970). Endocrine and metabolic effects of noise in normal, hypertensive and psychotic subjects.	Increased excretion of epinephrine and norepinephrine in urine after 30 minute exposure to 90 dB (2000 Hz).
(Negative) A	B	c	Carlestam, G., et al. (1973). Stress and disease in response to exposure to noise - a review.	No significant increase in catecholamine levels in 22 young female IBM operators exposed to their normal working noise at 76, 82, 88, and 94 dB for one day each. <i>CAVEAT</i> Authors cite "generally positive attitudes of these subjects to the job per se and to the experiment" and conclude that "noise may be a potential stressor under some circumstances and in some individuals, but need not generally be so."
A	B, C	d	Carlson, L. A., et al. (1972). Stressor-induced changes in plasma lipids and urinary excretion of catecholamines, and their modification by nicotinic acid.	"Monotonous but attention-demanding psycho-motor performance (sorting small ball-bearings) under unfavorable environmental conditions (noise, flickering light), shortage of time and criticism evoked moderate distress, accompanied and/or followed by increases in heart rate, blood pressure, urinary excretion of adrenaline and noradrenaline, and levels of free fatty acids and triglycerides in arterial plasma." <i>CAVEAT</i> Contribution of noise to the observed effects is obviously confounded with the contributions of several other stressors.
A	B	e	Slob, A., et al. (1973). The effects of acute noise exposure on the excretion of corticosteroids, adrenalin and noradrenalin in man.	"Noise exposure" appeared to cause a significantly different adrenaline excretion, insofar that among those exposed to noise no drop in excretion occurred in the afternoon. A similar effect, be it to a somewhat lesser degree, was noticed with regard to noradrenaline excretion. These results appear to be in good agreement with (positive) findings reported in the literature, provided that . . . the influence of two simultaneously occurring stressors is taken into account (1) exposure to noise, and (2) the fact that the subjects were confronted with an unfamiliar laboratory situation. <i>CAVEAT</i> Small, brief study. Noradrenaline effect not statistically significant

Table 11. Continued

Steps in Figure	Reference number	Author, year, title	Summary conclusions*
From	To		
A	C	Medoff, H. S., and Bongiovanni, A. M. (1945). Blood pressure in rats subjected to audiogenic stimulation.	Classic paper. Unquantified noise from air blast for five to ten minutes every weekday from weaning (21 days of age) to 400-900 days (elderly) produced a high incidence of chronic hypertension. <i>CAVEAT</i> : Induced hypertension accompanied by audiogenic seizures (convulsions) which are extremely rare in humans.
A	C	Smookler, H. H., et al. (1973). Hypertensive effects of prolonged auditory, visual, and motion stimulation.	"Hypertension was induced in rats by chronic intermittent exposure to environmental stress (audio, visual, and motion) . . . When audiogenic stress*** was used as the exclusive stressor, the same time course of hypertension was obtained as with the combined stressors but the magnitude of the hypertensive response was less."
(Negative) A	C	Etholm, B., and Egenberg, K. E. (1964). The influence of noise on circulatory functions.	No observed effect of noise on blood pressure up to intensities of 100 JB
		Klein, K., and Grubel, M. (1970). Hemodynamic reactions to acoustic stimuli.	
A	C, D	Lehmann, G., and Tamm, J. (1956). Über Veränderungen der Kreislaufdynamik des ruhenden Menschen unter Einwirkung von Geräuschen.	At intensities of "90 phon" the overwhelming majority of subjects experienced increases in arterial pressures and decreases in pulse volume. Exposures approximately one hour, resting subjects. <i>CAVEAT</i> : Possibly a transient effect.
A	C	Andriukin, A. A. (1961). Influence of sound stimuli on the development of hypertension. Clinical and experimental results.	"Among workers exposed for prolonged periods to intense noise (above 93 dB) (sic) . . . (hypertension) is encountered on an average twice as frequently as in workers of various (relatively quiet) factories . . ." <i>CAVEAT</i> : Controls for the influence of stressors other than noise not apparent. Additionally, data is presented only in percentage/bar graph form without statistical analysis. Our reconstruction of the original incidence data indicates few if any statistically significant differences between the groups.
A	D	Jansen, C. Effects of Noise on Physiological State.	Constriction of peripheral blood vessels and other "vegetative responses" occur transiently in response to continuous noise over 70 dB. <i>CAVEAT</i> : Precise pathological significance of these changes is unclear, although they would tend to increase systemic blood pressure.

Table 11. Continued

Steps in Figure	Reference number		Author, year, title	Summary conclusions*
	From	To		
A	D	m	Jansen, G. (1961). Zur Larmbelastung von Huttenarbeitern.	Epidemiological evidence suggesting "peripheral circulatory symptoms" somewhat more frequent in steel workers exposed to more than 90 dB, as compared to steelworkers exposed to less than this level. <i>CAVEAT</i> Environmental differences other than noise between the steelworker groups not controlled. Vagueness of the "peripheral circulatory symptoms" category invites replication using more objectively measurable parameters.
A	F	n	Maas, B., et al. (1973). Platelet adhesiveness during exposure to noise.	Experiment in rats demonstrating a large increase in platelet adhesiveness in response to "a standardized noise of 113 dB (sic)" for three days. Also a less directly relevant finding of increased platelet adhesiveness (compared to normal controls) in clinic patients with several types of hearing loss not obviously related to noise.
B	H	o	Haft, J. I., and Fani, K. (1973). Intravascular platelet aggregation in the heart induced by stress.	Stress (immersion of rats in ice-cold water for 25-45 min) induced platelet aggregates in myocardial small vessels. Similar aggregates not found in controls.
B	H	p	Haft, J. I., and Fani, K. (1973). Stress and the induction of intravascular platelet aggregation in the heart.	"Intravascular aggregation of platelets similar to that found in dogs after norepinephrine infusion was demonstrated using the electron microscope in the hearts of 20 of 23 rats subjected to two forms of stress (immersion in hot water, 7 of 8 rats; repeated small electric shocks to the feet, 13 of 15 rats). Only one of 14 unstressed rats was found to have similar intravascular platelet aggregates. These findings suggest that catecholamines secreted endogenously during stress are sufficient to cause platelets to aggregate intravascularly and raise the possibility that clinical myocardial infarction occurring during severe or prolonged stress may be caused by catecholamine-induced platelet thrombi which occur at, or travel to, and occlude a coronary artery already narrowed by previous atherosclerosis."
B	L	q, r	Haft, J. I., and Fani, K. (1973). Protection against epinephrine-induced myocardial necrosis by drugs that inhibit platelet aggregation.	(1) Confirmation of previous literature reporting myocardial necrosis after catecholamine infusion. (2) Prevention of this necrosis with three different inhibitors of platelet aggregation (aspirin, dipyridamole, and clofibrate).

Table 11. Continued

Steps in Figure		Reference number	Author, year, title	Summary conclusions*
From	To			
B	L	s	Nestel, P. J., et al. (1967). Catecholamine secretion and sympathetic nervous responses to emotion in men with and without angina pectoris.	<p>Patients who had suffered heart attacks 6-18 months previously were divided into those with and without various kinds of chest pain, including <i>angina pectoris</i>. Those with <i>angina</i> and those with other left chest pain were shown to secrete appreciably more norepinephrine (as measured by metabolites in the urine) than patients without such chest pain in response to a series of mild stressors (solving a series of puzzles, completing a questionnaire measuring anxiety, and undergoing a test of pain threshold from radiant heat to the forehead). Previous literature is also cited to the effect that "subjects with coronary heart disease as a group show a greater adrenergic response than do healthy subjects to procedures which stimulate the sympathetic nervous system ... "Myocardial ischemia, which may become manifest clinically as angina pectoris, occurs whenever the energy requirements of the heart exceed the available supply of oxygen. Emotion commonly induces angina pectoris in people with coronary heart disease, and several lines of evidence suggest that this effect of emotion may be brought about by an increased secretion of the catecholamines epinephrine and norepinephrine. The concentrations of urinary catecholamines rise during stress ... and a quantitative relationship has been demonstrated between the stress experienced by an individual and his urinary excretion of catecholamines ... The infusion of norepinephrine into normal subjects leads to an increase in the extraction of oxygen by the heart and a fall in the oxygen saturation of coronary venous blood ... changes which characteristically occur during exercise in patients with coronary insufficiency ... It is well known that the infusion of norepinephrine into subjects with coronary heart disease may induce angina pectoris."</p>
B	M	t	Pearson, H. E. S., and Joseph, J. (1963). Stress and occlusive coronary-artery disease.	

Table 11. Continued

Steps in Figure	Reference number	Author year, title	Summary conclusions*
From	To		
B	M	Russek, H. I., and Zohman, B. I. (1958). Relative significance of heredity, diet and occupational stress in coronary heart disease of young adults. Based on an analysis of 100 patients between the ages of 25 and 40 years and a similar group of 100 normal controls.	"The measurement of stress due to phobias, frustration, anxiety and fatigue is most difficult to accomplish particularly in retrospect in the coronary patient. Nevertheless, it was clearly evident in this study that 91 percent of the test subjects had been under unusual occupational stress for varying periods prior to the onset of clinical symptoms. Thus, 25 percent of the patients not only had worked at full time jobs during the day but also had engaged in similar or different occupations during evening hours. An additional 46 percent of the coronary group had worked 60 hours or more per week for long periods immediately preceding clinical manifestations. In another 20 percent of the group there was unusual fear, insecurity, discontent, frustration, restlessness or inadequacy in relation to employment. In marked contrast only 20 percent of all the subjects in the control series showed comparable stress and strain in relation to occupation." <i>CAVEAT</i> Same as preceding paper
B	M	Buell, P., and Breslow, L. (1960) Mortality from coronary heart disease in California men who work long hours.	Nonfarm occupations with relatively large percentages of workers working more than 48 hours per week showed somewhat higher mortality from arteriosclerotic and coronary heart disease than occupations with few workers working more than 48 hours per week. <i>CAVEAT</i> Title of paper somewhat misleading. Increased mortality not specifically tied to the subset of workers working long hours, but merely associated with <i>occupations</i> having larger percentages of people working long hours. Additionally, other possible causal factors known to be associated with cardiovascular disease (e.g., diet etc.) not controlled between the groups.
F F	L J	Nordoy, A., and Rodset, J. M. (1970). Platelet phospholipids and their function in patients with ischemic heart disease	"Patients with ischemic heart disease without recent myocardial infarction have a higher coagulant activity in platelet-rich plasma than the controls. As there is no difference between the activity in platelet-poor plasma of the two groups, this activity must be attributed to the platelets." Citing earlier literature, the authors say, "The tendency to thrombus formation in coronary arteries plays a fundamental role in the development of I.H.D. .... Firstly, thrombosis often represents the final occluding event in atherosclerotic arteries. Secondly, there is good evidence that mural thrombi may be transformed to atherosclerotic lesions. ...." <i>CAVEAT</i> The role of thrombosis as the final occluding event in most sudden cases of myocardial infarction is not as universally accepted as might be inferred from the phrasing here

Table 11. Continued

Steps in Figure	Reference number		Author, year, title	Summary conclusions*
	From	To		
F	I	X	Mustard, J. F. and Packham, M. A. (1969). Platelet function and myocardial infarction	A general review of literature including the following: "Platelets adhere to an injury site on a blood vessel wall, and a mass of platelets may accumulate at this point. ... Subsequently, the blood coagulation mechanism leads to the formation of fibrin around the platelet aggregate. ... The initial platelet-rich thrombus that forms in vessel injury is transformed to a mass of fibrin during the 24 hours after the initial injury. ... There have been a number of observations of abnormal platelet function in subjects who have had complications of atherosclerosis or in those who appear to be susceptible to it. ... (Including measures of increased platelet adhesiveness). "In man, platelet micro-emboli have been implicated in intermittent cerebral vascular and retinal ischemia. ... Moore and Meserieu ... have provided evidence that platelet emboli originating from mural thrombi in the aorta can interfere with the renal circulation, giving rise to glomerulosclerosis and elevation of blood pressure. It has recently been suggested that the localized circulatory disturbances in the myocardium precipitated by the formation of platelet aggregates may be a factor in death due to myocardial dysfunction. ..."
H I J	I M M	Y	Roberts, W. C. (1972). Coronary arteries in fatal acute myocardial infarction	A general review of literature summarized in part as follows: The coronary arteries are diffusely involved by atherosclerotic plaques in fatal acute myocardial infarction (AMI). Usually the lumens of at least two of the three major coronary arteries are narrowed more than 75 percent by old plaques in patients who die suddenly (less than 6 hours) from cardiac disease with or without myocardial necrosis. Coronary thrombi occur in about 10 percent of patients who die suddenly or in whom necrosis is limited to the left ventricular subendocardium, and in about 50 percent of patients with transmural myocardial necrosis. The infrequency of coronary thrombi in patients dying suddenly of cardiac disease and in those with transmural necrosis suggest that the thrombi may be consequences rather than causes of AMI. Although it may not precipitate AMI, coronary thrombosis may still be the underlying cause of the atherosclerosis. The findings of fibrin deposits in old atherosclerotic plaques and the findings of atherosclerotic type lesions in organized known thrombi suggest a strong relationship between thrombosis and atherosclerosis."

\*Material in quotation marks comes from the papers cited. Material outside of quotation marks represent our own interpretations, conclusions and reservations (the latter expressed under the heading "CAVEAT")

\*\*\* (not read on manuscript)

\*\*\*\* The 1/3 octave noise-band used had a middle range frequency of 4000 Hz and an intensity of 80 dB. Subjects reading, relaxing during 8-hour exposure

\*\*\*\*\* Tape recordings of noise sounds (compressed air blasts, bells, buzzers, and tuning fork impulses) presented so that each of 10 stimuli was delivered for 30 seconds followed by 1 minute of silence delivered on a randomized intermittent basis of a 4-hour period. The animals were exposed to 2 1/2 hours of sound stress and during the remaining 1 1/2 hours the animals were kept in silence. The mean sound intensity was 100 dBSPL with a frequency variation from 0.5 to 4 kHz.

\*\*\*\*\* Ischemic heart disease

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According to the EPA (ref 36).

Noise may arouse a person from sleep and/or prevent the person from falling asleep. At sub-arousal levels, noise may shift a person's sleep from a deep stage of sleep to lighter stages of sleep. The more frequent the noise is, the less likely a sleeper is to respond. Noises especially important to the sleeper can be a more effective arousing stimulus than the acoustic parameters of the noise would indicate. Adaptation to noise during sleep appears to be absent or slight.

Steady or regular periodic noises appear to affect sleep very little, although this finding appears not to have been well documented in the literature. Steady noise appears to be less disturbing to sleep than nonsteady noise of substantially lower level. According to Williams' review, Schieber, et al. (ref 40) found that relatively frequent high-density traffic sounds averaging 70 dB were less disruptive of sleep than were relatively infrequent (1 or 2 per minute) low-density traffic sounds averaging 61 dB. Using a steady noise level of 93 dB(A), which is normally unacceptably high due to hearing damage risk, Scott (ref 41) found a loss of REM sleep for the first night, although non-REM sleep states and other measures of sleep disruption were substantially unaffected. Although his data for subsequent noise nights are minimal, Scott interpreted them as suggesting that REM sleep was beginning to return to normal baseline levels.

Johnson et al (ref 42) studied the effects of long duration intermittent noise exposure on sleep. The author's summary states:

In one 15-day and one 55-day laboratory study and one operational 7-day training cruise, the effect on sleep of 24-hour-a-day exposure to pings of intensities ranging from 80 to 90 dB SPL was examined. The pings were less than a second in duration with an interstimulus interval of 45 or 22 seconds. Maximum duration of ping exposure was 30 days. In this young adult sample, exposure to the noise did not produce a decrease in sleep duration or an increase in number of awakenings. There were, however, reports of sleep onset difficulty and a decrease in percent of sleep stage 4 during ping exposure. No significant changes in waking performance or behavior were found as a result of the ping exposure during any of the three studies.

Note that this type of noise exposure is not typical of commercial shipping. However, generalization might be made to predict that repeated intermittent noise would have only minimal effects on sleep. Lukas (ref 43) states that sleep interference due to intermittent noise increases with the age of the subject and the intensity of the noise.

40. Etude Analytique en Laboratoire de l'Influence du Bruit sur le Sommeil, by JP Schieber, J Mery, and A Muzet; report of Centre d'Etudes Bioclimatique du CNRS, Strasbourg, France; reviewed in Williams (ref 39 above).
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Thiessen and Lapointe (ref 44) discuss the effect of intermittent truck noise on percentage of deep sleep. Their results suggest that adaptation to noise may take place, since both the probability of waking and the probability of a shift in sleep level decrease with continued exposure (fig 3).<sup>\*</sup> They subjected 17 subjects to 8 to 20 truck noises per night at a peak A-weighted level of 65 dB. Each subject was used for 24 nights, in 12 of which noise was presented. Stage 1, waking and dreaming (REM), was classified as light sleep, while stages 2, 3, and 4 were classified as deep sleep. The results indicated an average reduction of about 3% in the amount of deep sleep when noise was present, with great differences between subjects. The total sleep time for the noise-exposed and quiet nights was not significantly different. It was suggested that lost deep sleep could be made up for (1) in quiet intervals, (2) on subsequent quiet nights, or (3) by increasing total sleep time. The authors stated that the third suggestion was not supported by their data.

However, a recent study by Muzet and Ehrhart (ref 45) indicates that during sleep the cardiovascular system may not adapt as readily to noise exposure. This very recent paper demonstrates that low-level intermittent noise may have detrimental effects on the cardiovascular system. Also it demonstrates that cardiovascular effects of noise may be resistant to adaptation. The authors show that under laboratory conditions there appears to be no habituation of the heart rate response (HRR) to intermittent traffic noise with a peak level of 65 dB for 15 consecutive nights. The same nonhabituation features were found in the all-night average HRR to 45 and 55 dB(A) peak intensity noises, but with lower magnitude. The authors pose the question: What are the long term effects on the cardiovascular system of low intensity and even unnoticed noises that occur during sleep?

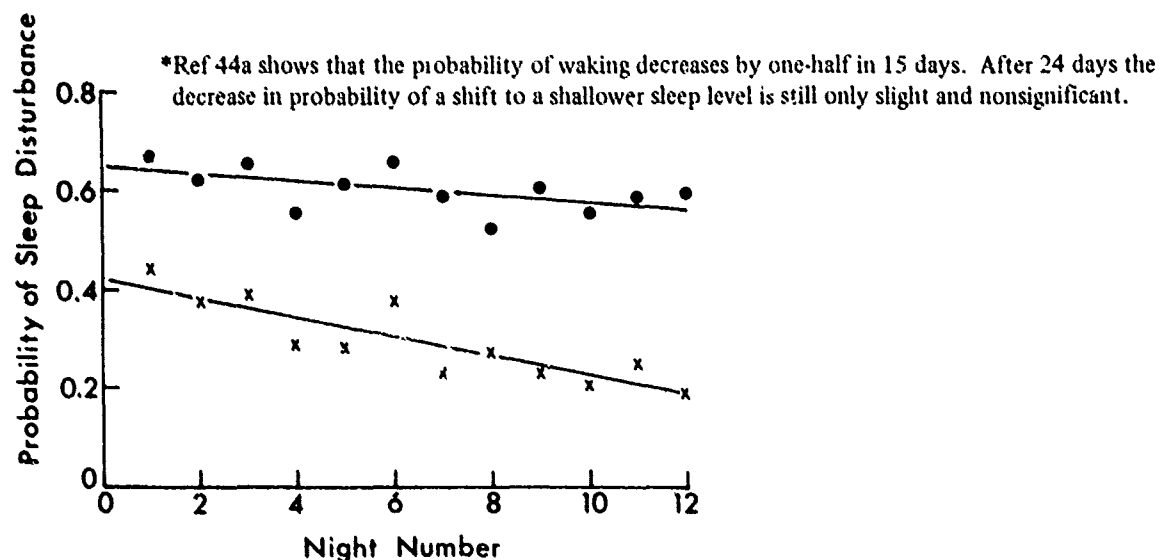


Figure 3. Adaptation to noise during sleep. The top set of points shows the probability of shift in sleep level due to truck noises with peak A-weighted levels of 65 dB presented 8-20 times per night, every other night for 12 nights for all 17 subjects. The lower set of points shows the corresponding probability of waking. The lines are linear regression lines. (From Thiessen and Lapointe (ref 44)).

44. Effect of Intermittent Truck Noise on Percentage of Deep Sleep, by GJ Thiessen and AC Lapointe; J Acoust Soc Am, vol 64 no 4, 1978, p 1078-1080.
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## SPEECH INTELLIGIBILITY

The effect of noise on speech intelligibility has been best described by Webster (ref 46). He has determined speech intelligibility as a function of distance and intensity. Figure 4 shows this relationship.

Figure 4 should be adequate to determine the speech interference to be expected from a given A-weighted sound pressure level aboard ships. Various other more accurate but more complicated measures of expected speech intelligibility in noise can also be found in Webster (ref 47).

## WORK PERFORMANCE

Noise usually causes no change in work performance. But sometimes it interferes with it, and sometimes it even improves it. Consequently this area is perhaps the most difficult to assess for purposes of social decision-making.

The human system is very flexible with respect to work performance. It can generally adapt to noise quite well, ignoring steady or periodic stimuli and using reserve processing capacity to provide constant performance. As a result, noise often has no measureable effect on work performance, even when it is annoying. After comprehensively reviewing the effects of noise on human task performance, Glass and Singer (ref 48) concluded that with three exceptions (included below) there is no compelling evidence that high-intensity noise per se has an adverse effect on task performance.

EPA criteria document 550/9-73-002 (ref 49) summarizes a large volume of experimental literature and presents a number of general conclusions (included below) which have been reaffirmed by the authors of EPA ref 50. The first general conclusion is that steady, meaningless noise does not seem to interfere with human performance unless the level exceeds about 90 dB(A). Even above this level, it does not interfere consistently. Glass and Singer attribute the lack of adverse effects of noise on performance to the human potential to adapt. Physiological measures such as galvanic skin response, vasoconstriction of peripheral blood vessels, and muscle action potentials indicate that there is a generalized stress response to noise that habituates with repeated stimulation.

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46. Updating and Interpreting the Speech Interference Level (SIL), by JD Webster; J Audio Engineering Soc, April 1970, p 114-118.
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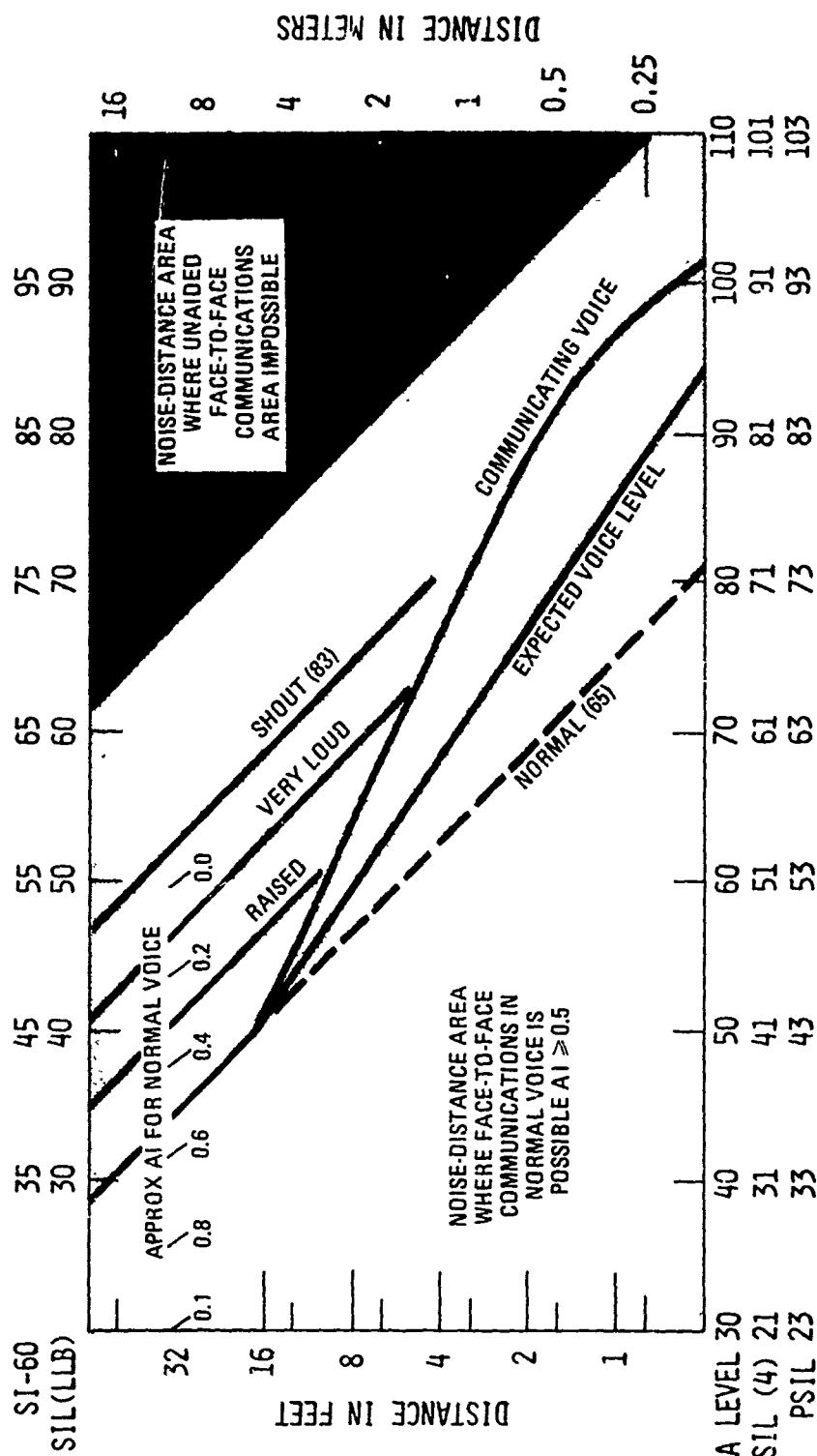


Figure 4. Necessary voice levels as limited by ambient noise for selected noise levels for satisfactory face-to-face communication. Along the abscissa are various measures of noise, along the ordinate distance, and the parameters are voice level. At levels above 50 dB(A) people raise their voice level as shown by the "expected" line if communications are not vital or by the "communicating" line if communications are vital. Below and to the left of the "normal" voice line communications are at an AI level of 0.5, 98% sentence intelligibility. At a shout, communications are possible except above and to the right of the "impossible" area line. (From Webster (ref 47), p 28).

Under some conditions, however, noise can indeed affect work performance. Steady noise levels above 90 dB(A) can sometimes affect performance (EPA ref 50). When a person is performing at the limits of his processing capacity, as in a complex or demanding task, noise may overload the system and cause a performance decrement. Performance decrements due to noise also occur in long-term vigilance tasks (Glass and Singer (ref 48)). Glass and Singer also conclude that although people adapt to noise, they may show behavioral deficits after its termination. They concluded that cognitive factors, not simply physical parameters of noise, are the important elements in the production of such aftereffects. Predictability is an important parameter. The adverse aftereffects of unpredictable noise are a function of a person's ability to predict and/or control the onset and termination of the noise. This is probably true because stressors which are unpredictable have a more aversive impact than predictable ones (Glass and Singer (ref 48)). Glass and Singer suggest that there is a tendency toward reduced physiological reactions when a person believes he can control the noise. Aperiodic noise bursts may interfere with performance, even at levels below 90 dB(A) (Glass and Singer (ref 48); EPA (ref 50)). Intermittent and impulsive noises interfere more than do steady noises (EPA ref 50). Noise components above about 2 kHz usually produce more interference than low-frequency components (EPA (ref 50)).

Noise is more likely to reduce the accuracy of work than the quantity of work. It usually does not influence overall rate of work. But high levels may increase variability in work rate by causing pauses which may be followed by compensating increases in work rate (EPA (ref 50)).

#### **ABSENTEEISM, ACCIDENT, AND INJURY RATES**

Increased absenteeism may be caused by psychological aversion to noise and/or a general lowering of immunological resistance to infection. Increased accident and injury rates may be caused by performance decrease due to noise or masking of warning signals.

A joint study by Raytheon and NIOSH found suggestively higher incidence of absenteeism, illnesses, and accidents when workers were subjected to greater effective noise exposure.

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