

SUBJECT TO ERRATA CORRECTION

**PUBLIC HEALTH AND WELFARE
CRITERIA FOR NOISE
July 27, 1973**

FOREWORD

The Noise Control Act of 1972 requires that the Administrator of The Environmental Protection Agency (EPA) develop and publish criteria with respect to noise. These criteria are to "reflect the scientific knowledge most useful in indicating the kind and extent of all identifiable effects of noise on the public health and welfare which may be expected from differing quantities and qualities of noise." This document meets that requirement.

The terms "criteria and standards" are generally used interchangeably in the scientific communities concerned with noise and its control. However, in accordance with the intent of the U.S. Congress, criteria for environmental pollutants are to reflect an honest appraisal of available knowledge relating to health and welfare effects of pollutants, (in this case, noise). The criteria are descriptions of cause and effect relationships. Standards and regulations must take into account not only the health and welfare considerations described in the criteria, but also, as called for in the Noise Control Act of 1972, technology, and cost of control. This criteria document, therefore, serves as a basis for the establishment of the recommended environmental noise level goals to be related to the "Effects Document" called for by Section 5(a)(2) of the Noise Control Act. That document, along with this criteria document, will become the basis for standards and regulations called for by Sections 6 and 7 of the Noise Control Act.

Further, the terms "health and welfare," as used in the Noise Control Act include, as in other environmental legislation, the physical and mental well being of the human populations. The terms also include other indirect effects, such as annoyance, interference with communication, and loss of value and utility of property, and effects on other living things.

In preparing this Criteria Document, EPA has taken into account the vast amount of data in the general professional literature and the information contained in the "Report to the President and Congress on Noise" and its supporting documents prepared under Title IV, PL 91-604. To bring to bear the views and opinions of some of the world's leading experts on current knowledge regarding the effects of noise, EPA sponsored an International Conference on Public Health Aspects of Noise, in Dubrobnik, Yugoslavia in May 1973. The proceedings of that conference have been applied to the preparation of this document. They are available, as stated in the Appendix to this document.

The criteria presented herein shall be revised and elaborated upon as the results of continuing investigations on the effects of noise on health and welfare become available.

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ABBREVIATIONS

AAOO	American Academy of Ophthalmology and Otolaryngology
AC	Air Conduction
AFR	Air Force Regulation
AHL	Average Hearing Level
AI	Articulation Index
AMA	American Medical Association
ANSI	American National Standards Institute (formerly USASI)
BC	Bone Conduction
CHABA	Committee on Hearing and Bio-Acoustics
dBA	A-weighted decibel (decibels)
DRC	Damage Risk Criterion
E _A	Noise emission level
EPA	Environmental Protection Agency
HL	Hearing Level
IEC	International Electrotechnical Commission
ISO	International Organization for Standardization
L _{eq}	Equivalent continuous sound level
NIHL	Noise-Induced Hearing Loss
NIOSH	National Institute for Occupational Safety and Health
NIPTS	Noise-Induced Permanent Threshold Shift
NITTS	Noise-Induced Temporary Threshold Shift
NPL	Noise Pollution Level (also National Physical Laboratory in England)
NR	Noise Rating
OSHA	Occupational Safety and Health Act
PB	Phonetically Balanced
PIHO	Percent Impairment of Hearing (Overall)
PIHS	Percent Impairment of Hearing for Speech
PTS	Permanent Threshold Shift
RMS	Root Mean Square
SIL	Speech Interference Level

SIR	Speech Impairment Risk
SPL	Sound Pressure Level
TTS	Temporary Threshold Shift

SECTION 1

NOISE & NOISE EXPOSURES IN RELATION TO PUBLIC HEALTH AND WELFARE

From a strictly scientific position, noise is discordant sound resulting from nonperiodic vibrations in air. In common usage, noise is defined more simply as unwanted sound and has sometimes been categorized as sound without value or noise pollution. To understand noise as an environmental issue affecting public health and welfare as discussed in this document, one must understand certain fundamentals of sound and human responses to it. However, a detailed discussion on the fundamentals of physics and bioacoustics is beyond the scope of this document. The following material is provided only as a general orientation for those unfamiliar with the subject of noise; to provide a better understanding of the effects of noise on man and its environment as discussed in the subsequent sections. Those desiring further information should consult Appendix A, which lists some of the numerous references available in the current literature. Attention is also directed to the Glossary.

HUMAN EXPOSURE AND RESPONSE

Physiological Response

Sound is generated by a source producing vibrations (sound waves) that may travel through any media and which, in air, actuate the hearing mechanisms of humans and animals. These vibrations set in motion the ear drum and small bones or ossicles of the middle ear as shown in the schematic drawing in Figure 1-1. The motion of the ossicles, in turn, produces vibrations in the fluid in the inner ear's sensory organ, the cochlea. The vibrations are then transduced into nerve impulses by sensory hair cells and transmitted to the brain, where they are perceived as sound or, depending upon circumstances, as noise.

Central to the health and welfare aspect of noise, is the wide range of response of the human hearing mechanism. The human ear can discern without pain sounds ranging from a threshold of detection to sounds 10^{12} times as intense. This should be contrasted with the human eye, which responds to light intensities from its threshold of response up to an intensity 105 times greater. This wide range of hearing response and the complexity of the various attributes of that response

AUDITORY PRESENTATION OF INFORMATION

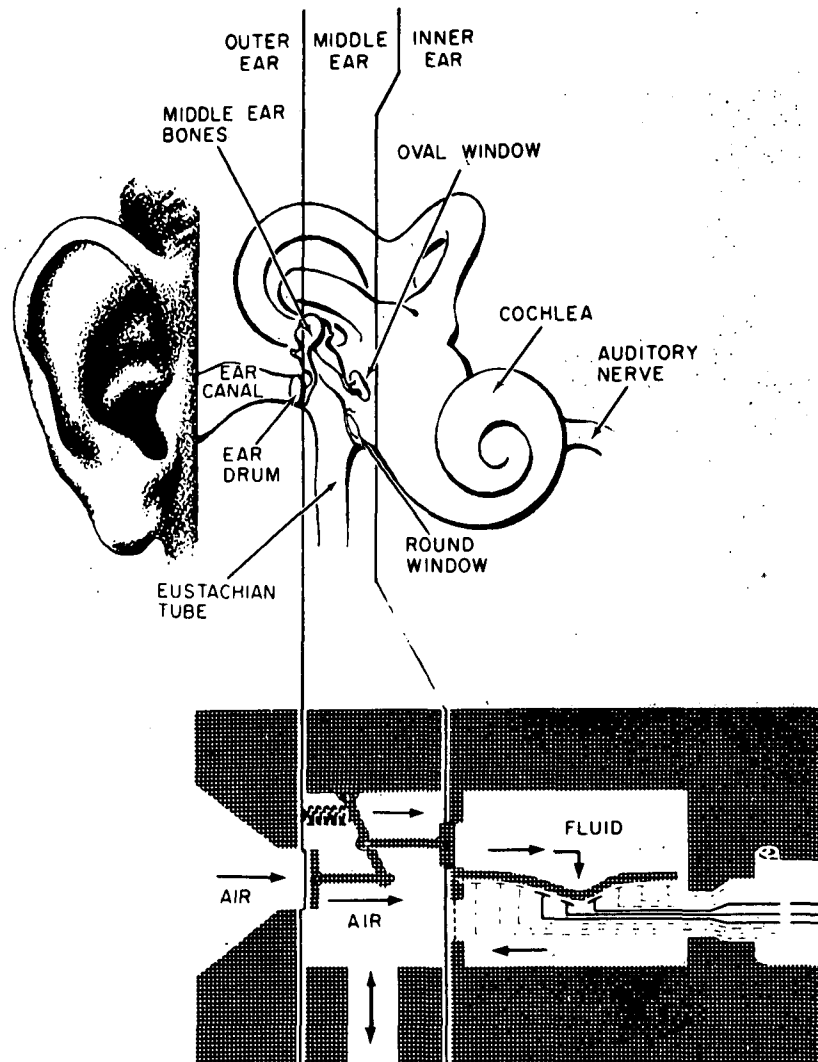


Figure 1-1. Functional Diagram of Ear

is reflected in the systems used in defining and measuring noise, as discussed in Section 2 of this document and elsewhere herein.

Another major element of public health concern, which is the subject of detailed discussion in Section 3, is that the hair cells, vital to the hearing process, are nonregenerative. Thus, if they are damaged or destroyed following certain sound exposures, there is no physiological restoration.

Also of major importance is the fact that the process of hearing (as used here meaning the perception and understanding of sound) is one of the main sensory contacts of man and other animals with their environment. Hearing is second only in importance to vision in this regard. Further, there are extremely complex relationships between these two processes that are far beyond the scope of this presentation and will not, therefore, be discussed here.

Psychological Response

Beyond the relatively obvious aspects of sensory-environmental contact, there is a deep and exceptionally intricate human emotional and psychological response to sound. Prayers, hymns, anthems, the soft and sometimes harsh sounds of nature, the blatancy of martial music, the thunder of applause, the welcome lilt of a loved one's voice, and the regular occurrence of an expected tone, such as a distant whistle, are indicative of the capability of sound to produce within people (and in some animals) profound individual and group responses and reactions. These responses range from pleasure to fear and include all other aspects of human emotional reaction. Some reactions may be attributed to the message conveyed by the sound, prior experiences and conditioning, and many other poorly identified processes.

Traditionally, in a great many cultures quiet is used to indicate respect, while loud sounds and noises are indicative of ridicule disrespect or disapproval. Even here, however, there are contradictions. As an example, a loud cheer indicates approbation but equally loud signals can be and are used to indicate disapproval.

Speech Interference

The effects of noise on the ability to communicate are perhaps an even greater influence on the human reactions to noise. These reactions are discussed in much greater detail in the following sections of this document. Interference with communication may arise either from actual impairment of the hearing process or from intrusions of sound so that the message cannot be understood by the listener. The expression "I could hardly make myself heard" is an example of a reaction of frustrations to such situations. It very well may reflect part of the origin of the annoyance reactions and other nonphysiological responses discussed in this document.

Still another problem is that what is pleasurable sound to a particular listener at a particular point in time may be noise to some other listener. Further, a pleasurable sound may also be considered as noise when heard at a different time and under different circumstances. An example

of this would be a situation in which a devotee of a particular type of music enjoys it in his domicile but causes annoyance to his neighbors because of the volume of his sound reproduction equipment. Later, that individual may himself be annoyed by the same musical composition if it interfered with his sleep.

HISTORICAL CONCERN

Noise is hardly a new concern for society. It has apparently been a problem for most of mankind's existence. There is reportedly an ordinance enacted some 2,500 years ago by the ancient Greek community of Sybaris banning metal works and the keeping of roosters within the city to protect against noise that interfered with speech and might disturb sleep.¹ There are many other examples to show this historical concern with noise. They include Juvenal's statement regarding noise from wagons and their drivers interfering with sleep in ancient Rome and Chaucer's poem of around 1350 complaining of noise by blacksmiths and that because of them "no man can get a night's rest." Also, Benjamin Franklin some 400 years later reputedly moved from one part of Philadelphia to another because "the din of the market increases upon me; and that has I find made me say somethings twice over."²

Over the past 200 years there has been a steady increase in the magnitude of the impact of noise, changing the nature and extent of the problem from that of primarily nuisance and annoyance to actual physiological damage. While the sources of noise are different, and their numbers and the magnitude of sound energy have created a larger impact, the character of the impact of noise is not new or radically different. It is the addition of new noise sources in already noisy situations and the proliferation of noise sources of increased output into previously relatively quiet areas that has stimulated greatly increased public concern and has created the need for increased governmental action. In many ways, the present situation regarding noise is not different from that of other pollutants, with the possible exception that, unlike some pollutants, once the noise source is controlled or reduced, the impact of the noise correspondingly changes almost immediately.

PHYSICS OF SOUND

Up to this point, some of the considerations of human exposure and response have been discussed. The following discussion highlights some of the essential information on the physics of sound, needed for a more complete appreciation of the material in the subsequent sections.

Sound Waves

At the outset of this section, it was stated that sound was the result of a source setting a medium into vibration. Generally, insofar as noise is concerned, that medium is air; and the following discussions are related solely to that medium. However, to a large extent, sound is to air what

waves are to water. Whenever an object moves back and forth in air, it causes the invisible molecules of the air to likewise move back and forth. This vibration produces, in a cyclical fashion, alternating bands of relatively dense and sparse particles, spreading outward from the source in the same way as ripples do on water after an object is thrown into it. This movement of particles is produced as a result of the energy delivered to a source, such as the clapping of the hand, the beating on a drumhead, or the pulling of a bow across the strings of a violin. The result of the movement of the particles is a variation in the normal atmospheric pressure, or sound waves. These waves radiate in all directions from the source and may be reflected and scattered or like other wave actions, may turn corners, or be refracted. They can be combined with or even be cancelled by other sound waves. Likewise, the energy contained in the sound can be absorbed. As the waves travel over increasing distances, the amount of energy per unit area contained in them is reduced proportionally to distance. Once the source ceases to be in motion, the movement of the air particles ceases and the sound waves almost instantaneously disappear and the sound ceases. Under normal conditions of temperature, pressure, and humidity at sea levels, these sound waves travel at approximately 1100 feet per second.

Intensity of Sound

Sound may be scientifically described in terms of three variables associated with the characteristics of waves:

1. Amplitude (intensity)
2. Frequency (pitch)
3. Duration (time)

Figure 1-2 depicts these elements.

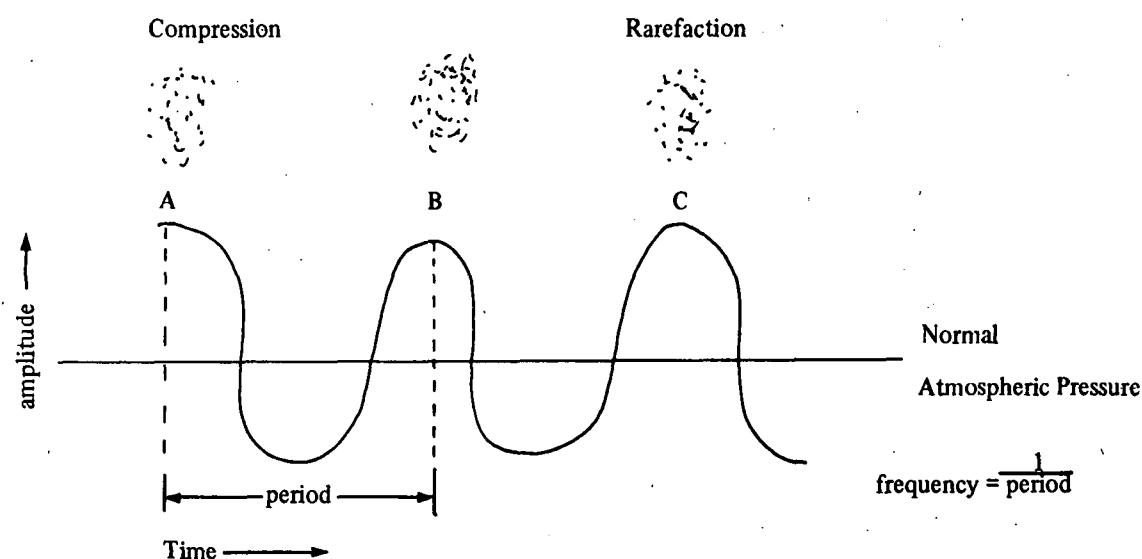


Figure 1-2. Elements of a Sound Wave

Sound intensity is the average rate of the sound energy transmitted through a unit area (usually stated as watts per square meter). (The large range of sound intensity involved in human response is shown in Table 1-1.) There are physical and mathematical relationships that exist between the energy of sound waves and the resulting variation from atmospheric pressure. Sound pressure (usually stated in terms of newtons per square meter) is the amplitude or measure of that variation from atmospheric pressure. Presently, there are no instruments to directly measure sound power (the total amount of energy radiated per unit time by the sound source) or sound intensity. Accordingly, sound pressure is used as the fundamental measure of sound amplitude and is one of the basic ingredients of the various measurement and rating schemes in systems described in Section 2.

Earlier in this Section, it was pointed out that the human ear has a wide range of response to sound. Sharply painful sound is 10 million times greater than the least audible sound (20×10^7 micro newtons per square meter as compared with 20 micro newtons per square meter). Such a wide range of values creates problems in measurement and computations associated with noise. Accordingly, in acoustics as in electrical engineering, the concept of level is used for defining sound (and thus noise) intensity. The level in this usage is the logarithm of the ratio of a quality (in this case, sound pressure) to a reference quality of the same kind (for sound pressure, 20 micro newtons per square meter). The unit of measure is the decibel and the formula for sound pressure level (SPL) is:

$$\text{SPL} = 10 \log \left(\frac{P_2}{P_1} \right)^2 = 20 \log \frac{P_2}{P_1},$$

where P_2 is the pressure in newtons per square meter and P_1 is the reference value. (See EPA NTID 300.15, "Fundamentals of Noise Measurement," or other references cited in Appendix A for details of the relationships between sound intensity in energy and sound pressure).

The relationship of sound pressure in terms of micro newtons per square meter to corresponding decibel levels is shown in Figure 1-3. Note that for each 20-decibel increase there is a corresponding 10-fold increase in acoustical pressure and sound pressure. Using this scheme, some complications may arise for those not well versed in its fundamentals. As an example, sound pressure levels expressed in decibels are not directly additive. That is, a source producing an 80-dB SPL when added to another one producing that same SPL at the same distance results in only a 3-dB increase, not a doubling to 160. Further, if there is a difference in the sound pressure level of the two sources, the amount of increase will be smaller to the point that if such a difference is 10 decibels, the lesser source will virtually be of no consequence in terms of increasing the sound pressure level.

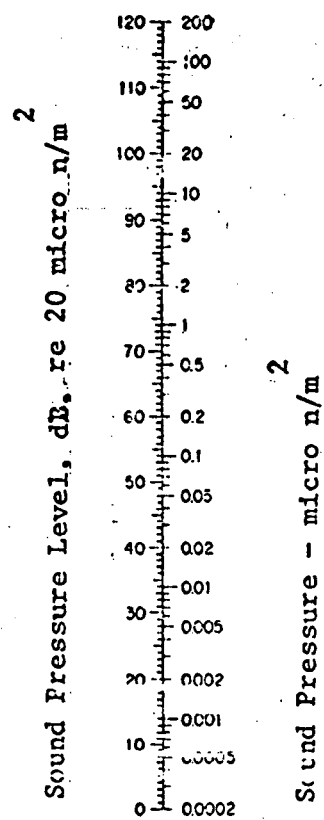
Frequency of Sound

The number of compressions and rarefactions of the air molecule density in a unit of time associated with a sound wave are described as its frequency. The unit of time is usually one second, and the term "Hertz" (after an early investigator of the physics of sound) is used to designate these

TABLE 1-1
ACOUSTIC POWER AND SOUND LEVELS OF
TYPICAL NOISE SOURCES

Power	Power Level	Source
watts/m ²	dB re 10 ⁻¹² watts/m ²	
100,000	170	Ram jet Turbojet engine with afterburner
10,000	160	Turbojet engine, 7000 lb thrust
1,000	150	4-propeller airliner
100	140	75-piece orchestra
10	130	Pipe organ Peak RMS levels in 1/8-second intervals
3*		Small aircraft engine
1.0	120	Large chipping hammer Piano Peak RMS levels in BB ^b tuba 1/8-second intervals
0.1	110	Blaring radio Centrifugal ventilating fan (13,000 CFM)
0.01	100	4' loom Auto on highway
0.001	90	Vaneaxial ventilating fan (1500 CFM) Voice — shouting (average long-time RMS)
0.0001	80	
0.00001	70	Voice — conversational level (average long-time RMS)
0.000001	60	
0.0000001	50	
0.000,000,01	40	
0.000,000,001	30	Voice — very soft whisper

* SPL = PWL -30 dB; 3 watts = 94 dB; OSPL = 10 X 10⁻⁶ PSI



NOTE

- 1 Newton /m² = 94 dB re 20 micro n/m²
- 1 Pound/sq in = 170 dB " " " "
- 1 Pound/sq ft = 127.6 " " " "

Figure 1-3. Relationship of Sound Pressure to Corresponding Decibel Levels

cycles per second. Again, the human ear and those of most animals have a wide range of response. Humans can identify sounds with frequencies from about 16 Hz to 20,000 Hz. The musical pitch "A" above middle "C" is produced on a piano by the key-activated hammer striking a string, which then oscillates back and forth at a rate of 440 Hz, producing a fundamental frequency of 440 Hz.

Pure tones are relatively rare in real life situations. Most human exposures consist instead of a complex mixture of many frequencies. Some typical examples are shown in Figure 1-4.

Duration of Sound

The temporal nature of sound relates to the duration of its generation and presence. Continuous sounds are those in which the source is producing sound for relatively long periods in a constant state, such as the noise of a waterfall. Intermittent sounds are those which are produced for short periods, while impulse sounds are those which are produced in an extremely short span of time.

TYPES OF NOISE AFFECTING PUBLIC HEALTH AND WELFARE

To evaluate the effect of noise on public health and welfare, it has been necessary to define different types of noise fairly explicitly, since a complex sound may, and usually does, involve a mixture of sounds of varying intensity, diverse frequencies and temporal patterns.

Types of noise frequently differentiated are ongoing noise and impulsive noise.

Types of Ongoing Noise

Ongoing noise continues for an appreciable period of time. It is further differentiated into steady-state, fluctuating, and intermittent noise as described in Table 1-2.

Steady-state noise is noise whose quality and intensity is practically constant (varying less than ± 5 dBA) over an appreciable period of time. Different types of steady-state noise are illustrated in Figure 1-5.

Fluctuating noise is continuous, but its intensity rises or falls more than 5 dBA. Intermittent noise is discontinuous and differs from impulsive noise in being of longer duration and less specifically defined.

Impulsive Noise

Impulsive noise is one or more transient acoustical events such as a gunshot, each of which lasts less than 500 milliseconds and has a magnitude (change in sound pressure level) of at least 40 dB within that time. A single impulse may be heard as a discrete event occurring in otherwise quiet conditions, or it may be superimposed upon a background of steady-state on-going noise. It may be characterized by the following basic parameters:

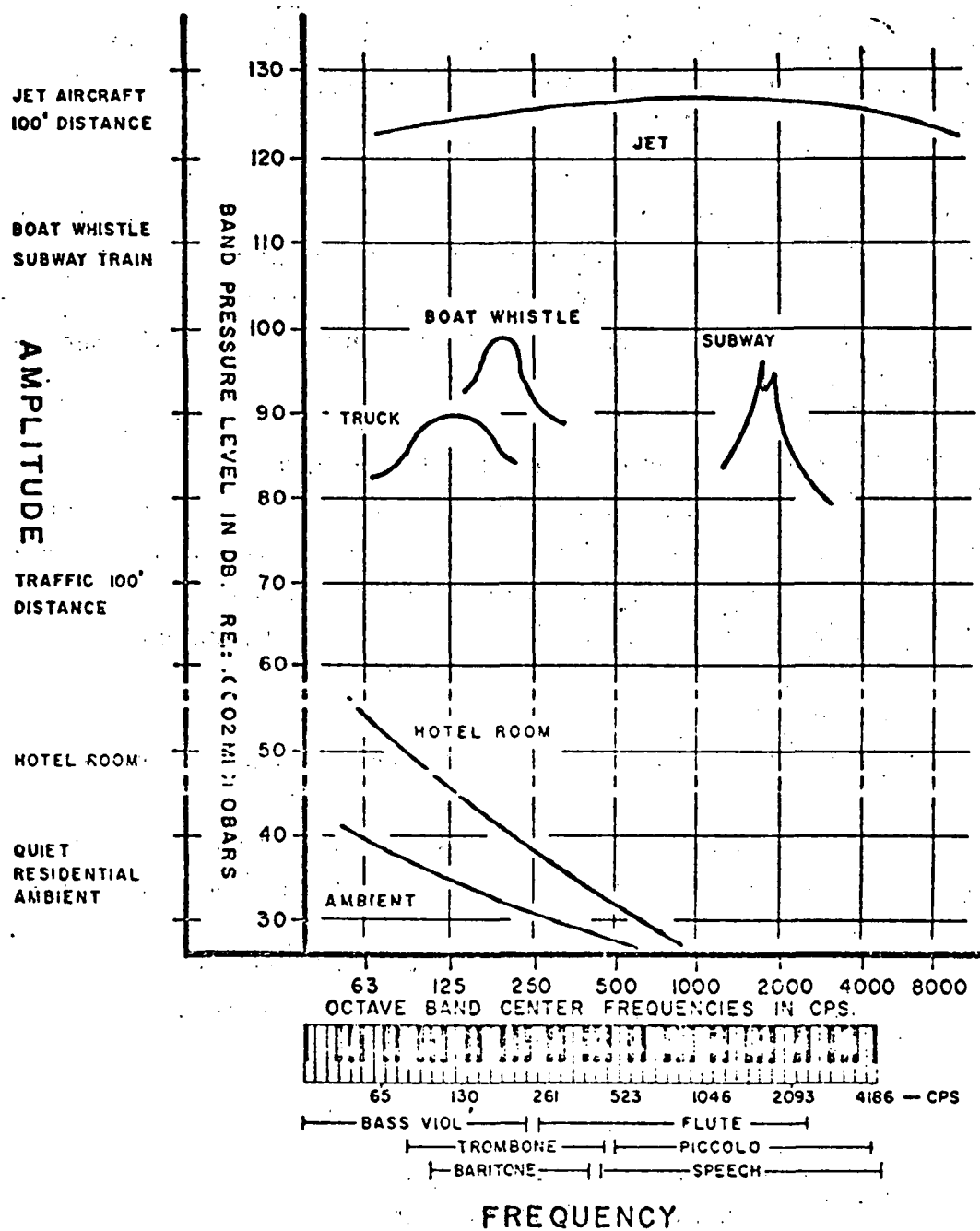
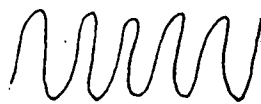


Figure 1-4. Noise Spectra

TABLE 1-2
CLASSIFICATIONS OF ONGOING NOISE EXPOSURE

Type of Exposure	Description	Typical Examples
Steady-State	Single continuous daily exposure (typically 8 hours but may be shorter or longer) at a constant level within ± 5 dBA.	Weaving room noise; sound of a waterfall; shipboard noise; interior of a vehicle or aircraft noise; turbine noise; hum of electrical sub-station.
Fluctuating Noise	Noise is continuous but level rises and falls (rapidly or gradually) more than 5 dBA during exposure.	Many kinds of processing or manufacturing noise. Traffic noise; airport noise; many kinds of recreational noise (e.g., vehicle-racing; powered lawnmowing; radio and TV).
Intermittent Noise	Noise is discontinuous: i.e., the level falls to unmeasurable low or to non-hazardous levels between periods of noise exposure of which more than one affects the ear during the day. Note: This can be regarded as a special case of fluctuating noise.	Many kinds of industrial noise (especially in construction work, ship building, forestry, aircraft maintenance, etc.); Many kinds of recreational noise (e.g., drag racing, rock concerts, chain-sawing); light traffic noise; occasional aircraft flyover noise; many kinds of domestic noise (e.g., use of electrical appliances in the home); school noise.



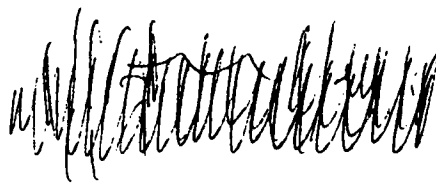
Pure Tones



Periodic Noise



A-periodic Noise



White noise

Figure 1-5. Representation of Various Types of Noise

1. Peak sound pressure level (in dB re 0.00002 N/m²). For reasons connected with measurement practice in the English-speaking countries, the over-pressures associated with sonic booms in aerospace operations are customarily expressed in pounds/ft² (psf) relative to atmospheric pressure. This convention is adhered to in this document when citing data expressed in psf by other authors.
2. Duration (in milliseconds or microseconds)
3. Rise and decay time
4. Type of waveform (time-course)
5. Spectrum (in case of oscillatory events, Type B—see Figure 1-6)
6. Number of impulses

Two types, "A" and "B," are shown in Figure 1-6. In the Type A impulse, there is a rapid rise to a peak SPL followed by a decay to a negligible magnitude. In the classical "Friedlander" type of event, a subsequent negative pressure wave occurs, of much smaller magnitude. In evaluating this type of wave only the duration of the positive part of the event is counted as the duration of the impulse. In the single Type B (oscillatory) event, the duration is taken as being the time taken for the envelope to decay to a value 20 dB below the peak. It is important to appreciate that impulse noises can be distinguished as to type and properly measured only by oscillographic techniques due to their short duration.

SUMMARY – TYPES OF NOISE AFFECTING PUBLIC HEALTH AND WELFARE

Historical evidence shows that excessive noise has long been considered a menace to the public health and welfare. Over the past two centuries, industrial development has resulted in a steady increase in the extent of noise impact.

Noise can affect the ability to communicate or to understand speech and other signals. This may arise from either actual impairment of the hearing mechanism or as a result of intrusions of sounds such that the desired ones cannot be understood by the listener.

The physics of sound provide the appropriate background for the difficult task of assessing human response to noise. As sound waves travel over increasing distances, their energy diminishes proportionally, being spread over an ever increasing area. Once the source ceases to be in motion, the movement of the air particles ceases and the sound waves usually disappear almost instantaneously.

Sound may be described scientifically in terms of three variables associated with the characteristics of waves. These are its amplitude (intensity), its frequency (pitch), and its duration (time). Sound intensity is the average rate of sound energy transmitted through a unit area. Frequency is the number of compressions and expansions of the air molecules in a unit of time associated with a sound wave. The temporal nature of sound relates to the duration of its generation and presence. The variables of sound make sound measurement a complex problem.

INSTANTANEOUS PRESSURE OF IMPULSE

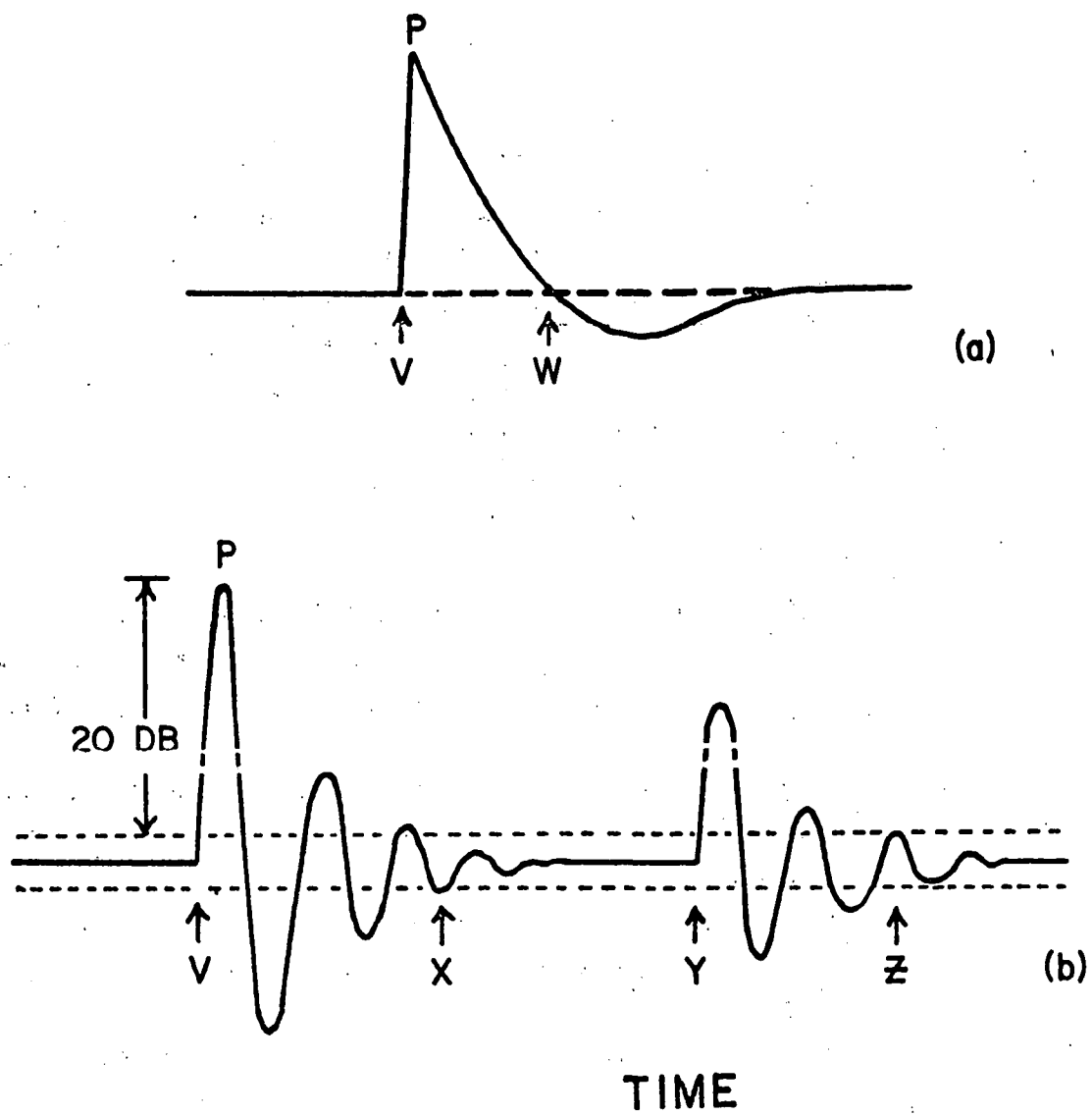


Figure 1-6. Two Principal Types of Impulse Noises
(Taken from Ward³)

Noise is frequently differentiated into ongoing and impulsive noise, to evaluate its effect on public health and welfare. Ongoing noise is further differentiated into steady-state, fluctuating, and intermittent noise.

REFERENCES

1. Cited in Communication of W. Dixon Ward to EPA, Office of Noise Abatement and Control, 1973.
2. Hilderbrand, James L., "Noise Pollution and the Law" William S. Hien & Co., Buffalo, N.Y., 1970, p. 22.
3. Ward, W.D. (ed.) PROPOSED DAMAGE-RISK CRITERION FOR IMPULSE NOISE (GUN-FIRE). Report of Working Group 57, NAS-NRC Committee on Hearing, Bioacoustics, and Biomechanics (1968).

Section 2

RATING SCHEMES FOR ENVIRONMENTAL COMMUNITY NOISE

The description of noise encountered in most living situations must account for:

1. Those parameters of noise that have been shown to contribute to the effects of noise on man (amplitude, frequency and duration). These parameters have already been discussed in Section 1.
2. The variety of noises found in the environment (transportation noises, construction, home appliances and others).
3. The variations in noise levels that occur as a person moves through various locations of the community.
4. The variations in noise levels associated with the time of day at any given location.

Thus, the task of describing community noise is to determine the time and location variations in the noise environment throughout the community so that the descriptions are relevant to the effects of environmental noise on people, whether they are located indoors or outdoors. This chapter will not completely describe all the schemes that have been developed over the years but, rather, selects a few rating schemes to illustrate the techniques and problems involved, so as to facilitate the understanding of the rest of this document. The interested reader can find a complete description of rating schemes in numerous texts such as the *Effects of Noise on Man*,¹ *Fundamentals of Noise Measurement, Rating Schemes, and Standards*,² and *Transportation Noises*³ and others. The following section of this document will review the actual findings regarding annoyance caused by noise and the community reaction to that noise.

BASIC PHYSICAL PARAMETERS

As pointed out in Section 1 the basic parameters of sound, in terms of its effects on man and its environment are:

- The amplitude of sound.
- Frequency content of sound.
- The variation in time.

Thus, a complete physical description of sound must account for its frequency spectrum, its overall sound pressure level, and the variation of both these quantities with time.

Because it is difficult and cumbersome to present or to understand data having three dimensions, considerable effort has been expended over the last 50 years to develop scales that reduce the number of dimensions into a one-number scheme.⁴ Most of the effort has been focused on combining measures of the frequency content and overall level into a quantity proportional to the magnitude of sound as heard by a person.⁵

Physical Parameters of Sound and Psychological Perception of Sound

There have been many studies as to the relationship between the physical parameters of sound and the psychological perception of sound.

Although there are disagreements in the results of these studies and in the views of their principal investigators regarding the actual values of the constants entering into the function relating loudness experience and intensity of stimuli, there appears to be some consensus regarding the form of the relationship. Loudness appears to grow as a power function of sound pressure. Thus, for a pure tone, loudness is proportional to the sound pressure. This relationship has been called the Power Law by Stevens.⁶⁻⁸ In practice what this means is that for a 1000-Hz tone, for example, the loudness of a tone increases by a factor of two for each 10-dB increase in the intensity of the stimulus.*

In making loudness measurements one often uses reference sounds. In the earlier work, the reference was a 1000-Hz tone. The choice of a 1000-Hz tone as the reference has been proposed originally by Fletcher and Munson.³ The reason for choosing a 1000-Hz tone is stated by Fletcher and Munson as follows:

- "1) It is simple to define; 2) it is sometimes used as a standard of reference for pitch;
- 3) its use makes the mathematical formulae more simple; 4) its range of auditory sensitivities . . . is as large and usually larger than for any other type sound; 5) its frequency is in the mid-range of audible frequencies."⁹

When an observer is required to compare the loudness of a tone to that of the reference, the process is done by having the listener adjust the intensity level of the tone being rated until its loudness matches that of the reference tone. The result is referred to as loudness level. Loudness level is expressed in phons. The units of the phon are the sound pressure level (SPL) of a 1000-Hz tone heard in a free field and judged to be equal in loudness to the sound in question.

*Stevens, provides a variety of evidence for this rule. In some of his experiments the subjects were asked to equate apparent loudness of sound to intensity on some other continua, such as mechanical vibration on the skin, brightness of spots of light, or force of hand grip. Results matched Stevens predictions based on the relation between the intensity of stimuli and various psychophysical scale. For example, it is demonstrated that it requires a change of about 9 dB to double the perceived brightness of a spot of light, whereas about 10 dB is required to double the loudness of the 1000-Hz tone.

Equal Loudness Contours

A number of experiments have concerned themselves with establishing equal loudness relation for pure tones or for bands of noise. The relationships thus obtained show at what intensities tones of different frequencies appear equal in loudness to a 1000-Hz tone presented at various intensities. An example of those are reproduced from Robinson and Dadson in Figure 2-1.

Observation of Figure 2-1 will reveal that the ear is most sensitive in the region between 500-Hz and 6000-Hz and that at low sound pressure level, the ear normally hears low frequency sound as less loud for equal sound pressure levels (frequencies below 250-Hz). Further, as the intensity is increased to moderate levels, the ear gives greater weight to sounds of low frequency. Finally, at very high intensity, the response of the ear becomes flat, that is the loudness of a pure tone depends primarily on the sound pressure level and is little affected by frequency.

The findings just described are embodied in the most commonly used instrument for measuring noise: the sound level meter. The typical sound level meter electronically weighs the amplitudes of the various frequencies approximately in accordance with a person's hearing sensitivity and sums the resulting weighted spectrum to obtain a single number.⁵ Typically, the sound level meter contains three different response weighting networks: the A, B and C networks. The A-weighting network is intended to match the response of the ear to sound of low intensity. The B-weighting network is intended to match the response of the ear to sound of moderate intensity. The C-weighting network is intended to match the response of the ear to sound of high intensity. The three weightings of the sound level meter are illustrated in Figure 2-2. Also shown is the proposed D-weighting curve for monitoring jet aircraft noise. From the curves it can be seen that for a 50-Hz pure tone the reading on the A scale (which discriminates against low frequency sounds) would be 30 dB less than the C scale reading.*

The most commonly used scale on the sound level meter is the A weighting, since it has been found to account fairly well, although not perfectly, for man's perception of sound.⁵

When using the sound level meter on the A-weighting, the quantity obtained is the A weighted sound level. Its unit is the decibel (dB) often popularly referred to as dBA.

Although the A weighting is a good indicator of man's perception of sound, it is not perfect. For this reason, many other scales have been developed that attempt to better quantify "loudness" or "noisiness."⁵ The evolution of only one of these will be presented here as an illustration. The interested reader is referred to standard texts that have already been listed at the beginning of this section.

*International Electrotechnical Commission (IEC) Recommendations 123 and 173 and American National Standard Institute (ANSI) Standard S1.4-1971.

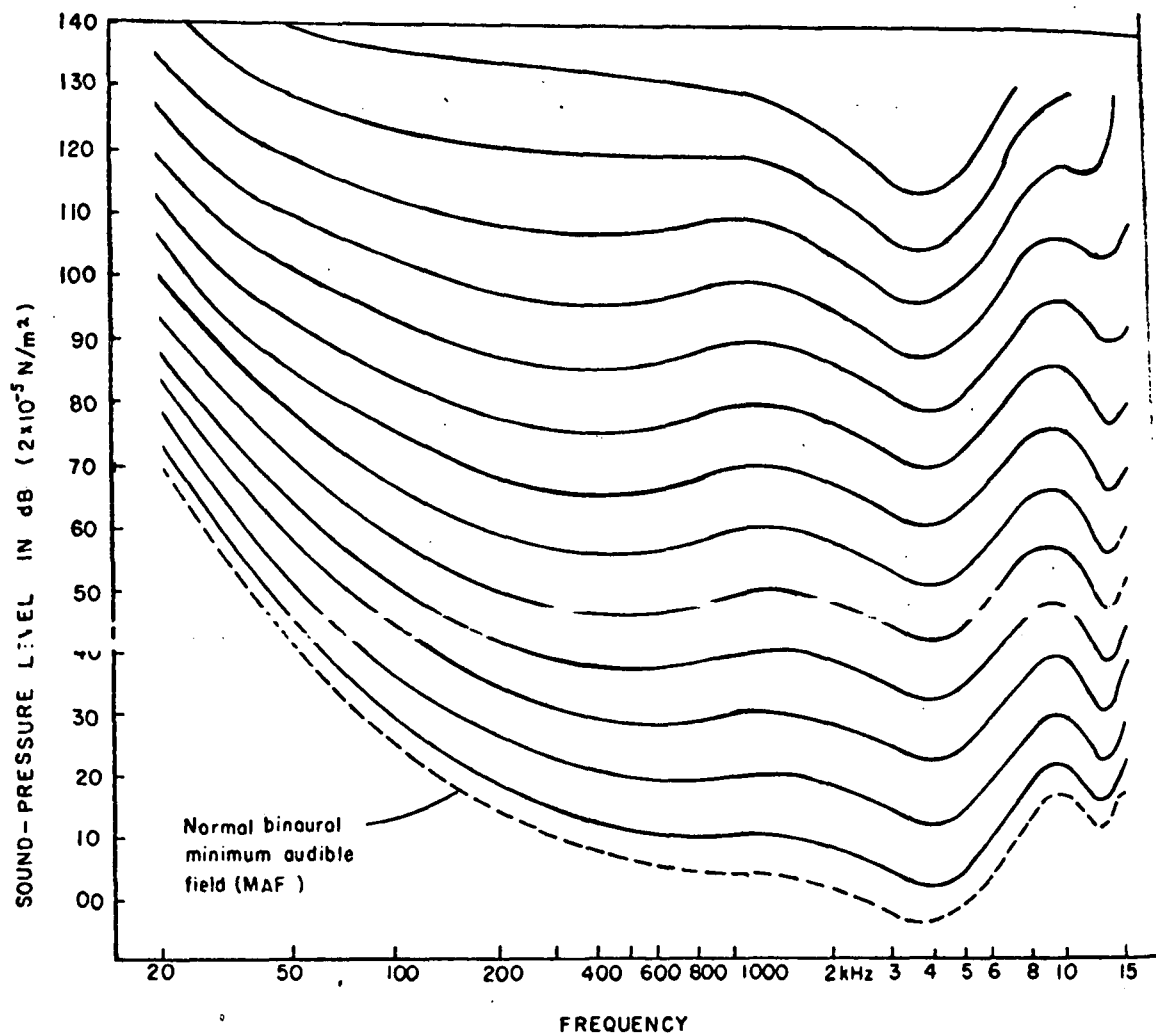


Figure 2-1. Normal Equal-loudness Contours for Pure Tones (Binaural Free Field Listening) Developed by Robinson and Dadson in Great Britain.*

*Robinson, D.W. and Dadson, R.S., A Redetermination of the Equal Loudness Relations for Pure Tones, *British Journal of Applied Physics*, I, 1956, 166-181.

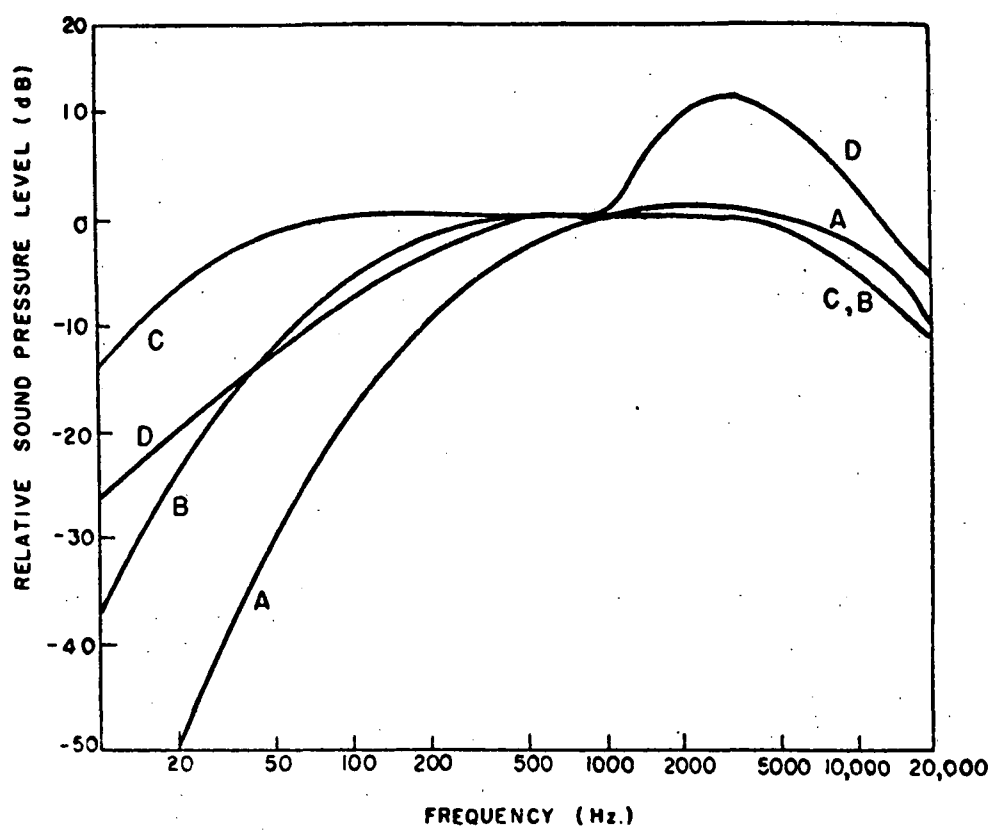


Figure 2-2. IEC Standard A, B, and C, Weighting Curves for Sound Level Meters

Perceived Noise Level

Kryter, in the late 1950's, developed a new scale of perceived intensity called the Perceived Noise Level.¹⁰ Its units are decibels. It is often popularly referred to as PNdB which:

“Was intended to present the sound pressure level of an octave band of noise at 1000 Hz which would be judged equally noisy to the sound to be rated. Equally noisy means that in a comparison of sound one would just as soon have one noise as the other at his home during the day or night.”

Later, Kryter and his associates refined this technique further to include discrete frequency components of tones associated with aircraft flyovers.¹¹ The resulting measure is the Tone Corrected Perceived Noise Level, abbreviated as PNLT. Finally, since long duration flyovers appear to be more annoying than short duration flyovers, a new correction was added by Kryter and Pearsons to account for the duration of the noise signal. This new quantity is called the Effective Perceived Noise Level (EPNL). This quantity is somewhat more exact than the A-weighting in relating man's perception of sound to the physical parameters of sound, particularly in the case of aircraft noise. For this reason, it has become a major element in the procedures utilized by the Federal Aviation Administration for the certification of aircraft noise.¹²

For most sounds, the Perceived Noise Level exceeds the A-weighted noise level by 13 dB, the differences ranging typically between 11 and 17 dB, depending primarily upon the amount of correction for pure tones.

The Tone Corrected Perceived Noise Level scale requires complex analysis and instrumentation to define a sound. Thus, it has not been utilized extensively, particularly since in most instances the simple A-weighted sound level appears to adequately describe environmental noise at a location, at a given time and does not require particularly complex instrumentation.

STATISTICAL MEASURES

One of the dominant characteristics of environmental noise at any location is that it fluctuates considerably from quiet at one instance to loud the next. Thus, noise at a location must be described by a statistical approach that takes time into account if it is to be accurately described. This can be achieved by giving the complete curve depicting the cumulative distribution of sound levels; that is, by showing what percent of the whole observation period each level is exceeded. Noise levels are often specified in terms of levels exceeded 10 percent of the time, 50 percent of the time, and 90 percent of the time.

The sound pressure level exceeded 10 percent of the time, expressed as L_{10} , gives an approximate measure of the higher level and short duration noise. A measure of the median sound level is given by the L_{50} and represents the level exceeded 50 percent of the time. The residual sound level is approximated by L_{90} , which is the sound level exceeded 90 percent of the time.

The Energy Mean Noise Level (L_{eq})

A measure accounting for both the duration and the magnitude of all the sounds occurring during a given period is the average sound level, sometimes called the equivalent continuous noise level. It is the continuous A-level that is equivalent in terms of noise energy content to the actual fluctuating noise existing at a location over the observation period. It is also called the Energy Mean Noise Level (L_{eq}). By definition, L_{eq} is the level of the steady state continuous noise having the same energy as the actual time-varying noise. In terms of assessing the effects of noise on humans L_{eq} is one of the most important measures of environmental noise, since there is experimental evidence that it accurately describes the onset and progression of hearing loss.^{3,7} There is also considerable evidence that it applies to human annoyance due to noise.¹⁴

The statistical measures described simplify the problem of quantifying environmental noise and are used extensively. These measures may, however, be misleading if used exclusively when comparing two environments differing with respect to how constant or stationary they are during the observation period.⁵

CUMULATIVE MEASURES

In most instances the noise problem is twofold. It involves either the constant high-level noise intrusion of the city or the intermittent single-event noise intrusions in residential areas. With the advent of jet aircraft, the latter type of problem has grown considerably over the years. Jet aircraft noise has contributed significantly to data on and insight into community annoyance and has stimulated the development of indices for assessing the cumulative effect of intrusive noises.

Rosenblith-Stevens Model

Rosenblith and Stevens¹⁵ developed, in the early 1950's, a model for relating the probable community reaction to intrusive aircraft noise. This model included seven factors that were corrected for.

1. Magnitude of the noise.
2. Duration of the intruding noise.
3. Time of the year (winter/summer; windows opened or closed).
4. Time of day (night/day).
5. Outdoor noise level when the intruding noise is not present.
6. History of prior exposure of the community to the intrusive noise.
7. Frequency components in the noise or its impulsive nature.

Other methods have been proposed. Most of these represent some modification of the basic model of Stevens and Rosenblith.

CNR and NEF

The Composite Noise Rating (CNR) was introduced in the early 1950's,¹⁶ followed by the Noise Exposure Forecast (NEF).¹⁷ The CNR and NEF are similar, except that NEF accounts for both duration and pure tone content of each single event, whereas CNR does not.

In the course of the studies relating to aircraft and airport noise, called for by the Noise Control Act of 1972, an effort has been made by Von Gierke and his staff to develop for EPA a suitable and simple method for defining and measuring cumulative noise exposure.¹⁹ This method utilizes a 24-hour average A-weighted sound level with a penalty of 10 dB applied to nighttime sound levels. This method, the day/night average sound level (L_{dn}) will be further discussed in Section 3.

Community Noise Equivalent Level

Recently, California introduced the Community Noise Equivalent Level (CNEL).¹⁸ This rating represents the average noise level determined for a 24-hour period, with different weighting factors for noise levels occurring during the day, evening, and night periods. Essentially, it is an L_{eq} for a 24-hour period with special corrections of 5 and 10 dB, respectively, for evening and nighttime. It is designed to account for the increased disturbance caused by noise events during the evening and the night.

To simplify the understanding of the cumulative methods described, a summary of the variables included in each is presented in Table 2-1.

While most of the developments described above were performed in the United States, Robinson, in England, developed a new scale, the Noise Pollution Level (L_{np}).^{20,21} This measure is derived from two terms, one involving the average sound level (L_{eq}) of the noise and one involving the magnitude of the time variation of the noise level. The L_{np} concept embodies some simple principles:

1. Other things being equal, the higher the noise level, the more the disturbance.
2. Other things being equal, the less steady the noise level, the greater its annoying quality.

In a more recent work, Robinson has further refined his Noise Pollution Level by taking the levels of variation of the sound pressure levels and their rate of change into account.²²

The preceding discussion by no means exhausts the list of various schemes devised in the ever-continuing efforts to develop new and better noise scales. It is intended to facilitate understanding of the following sections of this document.

Table 2-1

FACTORS CONSIDERED IN EACH OF FOUR METHODS
FOR DESCRIBING THE INTRUSIVENESS
OF NOISE ON THE COMMUNITY

FACTOR	COMPOSITE NOISE RATING CNR	NOISE EXPOSURE FORECAST	DAY/NIGHT AVERAGE SOUND LEVEL LDN	COMMUNITY NOISE EQUIVALENT LEVEL
Basic Measure	Maximum Perceived Noise Level	Tone Corrected Per- ceived Noise Level	A Weighted Noise Level	A Weighted Noise Level
Measure of Duration of Individual Single Event	None	Energy Integration	Energy Integration	Energy Integration
	day 7 a.m. - 10 p.m. night 10 p.m. - 7 a.m.			day 7 a.m. - 7 p.m. evening 7 p.m. - 10 p.m. night 10 p.m. - 7 a.m.
Weighting for Time Period	Day 0 dB Night 12 dB		Day 0 dB Night 10 dB	Day 0 dB Evening 5 dB Night 10 dB
Number (N) of Identical Events in Time Period	10 Log N		10 Log N	
Summation of Contributions	Logarithmic		Logarithmic	

SUMMARY—RATING SCHEMES FOR ENVIRONMENTAL COMMUNITY NOISE

The description of community noise must account for:

1. Those parameters of noise that have been shown to contribute to the effects of noise on man.
2. The variety of noises found in the environment.
3. The variations in noise levels that occur as a person moves through the environment.
4. The variations associated with the time of day.

Over the years, considerable effort has been expended to develop scales that reduce the dimensions of sound and perception into a one-number scheme. Much effort has been focused on combining measures of frequency content and overall level into a quantity proportional to the magnitude of sound as heard by a person. An example of this type of rating scheme is embodied in the sound level meter, although, other rating schemes are reviewed as well. Others have described noise by a statistical approach that takes time into account. This is done by giving the complete curve depicting the cumulative distribution of sound levels. Finally, schemes designed to assess the effects of the constant high-level noise intrusion or the intermittent single-event noise intrusion are also reviewed. It is found that to date one measure of noise that appears to be emerging as one of the most important measures of environmental noise in terms of the effects of noise on man is the Energy Mean Noise Level, L_{eg} , which by definition is the level of the steady state continuous noise having the same energy as the actual time-varying noise.

REFERENCES

1. Kryter, K.D., THE EFFECTS OF NOISE ON MAN, New York: Academic Press, 1970.
2. The National Bureau of Standards, Fundamentals of Noise Measurement, Rating Schemes and Standards, U.S. Environmental Protection Agency Document, *NTID 300.15*, 1971.
3. Chalupnick, J.D., (ed.), TRANSPORTATION NOISE, A SYMPOSIUM ON ACCEPTABILITY DATA, Seattle: University of Washington Press, 1970.
4. Young, R.W. Measurement of Noise Level and Exposure, in TRANSPORTATION NOISES, Seattle: University of Washington Press, 1970.
5. Wyle Laboratories, Community Noise, U.S. Environmental Protection Agency Document, *NTID 300.3*, 1971.
6. Stevens, S.S., Measurement of Loudness, *JASA*, 27, 815, 1955.
7. Stevens, S.S., On the Psychophysical Law, *Psychol. Review*, 64, 153, 1957.
8. Stevens, S.S., Concerning the Form of Loudness Function, *JASA*, 29, 603, 1957.
9. Fletcher, H. and Munson, W.A., Loudness Definition, Measurement and Calculation, *JASA*, 5, 84, 1933.
10. Kryter, K.D., Scaling Human Reactions to the Sound from Aircraft, *JASA*, 31, 1415, 1959.
11. Kryter, K.D. and Pearsons, K.S., Judged Noisiness of a Band of Random Noise Containing an Audible Pure Tone, *JASA*, 38, 1965.
12. Federal Aviation Regulations, Part 36, Noise Standards: Aircraft Type Certification, November 1969.
13. Robinson, D.W. and Cook, J.P., NPL Aero Report No. Ac31, June 1968, National Physical Laboratory, England.
14. Meister, F.J., The Influence of the Effective Duration in Acoustic Excitation of the Ear, *Larmbekämpfung*, 10, June/August 1966.
15. Rosenbl th, W.A., Sevens, K.N., and the Staff of Bolt, Beranek and Newman, Inc., *HAND-BOOK OF ACOUSTIC NOISE CONTROL*, vol. 2, Noise and Man, WADC TR-52-204, Wright Patterson Air Force Base, Ohio, 1953.
16. Galloway, W.J. and Pietrasanta, A.C., Land Use Planning Relating to Aircraft Noise, Technical Report No. 821, Bolt, Beranek and Newman, Inc., Published by FAA, October 1964.
17. Galloway, W.J. and Bishop, D.E., Noise Exposure Forecasts: Evolution, Evaluation, Extensions and Land Use Interpretations, FAA-NO-70-9, August 1970.

18. The Adopted Noise Regulations for California Airports: *Title H*, Register 70, No. 48-11-28-70, Subchapter 6, Noise Standards.
19. Von Gierke, H., Impact Characterization of Noise Including Implications of Identifying and Achieving Levels of Cumulative Noise Exposure, Draft Report, Task Group 3, 1973.
20. Robinson, D.W., The Concept of Noise Pollution Level, National Physical Laboratory, Aerodynamics Division, *NPL Aero Report Ac 38*, March 1969.
21. Robinson, D.W., Towards a Unified System of Noise Assessment, *J. of Sound and Vibration*, 14, 279, 1971.
22. Robinson, D.W., Rating the Total Noise Environment, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, May 1973.

SECTION 3

ANNOYANCE AND COMMUNITY RESPONSE

Annoyance as a result of exposure to noise is a psychosocial response to an auditory experience. Annoyance has its roots in the unpleasantness of noise, in the disruption by noise of ongoing activities, and/or in the meaning or messages carried by a given noise.

The degree of annoyance and whether that annoyance leads to complaints or produces rejection of or action against a noise source are dependent upon many factors to be discussed subsequently. Some of these factors are well understood, others are not.

MEASUREMENT TECHNIQUES FOR ANNOYANCE

Numerous techniques have been devised to measure annoyance, from a simple scale ranging from not annoyed to highly annoyed to very complicated techniques involving social surveys.

Individual Response

Individual responses of people to noise are often studied in the laboratory. Usually, these studies involve judgments of individual noise events in controlled environments. Such studies have been helpful in isolating some of the factors contributing to annoyance by noise. The annoyance factors include:

- The intensity level and spectral characteristics of the noise.
- The duration of the noise event.
- The presence of discrete frequency components.
- The presence of impulses.
- The abruptness of onset or cessation of the noise event.
- Degree of harshness or roughness of the noise.
- Degree of intermittency in either the loudness.
- The pitch or rhythm.
- The information content and the degree of interference with activity.^{1, 2}

Earlier Social Surveys

Community annoyance by noise is usually studied through social surveys. These surveys have revealed other variables that are important in eliciting annoyance. Such variables include:

1. The noise climate or background noise against which a particular noise event, such as aircraft flyover, occurs.

2. The previous experience of the community with the particular noise.
3. The time of day during which the intruding noise occurs.
4. Attitude of people towards the noise makers.
5. Socioeconomic status of the community.

A number of experimental investigations have been made since the early 1950's that have attempted to determine how people are affected by the noises they are exposed to and how to arrive at methodologies that allow predictions of their response from measurements of the physical characteristics of noise. Most of these studies have been in the form of social surveys and have included studies in the United Kingdom,^{3, 5} Sweden,^{6, 11} Austria,^{12, 14} France,^{15, 17} the Netherlands¹⁸ and the United States.^{19, 21}

The social surveys led to a series of noise ratings discussed in Section 2. Most of the ratings thus devised were primarily based on investigations of aircraft and traffic noise.²² While there was coordination between the various researchers involved in social surveys, less coordination existed among those involved with the measurement of various environmental noises studied. As a result, a variety of methods were utilized for measuring and reporting the noise exposures experienced by the survey respondents. Nevertheless, a number of consistent findings emerged. These findings are:

1. Even though each rating was developed independently, there exists a high degree of correlation among all ratings, of the order of 0.90.²³ Further, the community response criteria derived from these surveys are remarkably similar for a specified noise exposure.²⁴
2. The relationship between the statistical average annoyance experienced by a collection of individuals (a community) and the degree of noise exposure experienced is also highly correlated as shown by Alexandre.²⁵ This is depicted in Figure 3-1, which shows the correlation between degree of noise exposure and average annoyance for five surveys.
3. The individual annoyance response of a person living within a community is not predicted as accurately as that of the community as a whole. This is reflected in the poor correlation (correlations under 0.5) that exist between noise ratings and individual annoyance scores. This particular finding stems from the fact that there are a number of psychological and social factors that contribute to the large range in individual sensitivity to annoyance from noise.²⁶

Recent Social Surveys

Some of the criticisms generated by the earlier social surveys of the 1950's and early 1960's have resulted in new surveys. These new surveys have extended the range of noise sources

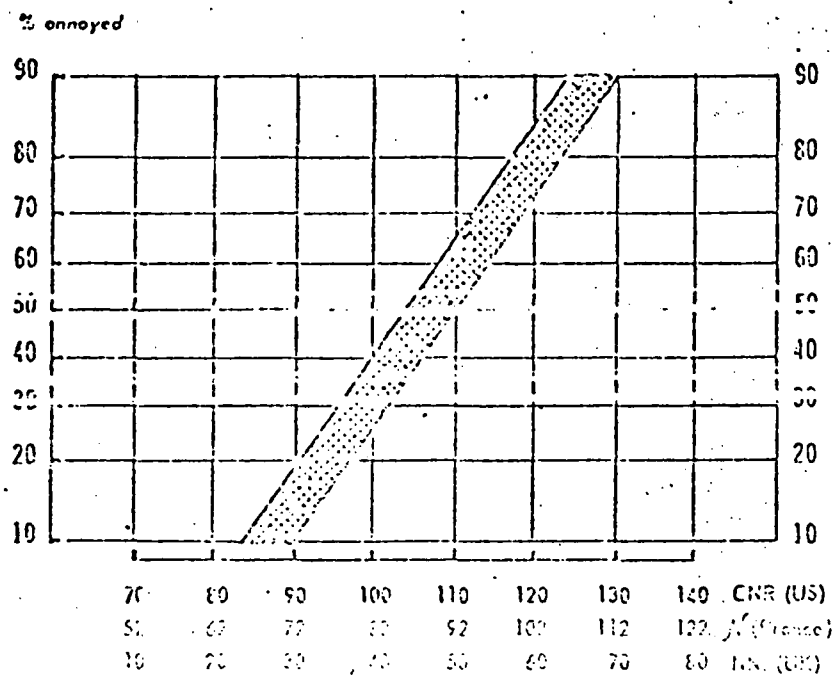


Figure 3-1. Percentage of Highly Annoyed Persons in Relation to Noise

considered, the noise levels, the mix of noise sources and have included additional questions related to personal factors into the questionnaire administered to people.^{27, 34} By and large, the new surveys have confirmed the findings relative to population average obtained in the previous surveys and have increased the correlation between individual annoyance scores and noise ratings. In the studies performed by Tractor, for example, it has been shown that the correlation between individual annoyance scores and noise rating is increased, when personal variables are included in the calculation of annoyance, from 0.37 to 0.79.²⁹

Further, the new series of surveys have shed considerable light on the nature of some of the personal factors that contribute to a person's reaction to noise. Some of these factors include:

1. Fear associated with activities of noise sources such as fear of crashes in the case of aircraft noise.
2. Socioeconomic status and educational level.
3. The extent to which residents of a community believe that they are being treated fairly.
4. Attitude of the community residents regarding the contribution of the activities associated with the noise source to the general well-being of the community.
5. The extent to which residents of the community believe the noise source could be controlled.

COMMUNITY RESPONSE

Another important aspect of community noise that has not been discussed has to do with what the community does about noise or sources. Much of what we know about this aspect of community reaction to noise comes from studies of complaints from individuals living around airports.

Complaints

Actions against a noise source may take various forms, ranging from registration of a complaint through a telephone call or a letter to the person or authority responsible for the operation of the noise source, to actual court action.

In general, people who complain do not appear to be unusual, neither are they particularly sensitive to noise.²⁵ Complaints have been found to be only a partial indicator of the number of persons annoyed in a community. In fact, complaints may represent only a fraction of those annoyed (2 to 20 percent).²⁹ This finding is shown in Table 3-1.

The Rating Scheme

A different approach for the assessment of the response of a community to noise was

TABLE 3-1

PERCENTAGES OF PERSONS HIGHLY ANNOYED WHO REGISTER COMPLAINTS AS A
FUNCTION OF L_{dn} .

L_{dn}	Percentage of Highly Annoyed	Percentage of Complaints
50	13	Less than 1
55	17	1
60	23	2
65	33	5
70	44	10
75	54	15
80	62	Over 20

utilized in pioneering work by Stevens, Rosenblith and Bolt, which culminated in the Community Noise Rating Scheme referred to in Section 2.^{36, 37} This rating method was based on an heuristic assessment of 1) the acoustical parameters thought to influence community response and, 2) the correlation between these effects and actual case histories of overt community action in response to noise. In this approach, specific overt responses are observed, and then inferences are drawn about community annoyance. In other words, there is no attempt to actually measure annoyance. Community responses mean a scale of complaints by citizens ranging from sporadic to actual law suits against the noise makers.

The Borsky Social Survey

In the 1950's Borsky began an extensive community noise social survey in response to criticism directed to the rating method developed by Stevens et al.³⁸ One of the initial survey results, as has been corroborated in subsequent surveys, showed that overt reaction by a community, as measured on a complaint type of scale, is clearly an underestimate of the degree of annoyance existing in a community. This finding is consistent with the finding that even at very low noise exposures, about 10 to 15 percent of the population will still display a high degree of annoyance even though no complaints may be registered.

An obvious step in the study of community response to noise was to compare the social survey results on the relationship between annoyance and noise exposure. This comparison showed that criteria for acceptable noise exposures based on annoyance data essentially agree with criteria based on community reaction observations.^{1,24} From these findings, it is inferred that the variability in the relationship of community reaction to a specified noise exposure is explainable by the variability in individual susceptibility to noise as compared with group averages. This hypothesis is clearly in need of further study, but the aggregated data show clearly that the envelopes of variability are highly correlatable, whatever the causal relationships.

Analysis of Studies

One of the real problems in evaluating the general relationship between noise exposure and community response is the fact that most of the data on which these relationships are based are primarily related to aircraft noise exposures. This problem is somewhat lessened by the results of several different analyses. First, the case studies used in developing the CNR system covered a wide range of noise exposures from transportation to industrial noise sources. The high correlation between these results and those from the airport related surveys, and the relationship between annoyance and noise exposure lead to the assumption that for the average response of the community, annoyance and community reaction to noise exposure can be predicted independently of the nature of the noise source. Second, the social surveys related to noise sources other than aircraft provide essentially identical relationships between annoyance and noise exposure as those found in the airport studies.^{30, 31, 34}

The highly convergent trend of the various investigations of annoyance and community response leads to the following conclusions:

1. The degree of annoyance due to noise exposure expressed by the population average for a community is highly correlated to the magnitude of noise exposure in the community.²⁵
2. Variations in individual annoyance or response, relative to the community average, are related to individual susceptibilities to noise; and these are highly correlated with definable personal attitudes about noise.^{26, 38, 39}
3. The numbers of complaints about noise registered with the authorities is small compared to the number of people annoyed, or who wish to complain. However, the number of actual complaints is highly correlated with the proportion of people in the community who express high annoyance.²⁹
4. The high correlation between those noise rating methods that account for the physical properties of noise exposure over a day's time suggests that the simplest acoustical measure that accounts for sound magnitude, frequency distribution, and temporal characteristics of sound over 24 hours is an adequate measure for noise exposure in communities.

The preceding factors were taken into account by the members of the Task Force of the EPA Airport/Aircraft Noise Study in their assessment of the impact of cumulative noise exposure on annoyance. Their conclusion was that the "energy" equivalent, or average, A-weighted sound level, taken over a 24 hour period, with a 10-decibel penalty applied to nighttime sound levels, is the simplest noise measure that provides high correlation with annoyance, complaint behavior, and overt community reaction.⁴⁰ This measure was named "day-night average sound level." A summary of the relationship between this measure and the various responses to noise exposure is shown shown in Figure 3-2, taken from the Task Group report.

SUMMARY – ANNOYANCE AND COMMUNITY RESPONSE

Numerous techniques have been devised to measure annoyance, from a simple scale of annoyance level to complicated techniques involving social surveys. Laboratory studies of individual response to noise have helped isolate a number of the factors contributing to annoyance, such as the intensity level and spectral characteristics of the noise, duration, the presence of impulses, pitch, information content, and the degree of interference with activity.

Social surveys have revealed several factors related to the level of community annoyance. Some of these factors include:

1. Fear associated with activities of noise sources such as fear of crashes in the case of aircraft noise.

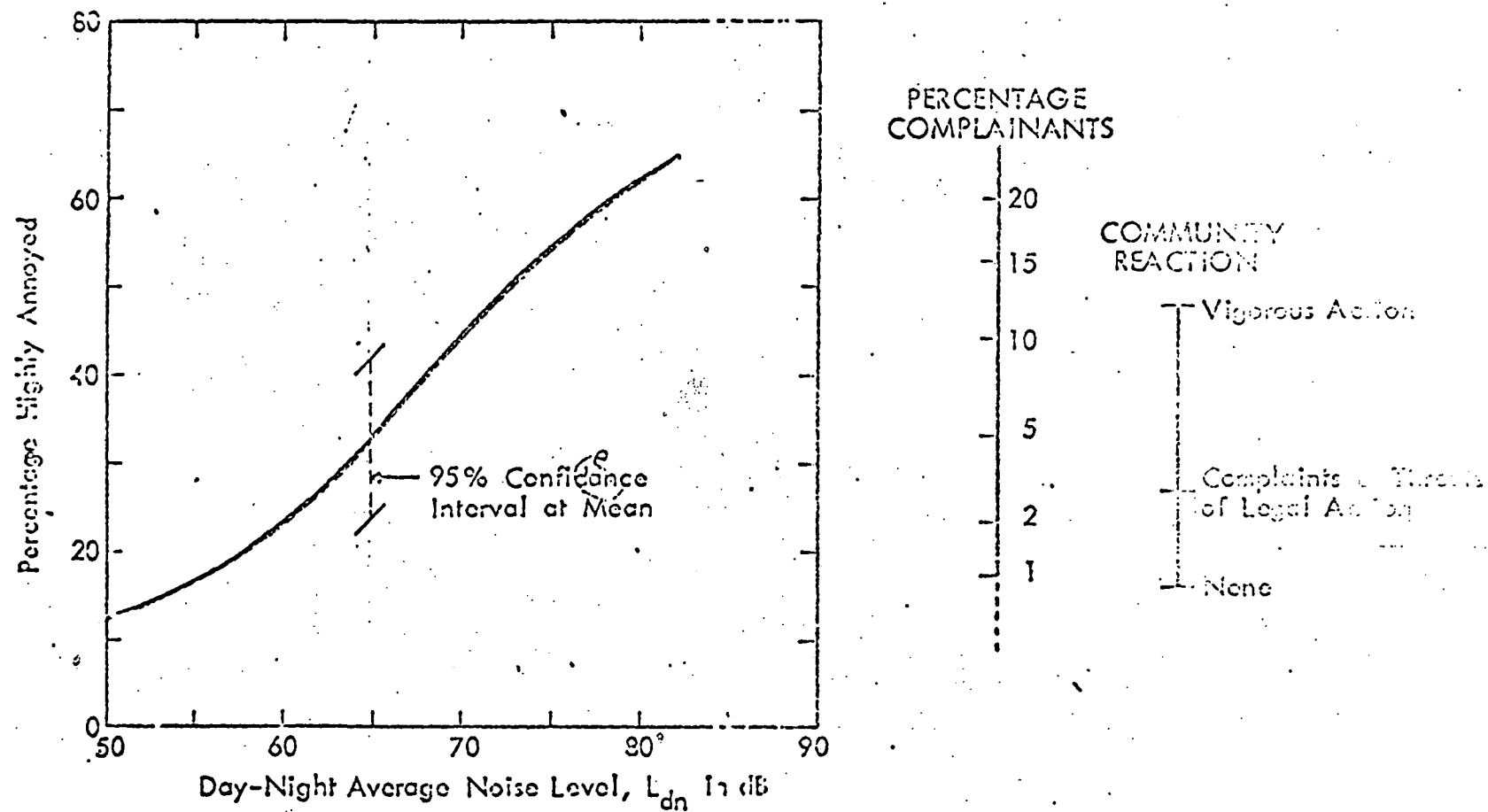


Figure 3-2. Intercomparison of Various Measures of Individual Annoyance and Community Reaction as a Function of the Day-Night Average Noise Level

2. Socioeconomic status and educational level.
3. The extent to which community residents believe that they are being treated fairly.
4. Attitude of the community's residents regarding the contribution of the activities
 - associated with the noise source to the general well-being of the community.
5. The extent to which residents of the community believe that the noise source could be controlled.

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REFERENCES

1. Galloway, W.J., Predicting Community Response to Noise from Laboratory Data, Transportation Noises, A Symposium on Acceptability Data, Chalupnik, J.D., ed., University of Washington Press, 269-291, Seattle: 1970.
2. Kryter, K.D., THE EFFECTS OF NOISE ON MAN, Academic Press, New York: 1970.
3. Noise, Final Report, prepared by the Committee on the Problem of Noise and presented to Parliament in July, 1963, Her Majesty's Stationary Office, London, England, Cmnd. 2056.
4. Parkin, P.H., Purkis, H.S., Stephenson, R.V. and Schlaffenberg, B., London Noise Survey, Building Research Station Report, S.O. Code No. 67-266, London, England, Her Majesty's Stationary Office 1968.
5. Purkis, H.J., London Noise Survey: A survey of Noise levels in London, Note No. 6/66, March 1966, Building Research Station, Garston, Herts., England.
6. Cedarlof, R., Jonsson, E. and Kajlaud, A., Annoyance Reactions to Noise from Motor Vehicles: An Experimental Study *Acustica*, 13, 270-279, 1963.
7. Kihlman, Tor, Björn Lundquist and Bertil Nordlund, Trafikbullerstudier, *Report 38: 1968*, Statous Institute for Byggnadsforskning, Stockholm, Sweden.
8. Fog, H., Jonsson, E., Kajlaud, A., Nilson, A. and Sorensen, S. Traffic Noise in Residential Areas, Statous Institute for Byggnadsforskning, Stockholm, 1968.
9. Jonsson, E., Erlaud, A., Kaylaud, A., Paccagnells, B. and Sorensen, S., Annoyance Reactions to Traffic Noise in Italy and Sweden, Stockholm: National Institute of Public Health, Karolinska Institute, University of Stockholm and University of Farrara, Italy.
10. Jonsson, E., Sorensen, S., On the Influence of Altitudes to the Source on Annoyance Reactions to Noise—An Experimental Study, *Nordisk Hygienisk Tidskrift*, SL VIII, 35-45, 1967.
11. Cederlof, R., Jonsson, E., Sorensen, S., On the Influence of Altitudes to the Source on Annoyance Reactions to Noise: A Field Experiment, *Nordisk Hygienisk Tidskrift*, XLVIII, 46-59 Stockholm, Sweden, 1967.
12. Bruckmayer, F., and Laug, J., Disturbance of the Population by Traffic Noise, *Oostereiche Ingenieurfeitschrift*, Jg. 1967, H.8, 302-206, H.9, 338-344, and H.10, 376-385.
13. Bruckmayer, F., Laug, J., Disturbance Due to Traffic Noise In Schoolrooms, *Oosterreichische Ingenicur-Feitschrift*, 11, 73-77, 1968.
14. Bruckmayer, F., Judgement of Noise Annoyance by Reference to the Background Level, *Oosterreichische Ingenicur-Feitschrift*, Jg. 1963, 315.
15. Auzou, S., Lamura, C., Le Bruet aux Abords des Autoroutes, *Cahiers du Centre Scientifique et Technique du Batement*, 78, 669, 1966.
16. Lamure, C., Auzou, S., les Niveaux de Bruet au Voisinage des Autoroutes Degages, *Cahiers du Centre Scientifique et Technique du Batement*, 71, 599, 1964.

17. Lamure, C. and Bacelon, M., La Gene due au Bruit de la Circulation Automobile, *Cahiers du Centre Scientifique et Technique du Batiment*, 88, 762, 1967.
18. Urban Traffic Noise, Status of Research and Legislation in Different Countries, Draft Report of the Consultative group on Transportation Research, DAS/CSI/68.47 Revised, OCED, Paris, France, 1969.
19. Waters, D.M. and Bottom, C.G., The Influence of Background Noise on Disturbance Due to Aircraft, Seventh International Congress on Acoustics, Budapest, 1971.
20. Robinson, D.W., The Concept of Noise Pollution Level, National Physical Laboratory Aero Report Ac 38, Teddington, England, 1969.
21. Seuko, Alexandre, A. and Krishnau, P.V., Urban Noise Survey Methodology, in two volumes, Report No. H-1262. Goodfriend and Associates, 1971.
22. Schultz, T.J., Technical Background for Noise Abatement in HUD's Operating Programs, HUD Report No. 2005 R, Washington, D.C., 1971.
23. Galloway, W.J. and Von Gierke, H.E., Individual and Community Reaction to Aircraft Noise; Present Status and Standardization Efforts, paper presented at the London Conference on Reduction of Noise and Disturbance Caused by Civil Aircraft, *Paper INC/C4/pg*, Nov. 1966.
24. Galloway, W.V. and Bishop, D.E., Noise Exposure Forecasts: Evolution, Evaluation, Extensions and Loud Use Interpretation, FAA Rpt No 70-9, 1970.
25. Alexandre, A., Aircraft Noise Annoyance in Europe: Special and Temporal Comparisons, resulted at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
26. McKennel, A.C., Noise Complaints and Community Action in Transportation Noise, A Symposium on Acceptability Data, Chalupnik, J.D., ed., 228-244, University of Washington Press, Seattle, 1970.
27. Aubree, D., Auzou, S. and Rapise, J.M. Etud de la Gene due au traffic Automobile Urbaise, Final report of contract D. G, R.S.T. No. 68-01-389. Centre Scientifique and Technique du Batiment, Paris, 1971.
28. Auzou, S. and Rapice, J.M., de briect du a la Circulation Automobile su Site Urbaiu: Ses Caracteristiques Physiques et sa Prevision, Centre Scientifique et Technique du Batiment Etablissement de Grenoble, Division Acoustique, 1971.
29. Tracor, Community reaction to airport Noise *NASA report CR-1761*, 1971.
30. Second Survey of Aircraft Noise Annoyance around London, (Heathrow), Airport, H.M.S.O. London, 1971.
31. Anderson, C.M.B., the Measurement of Altitude to Noise and Noises, National Physical Laboratory Acoustics, Rpt. AC 52, Teddington, England, 1971.

32. A Study of Annoyance from Motor Vehicle Noise, BB&N, Inc., *Rpt No. 2112*, 1971.
33. Motor Vehicle Noise: Identification and Analysis of Situations Contributing to Annoyance, BB&N, Inc., Rpt. No. 2082, 1971.
34. Delauy, M.E., Copeland, W.C., Payne, R.C., Propagation of Traffic Noise in Typical Urban Situations, National Physical Laboratory Acoustics *Rpt A, 54*, 1971, England.
35. Report to the President and Congress on Noise by the U.S. Environmental Protection Agency, Document No. 92-63, Washington, D.C., 1972.
36. Rosenblith, W.A., Stevens, K.N., and the Staff of Bolt Baranck and Newman Inc., Handbook of Acoustic Noise Control, vol 2, Noise and Man, WADC TR-52-204, Wright Patterson Air Force Base, Ohio, 1953.
37. Stevens, K.N., Rosenblith, W.A. and Bolt, R.M., A Community's Reaction to Noise: Can it be Forecast, *Noise Control, 1*, 63 71, 1955.
38. Borsky, P.N. Community Reaction to Air Force Noise, WADD Technical Rpt. No. 60-689, Pts 1 and 2, Wright Patterson Air Force Base, Ohio, March 1961.
39. Bolt, Baranck and Newman, Motor Vehicle Noise: Identification and Analysis of Situations Contributing to Annoyance, prepared for the Automobile Manufacturers Association Inc. BB&N Rpt. No. 2082.
40. Von Gierke, H.E. Draft Report on Impact Characterization of Noise Including Implications of Identifying and Achieving Levels of Cumulative Noise Exposure, prepared for U.S. Environmental Protection Agency, June 1973.

SECTION 4

NORMAL AUDITORY FUNCTION

Besides being sensitive to an enormous range of acoustic pressure variations, the ear is capable of precise discriminations of temporal, intensity, and frequency changes. Hearing is probably the most critical learning sense in childhood and continues in adulthood as the most frequently used sense for the communication of ideas.

Associated with the auditory portion of the ear is the sense of balance. Although not specifically a part of auditory function, disorders in the vestibular region of the ear can adversely affect the operation of the auditory sensor and vice versa.

NORMAL HEARING IN YOUNG POPULATIONS

Hearing normally means being able to detect sounds in the audio-frequency range, namely, 16 to 20,000 Hz (20 kHz), at levels that lie at or within 10 decibels of the normal threshold of hearing and below the threshold of aural pain in human beings (those boundaries define the domain of normally audible sounds heard by air conduction.) The human hearing process is such that at frequencies from 1,000 Hz down to 16 Hz, it takes increasingly more acoustical energy to produce the same sensation of hearing as at the 1,000 Hz level. Similar increases also are required with regard to the frequencies from 1,000 to 10,000 Hz but at a lower order of magnitude.

Many otologists define normal hearing more narrowly as the ability to respond appropriately to human speech (the spectral components of which are contained largely in the range 250 to 4000 Hz) in average everyday conditions: others dispute so restrictive a definition, however. When referred to in this document, hearing level is generally presumed to be determined by pure-tone audiometry using standardized instrumentation and procedures.

The entire audio-frequency range just defined may be considered to be the domain of human hearing. The appreciation, by nonauditory sensations in the ear or otherwise, of air- or structure-borne vibrations at frequencies lower (infrasonics) or higher (ultrasonics) than the audio-frequency range is not a part of hearing.

As to the boundaries of the domain of hearing, there is no evidence that these vary significantly between normal human populations around the world. The normal threshold of hearing for pure tones and the corresponding reference zero for audiometers have received

international standardization (ISO, 1961, 1964), which may be taken to apply to the American population. The upper boundary of normally audible sound (threshold of aural pain) has not yet received such definitive recognition, but is commonly deemed to lie in the region of 135 dB SPL, a value that is largely independent of frequency.¹

It is of interest to note that the typical average level of conversational speech without undue vocal effort, measured at a customary speaking distance of 1 meter from the speaker, is about 65 dB SPL. Peak intensities of vocal sounds usually exceed the average level by about 6 dB. A range variation of some 20 dB about the average is to be expected in the normal speech levels of different speakers.

HEARING STUDIES AND RESULTS

Approximately 5 percent of school age children in the USA had deficient hearing, according to a survey by Kodman and Sperrezzo in 1959.² A similar incidence has been reported in Lebanon by Mikaehan and Barsorimian.³ There is no evidence that any significant fraction of this hearing loss in American children below working age is noise-induced. Rosen and Rosen⁴ have published a comparative survey of the upper limits of hearing in school-age children and young people (aged 10 to 19 years) in several countries in Africa, Europe and North America. That survey suggests that the frequency range of "normal" hearing in that age group extends to at least 16 kHz (at which frequency, using a special audiometric technique, the authors obtained nearly 100 percent response in some of the groups), but that the percentage of children responding (able to detect tones) falls off rapidly at higher frequencies. A response incidence of less than 50 percent was obtained from all but one of the nine test groups at 20 kHz. However, responses in the range 0 to 15 percent were obtained at 22 kHz; and responses greater than zero (up to 10 percent in Maba'an youngsters) in 4 groups even at 24 kHz. Fewer than 4 percent of a group of American (New York) children responded at that frequency.

Rosen and his co-workers have tentatively suggested that the differences in hearing level of children of different cultures may be linked with differences in susceptibility to atherosclerosis and coronary artery disease in later life. Rosen, Olin and Rosen,⁵ citing work in Finland as well as their own studies, have also contended that a low saturated fat diet, said to protect against coronary artery disease, may also protect against sensorineural hearing loss.

Using a bone-conduction ultrasonic transducer in selected young adults (17 to 24 years of age), Corso⁶ also found that some hearing sensation exists above 20,000 Hz, above which frequency there is a fairly abrupt decrease in steepness of the threshold slope (which is steep—about 50 dB/octave—between 14 and 20 kHz). Corso found that some sensation persisted on bone conduction testing at high levels of stimulation at ultrasonic frequencies up to more than 90 kHz, but it is very questionable whether this can be regarded as part of “hearing.” There was little difference between the sexes in either sensitivity or range of sensation.

AUDIOLOGICAL UNIFORMITY OF THE POPULATION.

There is no inherent difference between the races comprising the population of the United States with regard to hearing levels as a function of either age or noise exposure. Human ears are much the same around the world. Public hearing surveys may, however, reveal demographic differences in hearing levels of adults of different races or social groups.⁷ Such differences may be attributed to the effect of differing environmental influences, including non-occupational noise exposure (sociacusis).

Surveys of hearing levels in general populations can yield values that are poorer (less sensitive hearing) than those obtained from samples, ostensibly from similar populations, from whom subjects with certain audiological abnormalities (sometimes arbitrarily selected) have been weeded out by a selection procedure.

SOURCES OF VARIATION IN HEARING LEVELS

Apart from the question of changes in hearing with advancing age, individual, and other factors, it is to be expected that some statistical variation in threshold will be seen even when a particular ear is audiometrically retested. The variation arises partly from intrinsic sources (e.g., changes in the subject's physiological state) but a substantial source of variation in practice is imperfection in the way in which audiometry is conducted (this is discussed in detail in the section on Audiometry found in a recent EPA/AMRL publication⁸). Test-retest variance can, however, be kept to a minimum when serial audiograms are obtained in accordance with standard procedures, carried out under properly controlled conditions.

Individual Variation.

Hearing surveys are always subject to possible bias because of the difficulties of sampling human populations. In voluntary public hearing surveys, for example, a substantial proportion of people selected to form a supposedly random sample of the adult American population may decline to be examined. One cannot know, in that event, whether or not those who will not be examined have group hearing levels similar to those who do participate. If, for any reason, those refusing do have different hearing as a group, then the survey cannot truly reflect the state of hearing of the population sampled. More reliable data are of course obtainable from "captive" (e.g., industrial or military) populations, of whom every member can perforce be examined;⁹ but such populations do not represent the general population.

Sex Related Variations

From the early teenage years onwards, and particularly in the age range 25 through 65 years, women in industrial countries, including the United States, generally have better hearing than men. In the elderly, however, above age 75, the difference tends to become insignificant. Paradoxically, the rate of increase in hearing loss in men over 50 years of age declines, while increasing in women of the same age. Female employees have been found to have better hearing than male employees, even when they work side by side in noisy industries.^{10-12.} Selection processes and circumstantial factors have been postulated to account for this. These factors included thoughts that the women were exposed less to non-occupational sociocoustic influences, such as small-arms noise; that they showed a high absentee rate—a questionable contention and that they are freer to leave a job in which they find the noise level objectionable). A more reasonable explanation, however, may be that, in the industries involved, women may benefit from more liberal and frequent rest periods than are allotted to men.¹³ The decline in differentiation between the hearing of the two sexes in old age may be linked with an enhanced aging effect upon the ear associated with post-menopausal changes in women,¹⁴ although this is admittedly speculative.

AUDITORY PRESENTATION OF INFORMATION

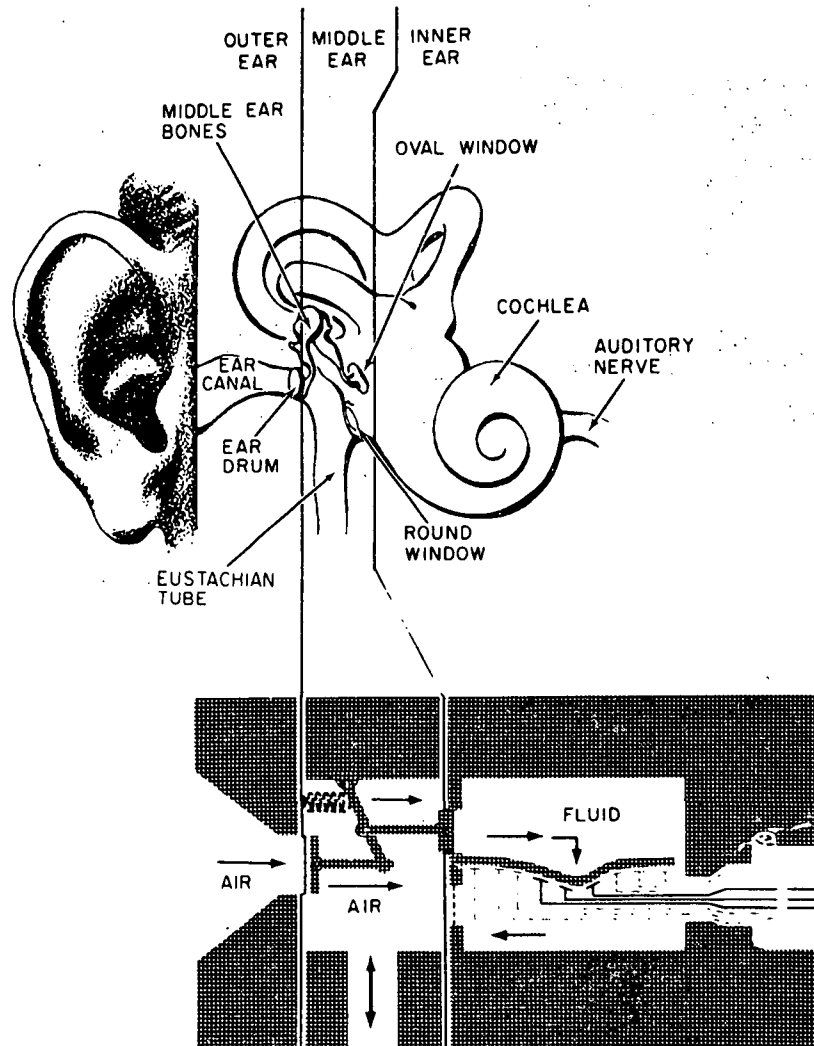


Figure 4-1 Functional Diagram Of Ear

Cited from "Human Engineering Guide to Equipment Decision" Editors - Morgan, Cook, Chapanis, Lund; McGraw Hill, 1963.

The Effect of Noise Stimulation on Mediating Mechanisms in the Middle Ear (Middle Ear Muscle Reflex).

In the normal auditory mechanism, sound is transmitted to the inner portion of the ear when sound vibrations imparted to the eardrum are mechanically transported across the middle ear via three tiny bones, the malleus, incus and stapes (the ossicular chain). See Figure 4-1.

Then, the inner most bone rocks in and out of its location, transferring the vibrations to the fluid-filled inner ear region. Attached to the outer and inner bones are the two smallest muscles in the body. The tensor tympani muscle, attached to the handle of the malleus, serves to pull the eardrum inward (toward the center of the head) when the muscle is contracted. The smaller of the two muscles, the stapedius, is located on the back portion of the floor in the middle ear and attaches to the head region of the stapes. Upon contracting, the muscle pulls the stapes in a lateral direction causing the eardrum to be moved outward. In effect, the two muscles work in opposition to each other. Therefore, if they both contract at the same time, there is a tightening of the ossicular chain into a comparatively rigid condition. The effect of this tensing of the conductive mechanism is to reduce the amount of sound energy delivered to the cochlea and thereby protect the inner ear from high intensity sound.

Contraction of the stapedius muscle is caused by high level sound. A bilateral neurological reflex arc has been described in which sound arriving at the cochlea is converted to neurological impulses and carried toward the higher brain centers by the nerve of hearing, Cranial Nerve VIII.¹⁵ If the neurological activity is sufficiently intense, stimulation of descending neurologic pathways of the facial nerve, Cranial Nerve VII, occurs. This set of nerve fibers serves many areas of the head, including the stapedius muscle. Thus, sound stimulation can result in the contraction of the stapedius muscle.

The middle ear muscle reflex, a popular name for the above-described activity, increases and decreases in muscle tension according to the amplitude of the auditory stimulus that sets off the reflex. According to Reger et al.,¹⁶ the shift in transmission efficiency results in a conductive loss of as much as 35 dB in the lower audiometric frequencies (250 Hz) but there is little loss in conductive capability for frequencies at 2000 Hz and above. This would indicate that there is relatively minor protective capability by the muscles for a significant portion of the frequency range at which the ear is maximally sensitive.

Indirectly, Ward¹⁷ observed that temporary threshold shift for a 700 Hz pure tone was reduced when masking noise of sufficient intensity to elicit a muscle reflex was introduced to the opposite ear from the one receiving the tone. The reduction in temporary hearing loss was of the same magnitude as one would find if the pure tone stimulus were approximately 10 dB lower in amplitude. Therefore, it might be concluded that for the frequency tested, there was a degree of protection afforded by the reflex. When Ward used a 2000 Hz tone, there was no apparent protection function in that the temporary threshold shift was the same with or without the reflex. In electrophysiological studies, Wever, et al.,¹⁸ found that the contraction of the stapedius muscle in cats resulted in 5.6 dB less transmission of a 300-Hz signal to the cochlea. The tensor tympani muscle contracting alone reduced the transmission efficiency 1.5 dB. When both muscles were contracted simultaneously, the resulting transmission loss was found to be 20 dB.

There is no firm agreement in the literature on the threshold of middle ear reflex activity for "normal" human ears. Perlman¹⁹ observed that reflex thresholds have been reported for sounds ranging from 40 dB to 100 dB depending upon the type of sound used. Thus, there appears to be a wide range of individual variation with respect to the reflex. In general, however, the reflex occurs when the stimulus is presented at levels between 75 to 90 dB. Perlman¹⁹ has also observed that during continuous stimulation by sound, the muscles tend to relax. This reduces their protective function.

The onset of muscle responses lags behind the onset of an intense sound by 15 to 17 milliseconds or longer.²⁰ The muscles reach peak contraction somewhat later. Wersall²¹ determined that these peaks occur 6 msec after onset of the stimulus for the stapedius muscle and 132 msec for the tensor tympani. This being the case, sounds of sudden onset and of short duration (e.g., gunshots, cap pistols, firecrackers, or stamping presses) are carried into the ear at full force without alteration by the middle ear muscles. It is thereby considered that the protective function of middle ear muscles for impulse-type sounds is nonexistent. Fletcher²² has demonstrated that some protection against noise can be obtained by introducing a moderate reflex-arousing stimulus prior to the occurrence of the more intense impulse noise. In industry, this principle has been applied by constructing a triggering device that presents a reflex-arousing tone to the ear of a drop forge operator prior to the impact of the forge itself. That this provides protection for the cochlea was dramatically demonstrated in animal experiments by Simmons.²³

He subjected one group of cats to gunfire without using a reflex-arousing stimulus immediately before each report of the gun. Histologic evidence was obtained that showed a marked difference in cochlear tissues of the cats receiving the reflex-arousing stimulus.

A possible additional mediating factor in the onset and extent of the reflex is the amount of attention one pays to the sound itself. Durant and Shallop²⁴ distracted subjects by diverting their attention with a mathematical mental task. Their conclusion was that the protective function of the middle ear muscles may be influenced by central factors, specifically, the state of attention.

HEARING LOSS ASSOCIATED WITH OLD AGE

The threshold of hearing rises, that is, hearing becomes less sensitive with advancing years, even in the absence of damaging noise exposure. This effect (presbycusis) involves primarily, and is most marked at, the higher audiometric frequencies, above about 3000 Hz.²⁵ At least in urbanized western populations, presbycusis appears to be more pronounced, at a given age, in men than in women, but the difference may be associated with occupational factors and the differences between the sexes in the pattern of day to day activity involving noise exposure, rather than with the sex difference *per se*.

Causes of Presbycusis

The loss of auditory sensitivity with advancing age is believed to be due to central nervous system deterioration as well as to peripheral changes in the auditory system.^{26,27} Aging people are apt to have increasing difficulty in discriminating auditory signals and in understanding speech heard against a background of noise. This may be due to an increasing susceptibility to masking by low-frequency (below 500 Hz) noise as well as to the loss of auditory sensitivity in the speech frequency range.

As Hinchcliffe²⁸ has remarked in a recent review, physiological aging is accompanied by degenerative changes affecting not merely the organ of Corti but the whole auditory system, including its central projections. This may explain some of the features of hearing handicaps typical of old age, such as loss of discrimination of normal, distorted and noise-masked speech, which are not amenable to prediction from pure tone audiometry alone. Rosen²⁹ believes that degenerative arterial disease in particular is a major factor in the etiology of presbycusis. Such changes affect individuals diffusely in different ways and

do not necessarily involve the Organ of Corti itself. To a marked degree, lesions of that organ due to noise are characteristically located discretely in the basal turn of the cochlea.

Glorig and Nixon³⁰ have restricted the definition of the term "presbycusis" to hearing losses caused by physiological aging, and it is used in this sense in this document, although some audiologists use it to embrace any sensorineural loss occurring in the elderly.

Presbycusis Corrections

Sufficient data now exists from surveys of general populations to permit estimations of average hearing loss due to presbycusis. These average hearing loss values due to aging are referred to as presbycusis corrections.

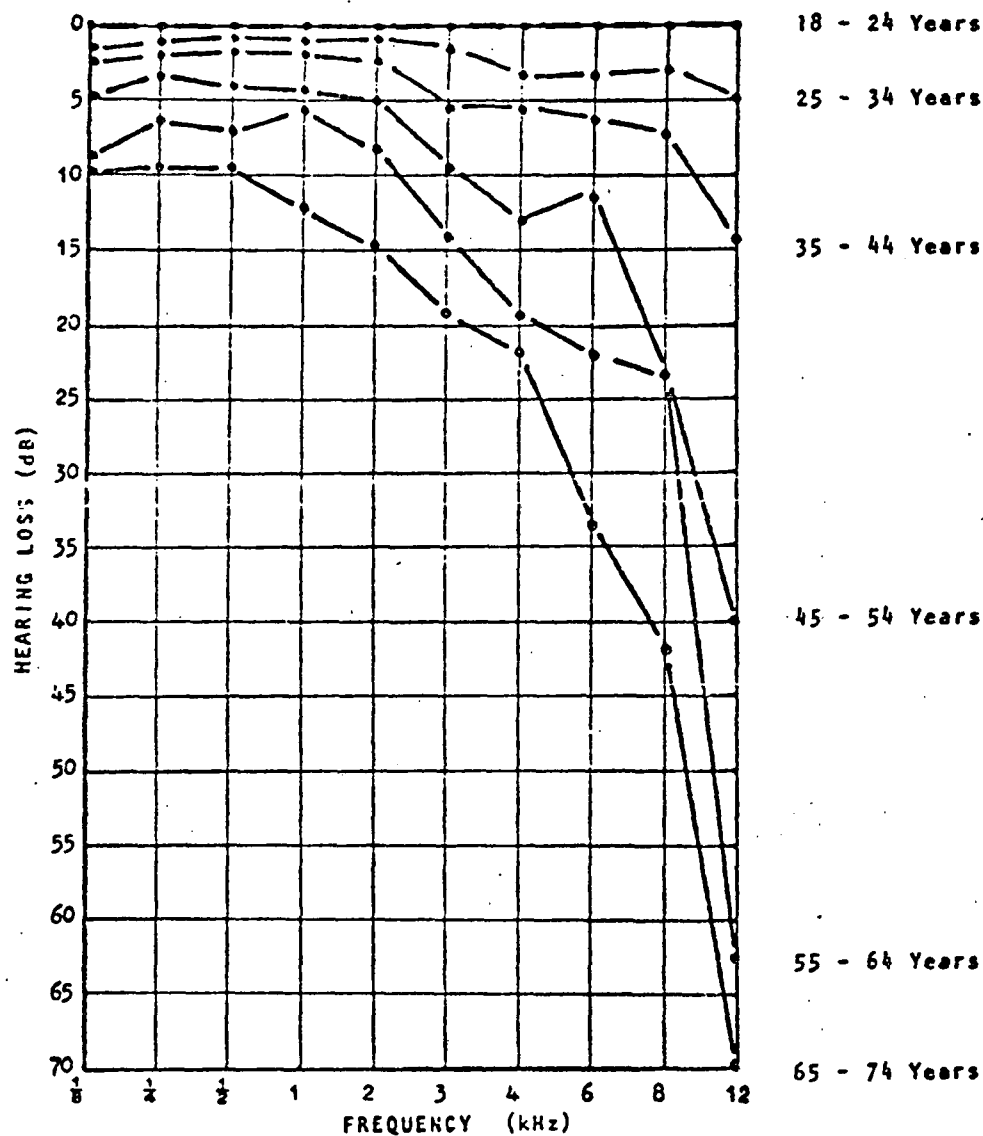
Glorig³¹ estimated a presbycusis correction applicable to the three "speech frequencies" (500, 1000 and 2000 Hz) important in the assessment of disability due to occupational noise-induced hearing loss. His figures are shown in Table 4-1 to illustrate the magnitude of the effect. Other presbycusis data, derived from industrial surveys^{32,33} are shown in Table 4-2. For comparison, the British data of Hinchcliffe,²⁵ which are used by Robinson³⁴ in his predictive method are summarized in Figure 4-2.

Table 4-1
Glorig's correction for 3F/3.³¹

Age (years)	25	30	35	40	45	50	55	60	65	70
Correction (dB)	0	+1	+1	+2	+2	+2	+3	+5	+7	+13

Presbycusis and Other Factors Affecting Hearing

Von Schulthess and Huelsen³⁵ and von Schulthess³⁶ have pointed out that, audiolgically, the endogenous and exogenous factors causing the rise in hearing level with age are not distinguishable. One can only say that group hearing levels rise naturally with age (presbycusis), due probably to both peripheral and central aging process,²⁶ and that this effect is enhanced (in a way which for lack of other evidence is generally presumed to be additive) by noxious environmental, mostly acoustic influences (Glorig's "sociacusis") and specific exposures to excessive noise.



Note: Median hearing loss is related to median threshold at 21.5 years of age (Hinchcliffe).²⁵
 For the purposes of the present document, clinically normal female ears may be equated with non-noise exposed clinically normal male ears.

Figure 4-2. Threshold of Hearing as a Function of Clinically Normal Female Ears (Random Sample Population)

TABLE 4-2
 PRESBYCUSIS DATA UPPER REGISTER: MEDIAN
 AGE-INDUCED HEARING LEVELS (NON-NOISE-EXPOSED MEN)
 ROUNDED TO NEAREST DECIBEL. FROM: PASSCHIER-VERMEER.³²

Age	Frequency (Hz)							
(Years)	250	500	1000	2000	3000	4000	6000	8000
25	0	0	0	0	0	0	0	0
30	1	1	1	1	2	3	4	3
35	1	1	1	2	4	6	7	16
40	2	2	2	4	6	9	12	11
45	3	3	3	6	9	13	16	15
50	4	4	4	8	14	18	22	22
55	5	6	6	11	18	23	27	28
60	7	8	8	14	22	28	33	35
65	9	10	10	18	27	33	40	43
70	12	13	13	24	33	40	47	53
75	14	16	17	30	40	47	55	62

Comparable data derived from Schneider *et al*³³ corrected to HL = 0 at Age 25

Age	Frequency (Hz)							
(Years)	250	500	1000	2000	3000	4000	6000	8000
25	-	0	0	0	0	0	0	0
30	-	0	1	1	3	3	4	2
35	-	1	1	3	5	5	7	5
40	-	1	2	4	8	9	10	9
45	-	2	3	6	12	14	14	13
50	-	3	5	8	15	18	19	19
55	-	4	7	12	20	25	25	25
60	-	6	9	16	27	32	33	36
65	-	8	12	22	34	42	42	50

SUMMARY – NORMAL AUDITORY FUNCTION

Normal hearing is regarded as the ability to detect sounds in the audio-frequency range (16 Hz to 20 kHz) according to established standards or norms. This range varies little in human populations around the world. However, there is considerable individual variation in hearing ability. As a general rule, for example, women in industrial countries typically have better hearing than men.

In the normal auditory mechanism, sound is transmitted to the inner portion of the ear when sound vibrations imported to the eardrum are transported across the middle ear.

The stapedius and tensor tympani muscles, when contracting, increase the tension of the conductive mechanism and thereby reduce the amount of sound energy delivered to the inner ear. Since high intensity sound causes these contractions, the ear has a limited built-in protective device. However, there is enough of a lag between sound onset and muscle contraction, that a sudden impulse is not attenuated by the protective mechanism.

Hearing sensitivity normally diminishes with age, a condition known as presbycusis. Consequently, corrections for aging should be considered in examining data on hearing loss due to noise exposure.

REFERENCES

1. BENOX (Biological effects of Noise) Group (Ades, H.W. & Colleagues) (1 December 1953). *BENNOX REPORTS: An exploratory study of the biological effects of noise*. Univ. Chicago: ONR Proj NR 144079.
2. Kodman, F. Jr., and Sperezze, A. Hearing factors in school-age children. *Acta Otol.* 50, 1959.
3. Mikaeman and Barimian (1971) (see related publication²³)
4. Rosen, S. & Rosen, Helen V. (1971). High frequency studies in school children in nine countries. *The Laryngoscope*, 81, 1007, 1013.
5. Rosen, S., Olin, P. & Rosen, Helen, V. (1970). Dietary prevention of hearing loss. *Acta Otolaryng*, 70, 242-247.
6. Corso, J.F. (November 1963) Bone-conduction thresholds for sonic and ultrasonic frequencies. *J acoust Soc Amer*, 35 (11), 1738-1743.
7. Roberts, Jean and Bayliss, David. Hearing Levels of Adults by Race, Region, and Area of Residence, United States 1960-1962. National Center for Health Statistics, Series 11, Number 26. U.S. Department of Health, Education, and Welfare, Public Health Service, Washington, D.C. (1967).
8. Environmental Protection Agency/Airforce Aerospace Medical Research Laboratory, A SCIENTIFIC BASIS FOR LIMITING NOISE EXPOSURE FOR PURPOSES OF HEARING CONSERVATION. EPA document EPA - 550/9-73-001 (in press) 1973.
19. Riley, E.C., Sterner, J. H., Fassett, D.W. and Sutton, W.L. Ten years' experience with industrial audiometry. *Am. Ind. Hyg. Assoc. J.* 22, 151-159 (1961).
10. Kylin, B. Temporary threshold shift and auditory trauma following exposures to steady-state noise. An experimental and field study. *Acta Otolaryng. Suppl.* 152, 1-93 (1960).
11. Dieroff, H. G. Sex differences in resistance to noise. *Arch. Ohren-Nasen-Kehlkopfheilk.* Ver, z, Hals-Nasen-Ohrenheilk, 177, (1961).
12. Gallo, R. & Glorig, A. (1964). Permanent threshold shift changes produced by noise exposure and aging. *Amer Ind. Hyg. Assoc. J.* 25, 237-245.
13. Ward, W. D., Glorig, A. & Sklar, Diane L. (April 1959). Temporary threshold shift from octave-band noise: applications to damage-risk criteria. *J acoust Soc Amer*, 31 (4), 522-528

14. Glorig, A., Wheeler, D., Quiggle, R., Grings, W., and Summerfield, A., 1954 Wisconsin State Fair Hearing Survey: Statistical treatment of clinical and audiometric data. American Academy Ophthalmology and Otolaryngology and Research Center Subcommittee on Noise in Industry, Los Angeles, California, 1957.
15. Jepsen, Otto, MODERN DEVELOPMENTS IN AUDIOLOGY, Middle-ear Muscle Reflexes in Man, in James Jerger, New York: Academic Press, 1963.
16. Reger, S. N., Mensel, O. J., Ickes, W. K. and Steiner, S. J., Changes in Air Conduction and Bone Conduction Sensitivity Associated with Voluntary Contraction of Middle Ear Musculature, in SEMINAR ON MIDDLE EAR FUNCTION. Report 576 U. S. Army Medical Research Laboratory, Fort Knox, Kentucky, J. L. Fletcher, (ed.), 171-180, 1963.
17. Ward, W. Dixon, Damage-risk Criteria for Line Spectra, *J. Acoust. Soc. Am.*, 34, 1610-1619, 1967.
18. Weaver, E. G., Vernon, Jack and Lawrence, Merle, The Maximum Strength of the Tympanic Muscles, *Ann OR and L.* 64, 383-391, 1955.
19. Perlman, H. B., The Place of the Middle Ear Muscle Reflex in Auditory Research, *Arch. Otolaryngol.* 72, 201-206, 1960.
20. Neergard, E. B. and Rasmussen, P. E. Latencies of the Stapedus Muscle Reflex in Man, *Arch. Otolaryngol.* 84, 173-180, 1966.
21. Wersall, R., *Acta Otolaryngol.*, 139, Supp., 1958.
22. Fletcher, J. L., Protection from High Intensities of Impulse Noise by way of Preceding Noise and Click Stimuli, *J. Audit Res.*, 5, 145-150, 1965.
23. Simmons, F. B., Individual Sound Damage Susceptibility: Role of Middle Ear Muscles, *Ann. Otol. Rhinol. Laryngol.*, 72, 528-547, 1963.
24. Durrant, J. D. and Schallop, J. K., Effects of Differing States of Attention on Acoustic Reflex Activity and Temporary Threshold Shift, *J. Acoustic. Soc. Amer.*, 46, 907-913, 1969.
25. Hinchcliffe, R. (1959). The threshold of hearing as a function of age. *Acoustica*, 9, 303-308.
26. Konig, E. Compt. Rend Seances, 11 Congr. Ord. Soc. Internat. Audiol. pp. 87-97, 1955.
27. Davis, H. and Silverman, S. R. HEARING AND DEAFNESS: New York: Holt, Rinehart and Winston, 1970.

28. Hinchcliffe, R. Report from Wales: Some relations between aging noise exposure and permanent hearing levels changes. Committee S3-W-40 of the American National Standards Institute, Anderson, Indiana, August 1969.
29. Rosen, Samuel. Noise, Hearing and Cardiovascular Function. In *PHYSIOLOGICAL EFFECTS OF NOISE*, Welch and Welch, eds. New York: Plenum Press (1970).
30. Glogig, Aram and Nixon, J. Hearing loss as a function of old age. *Laryngoscope*, 72 (1962).
31. Glogig, Aram, and Davis, H., Age, noise and hearing loss. *Ann Otol.* 70. 556 (1961).
32. Passchier-Vermeer, W. (April 1968). *Hearing loss: due to exposure to steady-state broadband noise*. Instituut Voor Gezondheidstechniek, Sound & Light Division, Rpt. 35, 18 pp.
33. Schneider, E. J., Mutchler, J. E. Hoyle, H. R., Ode, E. H. & Holder, B. B. (1970). The progression of hearing loss from industrial noise exposures. *Amer. Ind. Hygiene Assoc. Jour*, 31, May-June, 368-376.
34. Robinson, D.W. Estimating the Risk of Hearing Loss due to Continuous Noise. In *Occupational Hearing Loss*, D. W. Robinson, Ed. Academic Press, N. Y., 1971.
35. Schulthess, G. V. & Huelsen, E. (1968). Statistical evaluation of hearing losses in military pilots. *Acta oto laryngologica*, 65, 137-145.
36. Schulthess, G. V. (1969) Statistical evaluation of hearing losses in military pilots (2nd Rpt). *Acta oto-laryngologica*, 68, 250-256.

SECTION 5

NOISE-INDUCED HEARING LOSS – TEMPORARY AND PERMANENT SHIFTS IN AUDITORY THRESHOLD FOLLOWING NOISE EXPOSURE

The prevalence of hearing loss among workers in noisy industries has been recognized since ancient times, and a popular description of excessively loud noise is “deafening.” Yet, it is still not adequately appreciated by the general public that there is a causal link between noise exposure and hearing loss. If the hazard is understood, it is, perhaps, regarded by many as a remote contingency or as one that has little consequence for those afflicted. It is possible, too, that while people exposed to intense noise frequently experience a substantial Noise-Induced Temporary Threshold Shift (NITTS), sometimes accompanied by tinnitus (ringing of the ears), the fact that very often such symptoms largely disappear within a short time may mislead people into believing that no permanent damage has been done by the noise.

Observations in animals as well as in man show that noise reaching the inner ear attacks directly the hair cells of the hearing organ (the organ of Corti). As the intensity of the noise and the time for which the ear is exposed to it are increased, a greater proportion of the hair cells are damaged or eventually destroyed. The function of the hair cells is to transduce the mechanical energy reaching the ear into neuro-electrical signals, which are then carried by the auditory nerves to the brain. In general, progressive loss of hair cells is inevitably accompanied by progressive loss of hearing as measured audiometrically.

There is a great deal of individual variation in susceptibility to noise damage. However, any man, woman, or child whose unprotected ears are exposed to noise of sufficient intensity is, in the long run, likely to suffer some degree of permanent noise-induced hearing loss for which there is no foreseeable cure.

It remains an open question as to the level of noise which is within safe limits for all ears. In this connection, it is important to bear in mind the fact that neither the subjective loudness of a noise, nor the extent to which the noise causes discomfort, annoyance, or interference with human activity, are reliable indicators of its potential danger to the hearing mechanism.

Clinical observations of noise-induced hearing loss have been reported over more than a century. However, the problem has received intensive study only during the past three or four

decades. Since the World War II, substantial data have been gathered on the effects of intense sound (particularly industrial noise) on the ear. Based upon the available data, numerous criteria and noise limits have been established for the purpose of hearing conservation. Some of these have received national or international acceptance or standardization and some have been embodied in state and federal legislation. An important present difficulty for the legislator, administrator or noise control engineer concerned with protecting human hearing against noise is the fact that confusing and sometimes conflicting guidance is offered by the multiplicity of official or semiofficial standards, regulations or guidelines now in existence. Clearly, there is an urgent need for one set of guidelines to be elevated and urged for universal adoption. This document should help accomplish that task, since the conclusions reached in this work apply to both occupational and non-occupational exposure at work, in the home, in transportation, in recreation, or at large in the street and other public places.

The major topics to be discussed in this section will relate to the degree to which ear damage occurs in the wake of noise exposure. There will also be some discussion of the mechanism of noise damage in the ear, damage-risk criteria and related calculation, and factors influencing the incidence of Noise-Induced Permanent Threshold Shift. (NIPTS)

There are a large number of causes of permanent hearing damage, many of which are beyond the control of the individual who is victimized by destruction in his ear(s). Noise exposure, for the most part, can be avoided or reduced in a number of ways. Therefore, the damaging effects of noise upon the ear must be regarded as a preventable influence—preventable by abatement of the noise, by alteration of operations in and around the noise, or by protection of the ear with the use of sound reducing materials or devices.

TYPES OF ADVERSE EFFECTS ON HEARING

Noise-Induced Permanent Threshold Shift (NIPTS).

The permanent loss of hearing ascribable to noise exposure, as opposed to other factors (aging, drug toxicity, etc.) is called Noise-Induced Permanent Threshold Shift (NIPTS). The shift in threshold refers to the loss in sensitivity of the ear. Details of hearing test techniques may be found in a related publication.²³

Noise-Induced Temporary Threshold Shift (NITTS)

The temporary loss of hearing ascribable to noise exposure is called noise-induced temporary threshold shift (NITTS) and is mentioned frequently in this chapter.

THEORIES RELATING NOISE EXPOSURE AND HEARING LOSS

Because most of our data concerning the long-term hazard of noise come from 8-hour industrial-type noise exposures, there is a relative lack of information about shorter-term intermittent or incomplete daily exposures, and virtually no data about continuous exposure to noise going on longer than 8 hours, or around the clock. One is accordingly driven to make interpolations and extrapolations on the basis of theories of noise trauma. Two main theories have been supported by substantial amounts of field observation and experimental work. A continuing difficulty in setting guidelines for safe noise exposure is that predictions using these theories conflict in some circumstances. Because the conflict is not resolvable in many circumstances, an empirical decision has to be faced as to which theory to follow in evaluating a particular noise hazard.

The Equal Energy Hypothesis in Damage Risk Criteria

The "equal-energy" hypothesis argues that the hazard to the hearing is determined by the total energy (a product of sound level and duration) entering the ear on a daily basis. This rule is basic to the damage-risk criteria embodied in certain important and widely used regulatory or guiding documents, notably the 1956 U. S. Air Force Regulation AF 160-3.¹ The "equal-energy" rule allows a 3-dB increase in sound pressure level (expressed in dB) for each halving of the duration (below 8 hours) of continuous daily steady-state exposure. Extrapolation to durations of continuous noise exceeding 8 hours daily exposure and extension to extremely brief exposures or impulses have only recently been proposed. In practice, a cutoff is introduced by the widely recognized mandatory absolute limit of 135 dB² for unprotected exposure, irrespective of duration. Botsford³ has remarked, there is still a lack of experimental or empirical verification of the "equal-energy" hypothesis except perhaps for overall durations of daily occupational exposures extending over years, the only application for which the equal energy rule was originally proposed. The theory has the attractions of simplicity and a certain *a priori* reasonableness. (See Proceedings of the International Conference on Noise as a Public Health Problem⁴).

The "Equal Temporary Effect" Hypothesis

This theory, originally based largely on the work of Ward, *et al.*,^{5,6} argues that the long-term hazard (of PTS) of steady-state noise exposure is predicted by the average TTS produced by the same daily noise in the healthy young ear. As Botsford³ has noted in a recent review, this hypothesis is plausible because (unlike the "equal-energy" rule) it relates to an observable physiological function of the ear. Moreover, recent work suggests that a unifying hypothesis of metabolic insufficiency induced in the hearing organ by noise may underlie both the temporary and permanent hearing defects caused by excessive noise. The essence of the

supporting data is that noise intense enough to cause PTS in the long run is intense enough to produce TTS in the normal ear, while noise that does not produce measurable TTS is not associated with NIPTS.⁷ TTS studies also tend to support the observation (reflected in industrial studies of PTS) that intermittent noise is less harmful than unbroken exposure to steady-state noise at the same level.^{8,9} Adoption of this theory has led to a number of current criteria, including that of the Committee on Hearing and Bioacoustics of the National Research Council (1966), considered below.

CHABA Criterion for Steady-State Noise Exposure

CHABA's criterion is based essentially upon the hypothesis of "equal temporary effect" already alluded to. In essence it states that a noise exposure is unsafe if, upon testing the normal ear two minutes after the cessation of the exposure, an average TTS_2 of 10 dB is exceeded at audiometric frequencies up to 1000 Hz, 15 dB at 2000 Hz, or 20 dB at 3000 Hz and above.¹⁰ According to Ward¹¹ this criterion reflects the empirical observation that in most normal-hearing people, a TTS_2 of 20 dB or less recovers completely within 16 hours (when the worker would be due to renew a typical 8-hour industrial exposure). The corollary to that is that it is deemed unlikely that any PTS is building up when the TTS recovers completely before the commencement of the next waking day. (A fraction of "sensitive" ears, of course, will not recover completely.) This makes no allowance for post-work, non-occupational exposure, however.

DATA ON EFFECTS OF NOISE ON HEARING

Data on the effects on hearing are given for two main types of noise, namely, continuous (or steady-state) and impulsive noise. For purposes of hearing conservation criteria, noise refers to airborne sound contained within the frequency range of 16 Hz to 20,000 Hz (20 kHz). Sound energy outside that range (ultrasonics, infrasonics, vibration) is considered in a separate chapter.

Although some other noise-measurement units are alluded to, this section, in general, adopts A-weighted sound level (in dBA) for the specification of steady-state noise levels, and peak sound pressure level (SPL) in decibels (dB) relative to standard reference sound for the specification of impulse noises (see Section 1). When A-weighted sound levels are given, the use of international standard measurement techniques, instrumentation, and weighting characteristics is assumed.

Ongoing Noise and Hearing Loss

Procedures for calculating Equivalent Continuous Sound Level (Leq) in dBA, in the cases of atypical, interrupted or intensity-modulated, steady-state noise exposure are given in a recent EPA-Air Force publication.¹² This source also may be used to determine exposures in dBA from octave-band sound levels measured in decibels relative to 0.00002 N/m^2 .

Industrial Experience

There is a plethora of published information about the effects of long-term noise exposure upon the hearing of workers in the manufacturing and construction industries, as well as that of aviators and others in noisy occupations: several recent monographs and surveys have been published on this topic.^{13, 18} A recent survey by the National Institute of Occupational Safety and Health (NIOSH)¹⁹ contains a descriptive summary of some of the more important audiometric surveys carried out in the United States and abroad during the preceding decade.

Temporary hearing loss attributable to fatigue of the inner ear (or Noise-Induced Temporary Threshold Shift, NITTS) lasting from a few seconds to a few days can occur after brief exposure to high sound levels or from day-long exposure to more moderate levels of on-going noise. Regular (day-by-day) exposure to such levels over a long period (days to years) can result in damage to the inner ear, a sensorineural hearing loss (NIPTS) that is permanent and so far as is presently known, irreversible. It can be prevented only by protecting the ear from excessive noise exposure.

NIPTS is usually preceded by, and may at any time be accompanied by, 2 NITTS. The typical pattern of NIPTS seen in the audiogram is maximum loss in the range 4000 to 6000 Hz, with a somewhat smaller loss (initially) at higher and lower test frequencies. Because the loss is sensorineural, it is seen in both air- and bone-conduction audiograms.

Gallo and Glorig²⁰ examined audiometric data from 400 men (aged 18-65) and 90 women (18-35) exposed regularly to high-level industrial plant noise (102 dB SPL overall; 89, 90, 92, 90, 90 and 88 dB, respectively, in the octave bands spanning 150 to 9600 Hz). These subjects were selected from larger groups of 1526 male and 650 female employees, using a screening process designed to exclude otological abnormalities and irrelevant noise exposure (e.g., to military noise), and to maintain in the men a high correlation between age and time on the job. The purpose of the study was to look specifically at age and duration of steady-state noise exposure as factors in PTS. It showed quite clearly that hearing level tends to rise relatively rapidly over the first 15 years of exposure but then to level off as reflected in the higher audiometric frequencies, 3, 4 and 6 kHz. By contrast, hearing level at 500 Hz, 1 and 2 kHz rose more slowly but continued to rise in an essentially linear manner over exposures up to some 40 years.

A comparison of data for 4 kHz in the men with equivalent data from non-noise-exposed males showed that the effects of the age and noise were not simply additive. Examination of individual differences showed that the spread of hearing level within groups tends to increase with both increasing exposure time and with audiometric frequency (a similar effect has been reported by Taylor, *et al.*²¹ Also, the time and frequency dependence of noise-induced hearing level change was found to be similar for most subjects. Gallo and Glorig

concluded from this study that early evidence of PTS at 4000 Hz is the best indicator of susceptibility to noise-induced PTS on either a group or individual basis. A cognate study by Taylor *et al.*²¹ in female jute weavers supported Gallo and Glorig's finding that noise-induced deterioration in hearing takes place rapidly and mainly in the first 10 to 15 years of exposure, with, however, further deterioration at the speech frequencies continuing in later years.

Taylor, *et al.*²¹ carried out retrospective audiometric studies of groups of women working in or retired from the jute weaving industry in Scotland. The contributions to their group hearing levels attributable to the regular noise (99-102 dB SPL overall with higher peaks) to which they had been exposed were evaluated by comparison with non-noise-exposed control subjects and by corrections for presbycusis using Hinchcliffe's²² median data. Generally, this study supported the conclusions of Gallo and Glorig.²⁰ Namely, these findings were that the effect of noise on hearing levels is greatest, earliest and most rapid at the higher audiometric frequencies (4 and 6 kHz), where it mostly takes place in the first 10 or 15 years of occupational exposure,¹⁵ but that further deterioration involving frequencies in the range of 1 to 3 kHz (being most marked at 2 kHz) becomes manifest during the third decade of noise exposure. After as few as 10 years of on the job exposure in areas of high-level (90 dB SPL) industrial plant noise, men as young as 30 years old may have hearing levels worse than non-noise-exposed men twice their age and may, in some cases, already suffer impaired speech perception.²⁰

PTS produced by noise exposure and PTS produced by aging (presbycusis) may not be distinguishable on either a group or individual basis.²⁰ NIPTS is found primarily among industrial workers who have been exposed repeatedly and over a long period to high-intensity noise. Provided that the ears affected are otologically normal, the PTS found in noise-exposed people may be attributed to the combined effects of aging and habitual noise exposure. Moreover, the component attributable to noise exposure may be viewed as the result of repeated noise-induced TTS. Some audiologists subscribe to the view that noise-exposure merely hastens the aging process, although such a hypothesis can be based only upon circumstantial evidence.

Gallo and Glorig²⁰ have summarized some general characteristics of NIPTS, as seen in occupational contexts, namely:

1. The magnitude of the resulting PTS is related to the noise levels to which the ear has habitually been exposed.
2. The magnitude of the resulting PTS is related to the length of time for which the ear has habitually been exposed.
3. The growth of occupationally related PTS at 4000 Hz is most rapid during the first 10 to 15 years of exposure, after which it tends to slow down (see also Passchier-Vermeer²³).
4. There are large individual differences in susceptibility to noise-induced PTS.

Comparable variability is seen in individual hearing levels and in the effects of aging (presbycusis). Summar and Fletcher²⁴ have contended that age at the time of exposure is probably not a significant factor in industrial NIPTS.

Tinnitus Associated with Occupational NIPTS

Tinnitus (ringing in the ears) may be, at first, the only symptom in many cases of occupational hearing loss; and it is fairly frequently associated with the condition. Chadwick²⁵ has reported an incidence of 30 per cent in one industrial survey in Britain.

Patients with occupational NIPTS frequently notice symptoms upon changing from one noisy job to another, or from a noisy job to a quiet one, possibly because they have adapted to or learned to cope with any handicaps due to the noise in a familiar situation.

Social Significance of Hearing Loss at Retirement

Kell, *et al.*²⁶ have reported that more than two thirds of a surveyed group of elderly (mean age 64.7 years) women who had worked as weavers (with steady daily noise exposures of approximately 100 dBA) for up to 50 years had difficulty with such social intercourse as understanding conversation, using the telephone, and attending to public meetings or church services. By contrast, fewer than one in six age-matched women who had not been in a noisy occupation was similarly disadvantaged.

The Reliability of the Data from Industrial Studies

Unfortunately, much hearing loss data from industry is heavily "contaminated by" what Glorig and others¹¹ have called "sociocusic" factors (e.g., undeterminable losses due to non-occupational noise exposure in military, recreational or other pursuits, or to disease affecting the ear. The data was further contaminated by the effect of presbycusis, which is inextricably bound up with the time-dependent effect of noise exposure (and shift presumed largely on *a priori* rather than evidential reasoning to be simply additive); and even within the setting of industrial noise exposure, by lack of continuity (e.g., personnel changing jobs) affecting both retrospective studies.

EFFECTS OF LOUD MUSIC

Several recent studies have confirmed that the overall sound levels of very loud rock and roll and similar music frequently exceed current hearing damage-risk criteria and can produce large amounts of TTS in both musicians and listeners.^{27,33} Flugrath's²⁹ and other measurements have shown that typical rock music can be regarded, when considering the hair cells, as a steady-state noise with interruptions. Typically, the maximum acoustic output from the

bands' amplifiers lies in the region of 2000 Hz. Dey³² found that typical exposures averaging 100 to 110 dBA for up to 2 hours produced TTS₂ exceeding 40 dB in 16 percent of young adults tested. Rintlemann and Borus²⁸ measured typical levels of 105 dBA and found that some 5 percent of musicians (mostly quite young) showed evidence of NIPTS attributable to their music. Clearly, the hazard is an occupational one for the performer and usually a recreational one for the listener.

Lipscomb^{30,31} has demonstrated cochlear damage in guinea pigs exposed to 88 hours of recorded rock and roll music adjusted to peak at 122 dB, a level that can be exceeded at the ears of musicians and nearby listeners in some instances where excessive amplification of the music is used in reverberant rooms or dance halls. Dangerous levels can also be reached using domestic stereos.³⁴ In a comparative study of the noise hazard in young people's recreation, Fletcher³⁵ found playing rock-bands to be exceeded in degree of hearing hazard only by motorcycle and drag racing and by intensive sport shooting with inadequate ear protection. Fletcher showed incidentally, that young men and women are equally at risk of hearing damage when exposed to over-amplified rock music. A similar conclusion was reached by Smithley and Rintelmann.³⁶

EXPERIMENTAL SUPPORT FOR THE NOISE DAMAGE—RISK THEORIES

Many studies have been carried out in an attempt to obtain scientific support for the equal energy hypothesis and for the theories that relate TTS and PTS.

Burns' Approach

The search for a reliable prognostic test for individual susceptibility to PTS based on tests of TTS continues.³⁷ Some promising findings have recently been published by Burns.³⁸ He has developed a relative index (based on the regression of TTS on hearing level) of susceptibility to TTS (D_T) and, using the predictive method of Robinson,³⁹ an index (D_p) of PTS, being the deviation (dB) of the individual's age-corrected HL from the predicted median value of HL for his peers in age and noise-exposures to be grouped for purposes of correlation with the TTS index D_T . Having determined values of D_T for 3 groups of subjects divided by sound level (LA_2 in the range 93 to 104 dB) causing TTS, Burns has performed regression of D_T upon D_p for numerous combinations of audiometric test frequencies and found a positive if rather low (not greater than 0.34) correlation coefficient for several such combinations. Somewhat unexpectedly, the most promising result was found when D_T was based on low audiometric frequencies (1 and 2 kHz) and D_p on high (3, 4 and 6 kHz), for reasons that the author admitted remain obscure. Burns considers this test to have potentialities and has suggested possible ways of strengthening it: its present weakness rests largely in the large residual variance of D_T in the regression of D_T upon D_p .

TTS₂ as a Predictor of Hazardous Noise Exposure

Luz and Hodge⁴⁰ have recently presented complementary evidence, from studies of recovery from impulse-noise induced TTS in monkeys and men, to show that the recovery is not a simple process and that, accordingly, a single measure such as TTS₂ may not be a particularly reliable predictor in the construction of damage-risk criteria for hazardous noise exposure. Luz and Hodge have described multiple TTS recovery patterns and have postulated the existence of two types of threshold shifts, due to “metabolic” and “structural” auditory fatigue respectively. They adduce the “rebound” recovery phenomenon as strong evidence for a delayed component in recovery from TTS (evident from other work also) and hypothesize with some conviction that this is related to permanent damage.

“Equal-Energy” Hypothesis in Predicting TTS and PTS

Some recent work by Ward and Nelson⁴¹ on noise-induced threshold changes in chinchillas appears to confirm the observations of Eldredge and Covell⁴² in guinea pigs that there is an equivalence of time and energy—at least within certain ranges of parameters—for continuous, uninterrupted noise exposure. In other words, there is probably a limiting constant product of intensity and time (analogous to Robinson’s “immission”) for single unbroken exposures. Ward and Nelson⁴¹ urge caution, however, in extrapolation to repeated or to interrupted exposures. They cite the findings of Miller, Watson and Covell⁴³ that frequent interruptions of noise exposure by noise-free periods reduce both the TTS and the PTS produced by the noise.

Growth of TTS in Constant Noise

Miller, *et al.*⁴⁴ have shown in the chinchilla exposed to constant octave-band (300-600 Hz) noise at 100 dB SPL that TTS grows in magnitude and in audiometry range with duration of exposure over the first 1 to 2 days, then remains constant (asymptotic) with continuing exposures up to 7 days. After cessation of exposures of that duration, the TTS decays approximately exponentially over some 5 days (decay took about 2 days after identical exposures lasting only 193 minutes). These noise exposures produced demonstrable cochlear damage, although this was associated with only a small PTS measured 3 months after the noise exposure. A similar observation was also made by Lipscomb.⁴⁵

TTS from Prolonged Noise Exposure

Recent work in the chinchilla⁴⁶ and in man⁴⁷ has confirmed that TSS due to a maintained steady-state octave-band noise exposure reaches an asymptotic level after some (up to 12) hours, and that recovery from asymptotic TTS is slow (3 to 6 days for complete recovery in man) and exponential in form.

Asymptotic TSS as a Function of Noise Level

Using behavioral audiometry in monaural chinchillas, Mills⁴⁸ has further demonstrated asymptotic TTS following 4-kHz octave-band exposures of up to 9 days (see also Carder and Miller⁴⁶). The magnitude of TSS at asymptote (TS_{400}) was found empirically to be predicted by the equation:

$$TS_{400} = 1.7 (\text{SPL} - 47),$$

where SPL is the sound pressure level in decibels relative at 0.00002 N/m^2 . The frequency distribution, temporal pattern, and degree of persistence of the TTS were also found to depend on the noise exposure level. TTS caused by 80-dB noise was purely temporary, decaying from the asymptotic value to zero in 3 to 6 days. Noise in the range 86 to 98 dB, however, caused a "permanent" component to persist in the threshold shift, which had not decayed to zero after 15 days. The magnitude of this residual ("permanent") threshold shift was related to noise level, being of the order of 10 dB at the higher audiometric frequencies following 86-dB exposure, about 20 dB following 92-dB exposure, and up to 40 dB (at 5.7 kHz) following 98-dB exposure. It cannot, of course, be inferred that similar values or temporal patterns of TTS and PTS would be caused by the same exposures in man, but this work would appear to support a correlation between temporary and persistent threshold shift, both of which showed a similar dependence of magnitude on the noise exposure level. The persistent threshold shift found by Mills may reasonably be presumed to be an element of NIPTS.

Pitfalls of Generalizing from Animal Studies to Man

Price⁴⁹ has shown that, although the cat is regarded as being more susceptible than man to behaviorally measurable NIPTS (see Miller, Watson and Covell⁴³), as is the chinchilla,⁵⁰ the cochlear microphonic in the cat appears to be much more resistant to alteration by noise stress (at 5 kHz) than is the auditory threshold measured (TTS) in man (although both changes follow a rate law that is linear with the logarithm of time). Price urges caution in drawing parallels between cochlear microphonic and TTS data, although he suggests that mechanical factors in the peripheral auditory mechanism may explain certain paradoxes in the growth of TTS resulting from high intensity sustained versus impulse noise exposure (see Ward, *et al.*,⁵¹). Price⁵² has recently published similar findings at 500 Hz.

Poche, *et al.*⁵³ have shown that impulsive (cap gun) noise and pure tones (2 kHz at 125-130 dB SPL for 4 hours) produce similar patterns of hair cell damage in the guinea pig. They point out, however, that no firm correlation has yet been established between hair cell damage and hearing loss either in animals (see Miller, *et al.*⁴⁴) or in man.

Uncertain Relation of PTS to TTS and Cochlear Damage

Other observations in the chinchilla⁴⁴ have shown that quite a substantial and slowly decaying asymptotic TTS, as well as simultaneously induced external hair cell damage of a diffuse and extensive nature, can be associated with only a small (less than 10 dB) residual NIPTS measured

behaviorally 3 months following exposure to the prolonged causative noise (300-600 Hz octave-band noise at 100 dB SPL for up to 7 days). Poche, Stockwell and Ades⁵³ have also commented (following studies of impulsive noise and cochlear damage) on the lack of quantitative evidence correlating hair cell loss with hearing loss.

Asymptotic TTS in Man

In tentative observations upon his own ear, Mills⁴⁷ has found evidence that TSS in prolonged (24-48 hour) octave-band noise reaches an asymptote in man, as in the chinchilla. The time to reach it appears to be in the range 4 to 12 hours for man; and the time required for complete recovery some 3 to 6 days.

Miscellaneous Factors Considered in TSS

In 1958, Trittipoe⁵⁴ maintained that pre-exposure non-TTS-producing noise levels as low as 48 dB SPL could enhance subsequent TTS due to a high (118 dB) brief noise exposure. This has been taken as evidence that there is no threshold of noxiousness for noise hazardous to the ear. This observation and its interpretation have, however, been disputed by Ward.⁵⁵

Karlovich and Luterman⁵⁶ have shown that phonation might exert a slight protective effect against NITTS. They have found that TTS was smaller following a 3-minute exposure to 1000 Hz tones at 100 dB SPL when the subjects phonated during the noise than when they were silent or merely whispered the same vowel rather than voicing it. Two possible mechanisms have been suggested to account for this phenomenon:

1. That phonation elicits and maintains the acoustic reflex.
2. That during phonation Z-axis vibrations of the skull "protect" the hearing by causing changes in the mode of oscillation of the stapes.

IMPULSIVE NOISE

Most of our knowledge of the aural hazard due to impulse noise, and practically all the data systematically relating exposure parameter to threshold shift, comes from studies of the effects of gunfire on the ear, with some supporting evidence from industrial data.

Incidence of NIPTS as a Function of Peak SPL

If all other characteristics of an impulse noise are held constant, TTS increases with peak SPL. Presumably, this would be true for NIPTS as well. An estimate of hearing damage-risk following daily exposure to a nominal 100 rounds of gunfire (rifle) noise at 5-second intervals has been developed by extrapolation of TTS data.⁵¹ An important assumption implicit in their calculations is that a given TTS₂ (TTS measured at 2 minutes after cessation of stimulation) will eventually lead to an equal NIPTS. Further discussion of Kryter and Garinther's predictions are included in a recent EPA document.¹²

Effect of Impulse Duration

The present state of knowledge indicates that a clear hazard exists and, accordingly, that ear protection should be in use when impulsive noises exceed a peak sound pressure level of 140 dB at the ear for more than 5 milliseconds regardless of rise time spectrum, or the presence of oscillatory transients. As duration decreases below 5 milliseconds, higher peak values may be tolerable. Exceeding a level of 165 dB SPL for short durations is likely to lead to cochlear damage in at least 50 percent of ears, even in the case of isolated impulses (see Acton,⁵⁸ and Coles, *et al.*¹⁷).

The figure of 165 dB SPL absolute maximum is considered over-stringent by some authorities, in relation to extremely brief exposures. Coles and Rice,^{59,60} for instance, have allowed 172 dB SPL for single impulses of 100 microseconds duration, and over 180 dB for impulses of less than half that duration (irrespective of pulse shape). This may be over-lenient.

Allowance for Repeated Impulses

A CHABA Working Group has recently arrived at an empirical weighting factor for reducing permissible levels of exposure when multiple impulse noises are heard. Essentially, the working group's current recommendation is to add or subtract 2 decibels from permissible values for each halving or doubling, respectively, of the number of impulses (or 5 dB for every tenfold change in the total number in a series of impulses).

High-Frequency Hearing Losses Due to Impulse Noise

Coles,⁶¹ Loeb and Fletcher⁶² have drawn attention to the fact that, although hearing loss due to many kinds of intense short-lived or impulsive noise appear audiometrically identical with loss due to continuous noise (showing the characteristic audiometric notch at 4000 Hz and progressive upward spread), certain kinds of impulsive noise, such as gunfire, are frequently associated with a substantial immediate TTS and potential permanent loss at higher frequencies (6 to 8 kHz and upward). This may be associated with particular parameters of the noise exposure such as extremely rapid rise and high peak level.⁶¹

Such high-frequency loss is not predicted, or is not treated as significant, by many of the existing damage-risk criteria or methods of hazardous noise exposure evaluation, which are narrowly restricted to the so-called "speech frequencies" below 4000 Hz. Sensitivity for frequencies above 2000 Hz can, however, be vitally important for several purposes in life, especially for the reception of speech heard against a background of noise. It is also important for the localization and identification of faint, high-pitched sounds in a variety of occupational (including military) and social situations. Thus, high-frequency hearing loss, should be prevented when possible.

Factors Influencing Hazard Due to Impulse Noise

There is no unequivocal evidence that a practical distinction need be made between the sexes or between age groups when predicting hearing damage risk due to impulse noise as it is here defined. Nor does any definitive evidence exist for a significantly different degree of susceptibility to impulse-noise-induced PTS in the case of children or persons with otological abnormality.

Combined Exposure to On-Going Noise with Added Impulsive Noise – Allowance for Impulsiveness

When impulsive noise exposure takes place at the same time as on-going (steady-state) noise, the hazard of each element to the hearing mechanism should be evaluated separately against its respective criterion. A conservative and greatly simplified approach is then to treat combined hazards as simply additive. For example, if for a given centile of the population at risk, a continuous noise exposure were predicted to cause NIPTS of 10 dB and a concurrent impulse noise exposure were predicted to produce 5 dB of NIPTS, then the combination may be predicted to produce 15 dB of NIPTS at that centile. Alternatively, some authorities might argue in favor of a logarithmic rule which would be somewhat less conservative.

Effects Found in Studies of Children

Gjavenes⁶³ has cited Scandinavian data showing that between about 1 and 4 percent of teenaged children may show hearing injuries resulting from the impulsive noise from fire-crackers or other noisy toys. He has also argued that this degree of risk accords with a damage risk criterion of 155 dB peak pressure for impulsive toy noise. He points out that there is no evidence that children's ears are more easily damaged by impulsive noise than are those of adults. All the data upon which existing impulse noise damage risk criteria are based have come, of course, from adults (mostly exposed to gun noise).⁶⁴

Methods for Predicting the Expected Hearing Loss Due to Exposure to Ongoing Noise

In the following paragraphs we present procedures for predicting the risk or amount of hearing loss to be expected from occupational-type noise exposure. This information is based upon the work of four international authorities in the field of industrial noise-induced hearing loss namely Baughn⁶⁵ Passchier-Vermeer,^{23,66} Robinson^{17,39} and Kryter.⁶⁴ Their methods may be used to predict the effect upon hearing, at selected centiles, of the adult population produced by daily 8-hour exposure to steady-state noise at levels in the range 75 to 90 dBA,

sustained for periods up to 50 years. The first three predictive methods summarized in the following paragraphs have been selected because:

1. They permit calculation of NIPTS (i.e., the noise-induced part of hearing level) for designated percentiles of the adult population.
2. They also include data permitting the inclusion of 4000 Hz in the computation, although they are based mainly upon the audiometric test frequencies 500, 1000 and 2000 Hz ("speech frequencies") currently accepted as essential to the evaluation of hearing impairment by most otologists in the United States.
3. They show fair agreement with one another.

Kryter⁶⁷ presents a fourth method that differs significantly from the other methods summarized here. He proposes 55 dBA as the threshold of significant hearing changes to the speech frequencies of 0.5, 1.0, 2.0 kHz, a value roughly 20 to 25 dB over the values obtained with the other commonly used methods. Some aspects of his procedure have been discussed recently in the literature⁶⁸⁻⁷¹ and Kryter has responded to these critiques.⁷²

"Industrial" Methods of Predicting Long-term Hazard from Daily Continuous Noise Exposure

These methods permit predictions of the amount of noise-induced change in hearing level to be predicted for designated fractions of otologically normal working adult populations exposed day after day to steady-state industrial-type noise, as a function of average noise level (or equivalent continuous sound level). These techniques are elaborated upon in a recent EPA - U. S. Air Force publication;¹² therefore, they will be treated quite briefly here.

Method and Data of Passchier-Vermeer

Passchier-Vermeer^{23,66} has analyzed the audiometric data from several surveys of industrial hearing loss. Making allowances for presbycusis, in 1968, she published procedures with graphs for determining the noise-induced part of hearing level evaluation as a function of daily noise exposure for the 25th, 50th, and 75th centiles of a working population.²³ In 1971,⁶⁶ she published some additional data including 10th and 90th centile estimates. Her results are applicable to daily 8-hour exposures to industrial-type noise up to 100 dBA. (For more detail, see related document published by EPA¹²).

Method of Robinson

Robinson^{17,39} has devised an idealized method for predicting hearing loss resulting from noise exposure. His method is based on a unique mathematical relationship (the hyperbolic tangent) between noise exposure and NIPTS, which is adjusted parametrically for population centile and audiometric frequency. The method applies to otologically normal adults

exposed to industrial noise for 8 hours per day over a period ranging from 1 month to 50 years. It yields estimates of the percentages of the exposed population that may develop NIPTS as a function of noise exposure (noise "immission"). Robinson's method has been criticized on the grounds that:

1. It is based upon a single, although substantial, study of otologically screened British industrial workers (Hinchcliffe).²²
2. The mathematical niceness of the predictive theory may not be entirely justified by the realities of industrial audiometric data and their sources of variance. (Discussed in detail in a related EPA document¹²).

Method of Baughn

Baughn⁶⁵ has amassed data from extensive industrial audiometric surveys in the United States. His work provides insight into how NIPTS develops at various centile points as a result of typical industrial noise exposure in the range 78 to 92 dBA. The prediction of NIPTS may in some respects be too high, however, owing to a probable contamination of the data by residual TTS and masking in the circumstances in which the audiometry was conducted. In some measurements, only 20 minutes recovery from the industrial noise was allowed before testing. (This method also is treated in a related EPA document¹²).

Averaging NIPTS Predictions Over the Three "Industrial" Methods

A summary chart of certain predictions that can be made concerning NIPTS and risk by combining the predictions of Passchier-Vermeer, Robinson and Baughn is presented in Table 5-1. (Extracted from a related EPA publication¹²).

The table gives the NIPTS for three frequency configurations: The average shift over .5, 1 and 2 KHz denoted by Speech (.5, 1, 2), the average shift over .5, 1, 2 and 4 KHz denoted by Speech (.5, 1, 2, 4) and the shift at 4KHz. A brief explanation of the table follows:

- o *Maximum NIPTS (90th percentile)* The NIPTS that can be expected after 40 years of noise exposure during adult life for the 90th percentile (i.e., 90 percent of the population will expect NIPTS less than the value in the Table and 10 percent greater than the value). This value can be considered a lifetime maximum since little or no further shift will take place due to this type of noise exposure."
- o *NIPTS (90th percentile) at 10 years.* The expected NIPTS after ten years of exposure during adult life not exceeded by 90 percent of the population.

- *Average NIPTS.* The gross average value of NIPTS obtained by averaging over a 40-year exposure duration and also over all the population percentiles.
- *Maximum Hearing Risk.* Hearing risk is defined as the difference between the percentage of people with a specified hearing handicap in a noise-exposed group and the percentage of people with a handicap in a non-noise-exposed (but otherwise equivalent) group. The hearing risk varies with exposure duration, and the Maximum Hearing Risk is defined as the peak value (largest difference) that occurs during the 40 years of exposure. Normally, but not always, this peak value occurs after 40 years of exposure.

Use of Industrial Exposure Tables to Approximate Effect of Less Uniform Noise Exposures

Most of our knowledge of the effect of noise upon the human ear comes from industrial audiological experience. More people are at risk from quasi-steady-state noise exposures of about 8 hours a day, 5 days a week for a working lifetime than from any other variety of noise exposure.¹² One method for applying our knowledge of the effects of industrial noise to non-industrial situations, is to rely on the equal energy hypothesis as an estimate of equivalent noise exposures.

Exposures to Continuous Noise Exceeding 8 Hours

An equivalent continuous sound level (L_{eq}) in dBA can be calculated for varying exposure times, based upon a normal daily exposure of 8 hours (discussed in a related EPA document¹²). For that duration only, L_{eq} is numerically equal to the energy equivalent of a continuous sound level in dBA. As in the case of unbroken steady-state exposure lasting less than 8 hours the nomogram (Figure 5-1) may be used to find L_{eq} for unbroken steady-state exposures of more than 8 hours. For an uninterrupted 24-hour exposure, L_{eq} is 4.8 dB greater than for an 8-hour exposure (this can be rounded off to 5 dB). Expressed another way, the hazard to hearing from a continuous 85 dBA noise lasting 24 hours is similar to the hazard of an 8-hour exposure to 90 dBA, provided, of course, that the noise is steady-state (not fluctuating markedly in level), broadly distributed (spanning a number of octaves), fairly uniform in spectrum without substantial discrete tonal components, and free from any significant addition of impulse sounds.

An exposure exceeding 24 hours may be treated as indefinite exposure. Allowances for level fluctuations in continuous noise, for intermittency (interruptions), and for the significant presence of simultaneous tonal components or impulses during prolonged exposure may be considered to obey rules similar to those governing these allowances in the case of exposures shorter than 8 hours (see below).

TABLE 5-1
SUMMARY OF EFFECTS PREDICTED FOR CONTINUOUS NOISE
EXPOSURE AT SELECTED VALUES OF A-WEIGHTED* SOUND LEVEL

75 dBA for 8 hours			
	Speech (.5,1,2)	Speech (.5,1,2,4)	4 kHz
Max NIPTS (90%-ile)	1 dB	2 dB	6 dB
NIPTS at 10 yrs (90%-ile)	0	1	5
Average NIPTS	0	0	5
Max Hearing Risk**	NA***	N/A	1
80 dBA for 8 hours			
	Speech (.5,1,2)	Speech (.5,1,2,4)	4 kHz
Max NIPTS (90%-ile)	1 dB	4 dB	11 dB
NIPTS at 10 yrs (90%-ile)	1	3	9
Average NIPTS	0	1	4
Max Hearing Risk**	5%	N/A	N/A
85 dBA for 8 hours			
	Speech (.5,1,2)	Speech (.5,1,2,4)	4 kHz
Max NIPTS (90%-ile)	4 dB	7 dB	19 dB
NIPTS at 10 yrs (90%-ile)	2	6	16
Average NIPTS	1	3	9
Max Hearing Risk**	12%	N/A	N/A
90 dBA for 8 hours			
	Speech (.5,1,2)	Speech (.5,1,2,4)	4 kHz
Max NIPTS (90%-ile)	7 dB	12 dB	28 dB
NIPTS at 10 yrs (90%-ile)	4	9	24
Average NIPTS	3	6	15
Max Hearing Risk**	22.3%	N/A	N/A

* Values given are arithmetic averages obtained from predictions using the methods of Baughn, Passchier-Vermeer and Robinson (see text).

** 25 dB ISO Fence for Hearing Handicap (re ISO: 1964). Averaged from the methods of Baughn and Robinson (see text).

*** Not available.

Exposures to Continuous Noise for Periods Less Than 8 Hours

The risk to hearing in the case of daily exposures to on-going noise for periods (minutes to hours) less than 8 hours can be evaluated by calculating an equivalent continuous sound level, L_{eq} , provided that the noise is approximately steady-state and is free from impulsive components. The calculation of L_{eq} normalizes the daily exposure to a duration of 8 hours for the purpose of Table 5-1.

Allowances for Level Fluctuation or Interruption of Noise

The 73 International Organization for Standardization (ISO, 1970), in its current Draft Recommendation (ISO/DR 1999) for assessing noise exposure at work, recommends a method that embodies the A-weighted equal-energy rule, namely the previously mentioned computation of an equivalent continuous sound level L_{eq} in dBA (see Figure 5-1). This method is probably the best available method of predicting the effects of noise on hearing in the case of continuous noise for which the level fluctuates slowly (seconds to hours) during the working day. It may, with circumspection, be extrapolated to cover distributed noise of fluctuating levels that go on for longer than the typical working exposure of 8 hours. The fluctuation in level must be non-impulsive; i.e., slow enough to be followed by a standard sound level meter on the "slow" setting.

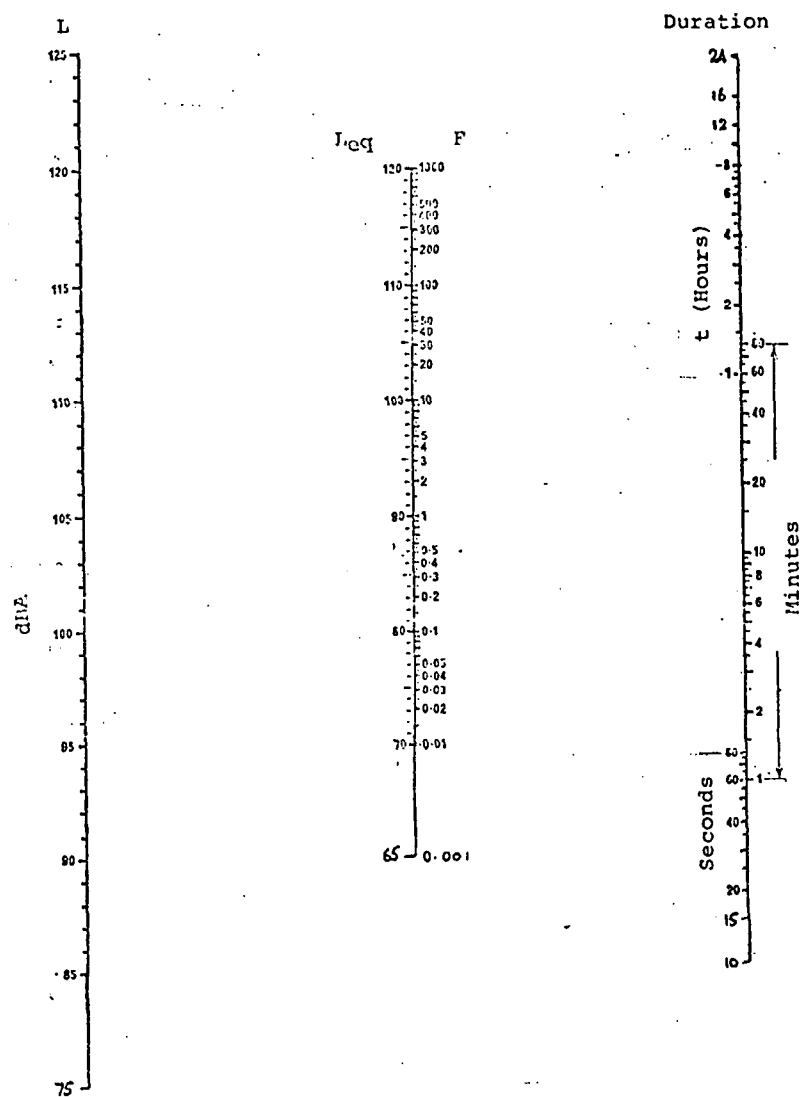
The arbitrary ISO protective weighting of 10 dBA for impulsiveness in the noise is open to question. Recent work by Passchier-Vermeer⁶⁶ has indicated that this figure may not be realistic in the case of distributed industrial noise with impulsive components. However, her work does in general confirm the validity of the equivalent level method based on equal-energy in the case of ongoing noise with slow but not impulsive fluctuations.

In the case of slowly varying levels in continuous noise with a rate of change less than 40 dB/second, it is appropriate to determine the equivalent continuous sound level, L_{eq} , in dBA and to enter the tables at the resulting value when evaluating the hazard or risk of NIPTS due to on-going noise.

Intermittent Noise

It is reasonable to treat intermittent exposure to steady-state nonimpulsive noise as a special case of fluctuating level. Intermittent noise is generally regarded as sound undergoing a substantial change in level from some potentially hazardous level to a very low level (below 55 dBA).

Such intermissions are known to be protective, probably by allowing recovery of normal physiological functions in the auditory system. Because there is no evidence for a threshold of noxiousness of noise so far as the hearing organ is concerned, it is desirable that the noise during any period of relative quiet be measured and included in the computation of L_{eq} . Intermittent noise may thus be treated in the same way as noise of varying level and may be equated analytically with continuous noise for the purpose of predicting hazard or risk.



NOTE: For each noise exposure, connect sound level, L, in dBA with exposure duration, t, and determine fractional exposure, F, from the scale at center, right. Determine total F by summing all values received in day. Read off value of L_{eq} from scale at center, right.

Figure 5-1. Nomogram for calculation of L_{eq} equivalent continuous sound level (from $F = \frac{t}{8} \text{antilog } [0.1(L-90)]$, where t is in hours and F is fractional exposure value)

However, Ward¹¹ has drawn attention to the particular weakness of the evidence in relation to intermittent exposure, pointing out that the equal-energy rule makes no allowance for different patterns of recovery from TTS in different patterns of intermittency. For example, the rule cannot distinguish the effect of a single 2-hour exposure from two 1-hour exposures to the same noise with variable amounts of intervening quiet.

In fact, a number of factors may affect the auditory tolerance of intermittent noise exposure. These include the number and duration of interruptions;^{51,74} the relationships between continuous and intermittent noise exposures;^{75,77} and possibly the level of noise below 80 dBA during the interruption.

Factors Influencing Incidence of NIPTS

Factors influencing the incidence of NIPTS are listed in Table 5-2. The table shows that some factors appear to increase the risk of NIPTS while others decrease it; and that some, while they may be significant factors determining group hearing levels measured in population surveys, show no clear evidence of being related casually to NIPTS.

TABLE 5-2
EFFECT OF VARIOUS FACTORS ON INCIDENCE OF NIPTS

FACTOR	INCREASES	DECREASES	NO SIGNIFICANT EFFECT
Age	?	?	
Sex			?
Nationality			?
Race			?
Physiological state:			
i. General Health	?	?	
ii. Activity			+
iii. Defensive Mechanisms*		+	
Prolonged exposure	+		
Interrupted or modulated exposure		+	
Ear protection		+	
Adverse environments:			
i. Vibration + noise			?
ii. Hypoxic states	?	?	
iii. Ototoxic drugs	?	?	
"Public awareness"		+	

*Principally the acoustic reflex

Factors Increasing the Risk of NIPTS

The only factor known to increase the likelihood of a person developing NIPTS is increased exposure to hazardous noise. Although it is possible that the older ear may be more susceptible than the younger ear, such a phenomenon is difficult to distinguish epidemiologically, and the question of age-enhanced susceptibility to NIPTS remains open. The hypothesis that certain defects or diseases of the ear, or a poor general state of health might increase predisposition to NIPTS remains to be proven. There is some evidence that certain ototoxic drugs may act synergistically with noise to damage the hearing organ.⁷⁹ (This subject is discussed in detail in Section 9.) However, Glorig and Nixon's¹²⁵ contention that aging and noise exposure alone determine group hearing levels in otologically healthy members of the general American population has received support from more recent data and from industrial experience in other Western countries, notably, the United Kingdom (Burns and Robinson,¹⁴ and Robinson¹⁷).

Factors Mitigating Risk

Physiologically, the acoustic reflex is known to protect, to a limited degree, hearing against noise. This mechanism was discussed in detail in Section 4.

The use of artificial ear protection (earplugs, earmuffs and kindred devices) substantially decreases the risk of NIPTS but this again is a difficult factor to allow for in predictive formulas, because the use of ear protection (especially in non-occupational noise exposure situations) is neither universal nor uniform. In this connection, however, it is reasonable to presume that, as the population at large is made increasingly aware of the hearing hazard from noise, the public response (e.g., use of ear protector as well as noise-avoidance and noise reduction) will be reflected in a decreasing incidence of NIPTS attributable to environmental noise.

Factors not Directly Affecting Susceptibility to NIPTS

Differences Related to Sex

Ward⁷¹ investigated various aspects of NIPTS in relation to sex differences, finding that, whereas men were more susceptible to TTS following low-frequency (less than 700 Hz) sounds, they were less susceptible than women to high-frequency (greater than 2000 Hz) exposures. Women also appeared to show a greater benefit (in terms of reduced TTS) from intermittency in the noise exposure. Ward has suggested another explanation for these findings, namely, that females have a more efficient acoustic reflex than males. However, evidence for sex-linked differences in the fragility of the hearing organ (or fatigability of the auditory nerve by noise) was negative in this study.

Generally, it can be argued that intrinsic differences between the sexes are of no practical significance in relation to hearing hazard in noisy environments, or in relation to the setting of hearing damage-risk criteria.

Differences Related to Urban Environment

Certain primitive people, living in remote areas of the world in which they are not exposed to the constant din of mechanized civilization, have been found to have unusually sharp hearing in comparison with urban populations of corresponding ages: in this connection particular attention has been given to the Maba'an people of Sudan. But it is debatable whether such audiometric differences are due to the lack of noise exposure alone, for many factors (including cultural, genetic and general environmental differences) may underlie differences in the pattern of hearing found between dissimilar communities who are widely separated geographically and culturally.^{81,82}

Differences Related to Age

Although it has been suggested that older people are more susceptible to NIPTS⁸³ it is debatable whether individual susceptibility to noise-induced hearing loss changes appreciably with age. Some authors have contended that young ears are more susceptible to noise damage (more "tender") than older ones.^{84,86}

The evidence, however, is inconclusive, having in some studies been confounded by non-occupational influences (e.g., noise-exposure in military service) that were not the same for the age-groups compared. Recent studies^{87,88} indicate that there is probably no casual relationship between age *per se* and susceptibility to NIHL, at least in men of working age. This view is supported by the work of Loeb and Fletcher.⁸⁹

That the effect of age on hearing is very difficult to distinguish audiometrically from the influence of noise exposure and related environmental variables is evident from data summarized by Burns and Robinson¹⁴ and from several studies dealing with or touching on noise susceptibility as a function of age.

DAMAGE-RISK CRITERIA

That there is a time/intensity tradeoff for hazardous steady-state noise is well established, but this has been embodied in existing criteria in different ways. The tradeoff is not a simple one and differing theories underlie the various damage risk criteria currently in use. The picture is complicated when the noise exposure is intermittent, which is frequently the case in practice. Evidence from TTS experiments generally supports the view that the effect of intermittent exposures to high levels of noise separated by relative quiet is less than the effect of the same total noise exposure received unbroken.⁹⁰ Moreover, the generation of a given TTS by continuous noise requires progressively less time as the exposure level is increased.

The CHABA Criterion

The CHABA DRC was based on such observations; its principal assumption was that, for a given octave of frequency, all noise exposures producing the same TTS_2 are equally likely to produce a given PTS (Kryter, Ward, *et al.*⁹¹). This criterion, in which the trade-off between time and intensity varies (e. g., between 2 and 7 dB per doubling of time for the 1200-2400 Hz band), represented a departure from the simple adoption of the "equal-energy" rule (3 dB per doubling of time) seen in earlier criteria (such as AFR 160-3¹). The resulting differences between DRC's are illustrated in Table 5-3 which compares simply the limiting values for continuous exposure to an octave band of noise from 1200 to 2400 Hz in CHABA and AFR 160-3 criteria. The latter is more conservative for nearly all durations.

TABLE 5-3
COMPARISON OF CHABA DAMAGE RISK CRITERIA AND AFR 160-3

Exposure time	8h	4h	2h	10 min	5 min
CHABA	85	87		105	112 dB
AFR 160-3	85	88	91		105 dB

The 5 dB rule adopted under the Walsh-Healey Act in 1969 (Federal Register 34, (96): 7948-7949 (May 20, 1969) appears to have been an expedient compromise: it has some justification in that it effectively makes an allowance for intermittency.

Criteria for Steady-State Noise

There is generally firm agreement that, for typical 8-hour everyday exposures to continuous industrial noises, levels below 80 dBA are, for most hearers innocuous. Also, as the noise level increases, an increasing number of people are put in risk, and the average magnitude of hearing loss grows commensurately. This picture is well supported by a number of substantial

audiometric surveys of industrially exposed people in the United States and elsewhere (Baughn,⁶⁵ Rasschier-Vermeer,^{23, 66} Robinson¹⁷). Based on such evidence, a recent DRC, provided for in 1969 under the Walsh-Healey Act governing the welfare of workers under public contracts, was adopted in the United States. This allows 90 dBA for continuous 8-hour exposures.

“AAOO” and Cognate Criteria

It is a basic premise of these criteria that the chief (a rigorous interpretation might say the sole) function of human hearing is to receive speech signals. Arguing that telephoned speech (band-limited to some 300 to 3000 Hz) is generally intelligible, Fletcher⁹² introduced his “point-eight” rule for evaluating hearing damage in accordance with this philosophy. This led to the practice of averaging hearing levels at 500, 1000, and 2000 Hz.

The AAOO and cognate rules attempt, *inter alia*, to find pragmatic answers to the following questions:¹¹

1. How much hearing loss must occur before the person affected notices any difficulty?
2. What values of HL constitute complete loss of hearing?
3. What is the relative importance of different audiometric frequencies?
4. How important is it to have two working ears?

The Intersociety Committee (1970) Guidelines

A group of professional associations (The American Academy of Occupational Medicine; American Academy of Ophthalmology and Otolaryngology; American Conference of Governmental Industrial Hygienists; Industrial Hygiene Association; and Industrial Medical Association) concerned with industrial noise recently revised some previously published guidelines intended “. . . to aid industrial management and official agencies in establishing effective hearing conservation programs.” The document has also defined hearing impairment as an average threshold level in excess of 15 dB (ASA-224.5–1951) which is equivalent to 25 dB, ISO: 1964 at 500, 1000 and 2000 Hz. The guidelines were intended to prevent the development of that portion of permanent hearing loss due to occupational exposure to steady-state noise, continuous or intermittent.

The evaluation of noise in dBA using standard meters and procedures was recommended by the Committee, as was the determination or estimation of the total time and temporal distribution of noise exposure “throughout the working day.” The guidelines, subject to revision, contain numerical data and procedures for rating the auditory hazard of occupational noise exposure in terms of risk as a function of age, noise level and exposure time. Overall, the Committee in 1970 deemed 90 dBA for 8 working hours of steady-state noise daily, with a permissible increase of 5 dBA (up to a permissible maximum of 115 dBA) for each halving of exposure time, to be a “reasonable objective for hearing conservation.” It was pointed out explicitly that the rating procedure applies only to groups, not to individuals.

The document included some general guidance on methods of noise control for hearing conservation in industry and some recommendations concerning audiometry in industrial settings. The recommended audiometric frequencies adopted by the Intersociety Committee were 500, 1000, 2000, 2000, 4000 and 6000 Hz. The guidelines are subject to triennial review and revision.

Use of A-Weighted Decibels

The Intersociety Committee on Guidelines for Noise Exposure and Control, influenced mainly by the work of Baughn⁶⁵ in the USA and Robinson¹⁷ in the United Kingdom, decided to recommend the use of dBA to yield a single-number rating of continuous noise hazard.⁹³ This unit, as recommended in this document, has a number of advantages, including convenience of measurement using standard sound level meters; and it can, incidentally, be easily related to the ISO standardized NR numbers using the approximate difference of 5 decibels ($\text{dBA} \approx \text{NR} + 5$). Measurements on the A-weighting scale may, however, underestimate hazard to hearing when the noise contains a strong tonal component^{1,108} or a markedly uneven spectrum.

Index of Cumulative Noise Exposure—Robinson's "Sound-Immission" Rating

Robinson¹⁷ and Robinson and Cook³⁹ contended that NIHL is expressible in terms of a composite noise exposure measure (noise or sound "immission") that is proportional to the total frequency-weighted sound energy received by the ear over a designated exposure period. Robinson and Cook³⁹ have presented industrial hearing level and noise exposure data in support of this predictive model. The data is valid for 8-hour daily exposures from 1 to 600 months (50 years), to industrial-type noise at levels ranging from 75 to 120 dBA.

Inadequacy of Conventional "Speech Frequencies" Assessment

Harris¹⁰⁹ has contended that the widely adopted convention of using the average pure-tone auditory sensitivity at 500, 1000, and 2000 Hz to predict a person's ability to understand everyday speech may not be adequate when, as is often the case, the speech is of poor quality, is interrupted, is distorted, or is noise-masked. From a study of speech intelligibility among 52 subjects with sensorineural hypoacusis, listening to various kinds of degraded speech, he concluded that a better assessment of hearing disability for realistic everyday speech is obtained when the audiometric frequencies 1, 2 and 3 kHz are used instead, as is the convention in British practice. This supports a finding of Kryter, Williams and Green,⁹⁵ who reported that the triad 2, 3 and 4 kHz was the best predictor of speech reception for phonetically balanced words (not sentences) in subjects with high-tone hearing losses. However, they recommended as a compromise a triad similar to Harris's in view of the already well-established AMA convention of 500, 1000 and 2000 Hz. Kryter and his co-workers⁹⁵ showed that some speech

tests and methods of hearing evaluation hitherto adopted introduce a bias that is apt to lead to underestimation of the importance of auditory sensitivity at frequencies above 2 kHz. Some authorities, notably the state of California, include 3000 Hz in the assessment of disability.

Impulsive Noise

Kryter¹⁵ has adduced evidence from his own and other recent work to show that TTS_2 at 4000 Hz and, by implication, the risk of NIPTS, can in many circumstances be predicted with fair accuracy from a knowledge of the peak overpressure, spectral composition and number of impulses. For the noise of gunfire, Kryter maintains that damage risk to hearing can be evaluated from the peak overpressure and number of impulses. An important assumption implicit in this data is that a given TTS_2 will eventually lead to an equal NIPTS.

Some procedures proposed by Kryter¹⁵ and others for predicting damage risk to hearing due to gunfire and similar noises have been summarized elsewhere.¹² The risk to hearing from such noise depends primarily upon the peak overpressure and the number of impulses experienced and to some degree upon the spectral and temporal characteristics of the noise. Although, in general terms, the pattern of NIHL produced by impulsive noise is similar to that produced by steady-state noise, namely, loss beginning and advancing most rapidly at 4 kHz and above, the different stimulus parameters call for rather different criteria and methods for evaluating impulse noise. For this reason the current ISO Recommendation (ISO, 1971) on the assessment of occupational noise-exposure for hearing conservation purposes states specifically that the method is not applicable to such noises.

Impulse Noise and TTS

In 1962, Ward¹¹¹ argued that damage-risk criteria for impulsive noise should best be expressed in terms of the number of impulses rather than exposure time *per se*. The importance of number of impulses has again, more recently, been brought out by Coles, *et al.*,^{64, 96} Ward's argument was based on his observations that the TTS in the range 500 to 13000 Hz (and, by implication, the PTS) produced by impulse noise is relatively independent of the interval between pulses—at least for intervals in the range 1 to 9 seconds (a 30-second interval, however, apparently permitted slight recovery between stimuli).

Impulses With an Oscillatory Component

When the impulse contains an oscillatory component ("Type B" of Coles, *et al.*⁶⁴), the assumptions of Kryter¹⁵ applying to simple, Type A gun noise may require modification, and spectral information may be needed in the evaluation of hazard, in addition to a knowledge of the peak pressure, number, and temporal spacing of impulses (Coles, *et al.*,⁶⁴ Kryter,¹⁵

Ward;⁹⁷ Ward, *et al.*⁹⁸ (CHABA); Ward, Selters and Glorig⁹⁹). Oscillatory waveforms can be recorded from gunshots fired in reverberant areas and from other sources of impulsive noise. It has been argued that even spike impulses must generate an oscillatory component upon entering the ear, by exciting the resonances of the ear canal and middle ear structures.¹⁰⁰ This would in part explain the general similarities between the patterns of threshold shift produced by both impulsive and distributed steady-state noise.

SUMMARY – NOISE-INDUCED HEARING LOSS-TEMPORARY AND PERMANENT SHIFTS IN AUDITORY THRESHOLD FOLLOWING NOISE EXPOSURE

Ongoing noise has been proven to cause permanent hearing loss in industrial settings and among young people exposed to loud music over extended periods of time. Noise is also known to cause temporary hearing loss and ringing in the ears (tinnitus).

However, since there is a relative lack of information about the effect of shorter-term intermittent or incomplete daily exposures, several theories have been postulated to relate noise exposure to hearing loss in these situations.

One theory that has been fairly widely used is the Equal-Energy Hypothesis, which postulates that hearing damage is determined by the total sound energy entering the ear on a daily basis.

Another theory suggests that the long term hazard is predicted by the average temporary threshold shift produced by daily noise exposures. There is evidence to support both of these theories within reasonable limits of extrapolation.

Impulsive noise (such as gunshots) has also been shown to cause damage. CHABA has recently developed a noise hazard numerical weighting system that takes into account such factors as intensity, duration, and number of noise impulses.

Averaging the NIPTS predictions over various industrial noise hazard prediction methods gives a fairly dependable measure of the hearing risk of noise-exposed populations. Hearing damage has been noted at levels as low as 75 dBA after 10 years.

The only important factor in increasing hearing risk appears to be noise exposure, and artificial ear protection devices do appear to be of value in preventing damage. Neither sex-related nor cultural differences appear to significantly affect hearing risk due to noise-exposure.

It is evident from the noise exposure data that noise can damage hearing and can cause both NITTS and NIPTS. The relationship between noise exposure and hearing loss is well understood in industrial settings and in the case of high intensity impulsive sound (i.e. gunshots). However, in the case of fluctuating or intermittent noise, data is generally lacking and it is necessary to rely on data extrapolations to estimate effects.

REFERENCES

1. Anon. Hazardous Noise Exposure. Air Force Redulation 160-3, Department of the Air Force, 1956.
2. BENOX (Biological effects of Noise) Group (Ades, H. W. & Colleagues) (1 December 1953). *BENNOX REPORTS: An Exploratory study of the biological effects of noise*. Univ Chicago: ONR Proj NR 144079.
3. Botsford, J. H. (1970). Damage Risk. In: *Transportation Noises: A Symposium on Acceptability Criteria*. Chalupnik, J. D. (ed.) Seattle Univ. Washington Press. pp. 103-113.
4. Environmental Protection Agency. PROCEEDINGS OF THE INTERNATIONAL CONGRESS ON NOISE AS A PUBLIC HEALTH PROBLEM (W. D. Ward, ed.) In press, 1973.
5. Ward, W. D., Glorig, A & Sklar, Diane L (June 1959). Temporary threshold shift produced by intermittent exposure to noise. *J acoust Soc Amer*, 31 (6), 791-794.
6. Ward, W. D., Glorig, A. & Sklar, D. L. (October 1958). Dependence of temporary threshold shift at 4 kc on intensity and time. *J. Acoust. Soc. Amer.*, 30 (10), 944-954.
7. Ward, W. D. (January 1960). Latent and residual effects in temporary threshold shift. *J. Acoust Soc. Amer.*, 32, (1), 135-137.
8. Sataloff, J. Vassallo, L. & Menduke, H. (June 1969). Hearing loss from exposure to interrupted noise. *Arch Environ Health*, 18, 972-981.
9. Cohen, A, Anticaglia, J. R. & Jones, H. H. (May 1970). Noise-induced hearing loss: exposures to steady-state noise. *Arch Environ Health*, 20, 614-623.
10. Kryter, K. D. (April 1966). *Review of research and methods for measuring the loudness and noisiness of complex sounds*. NASA Contractor Rpt No. NASA CR-422.
11. Ward, W. D. (March 1970). Temporary threshold shift and damage-risk criteria for intermittent noise exposures. *J acoust Soc Amer*. 48 (2 part 2), 561-574.
12. Environmental Protection Agency, U. S. Airforce Aerospace Medical Research Laboratory, A SCIENTIFIC BASIS FOR LIMITING NOISE EXPOSURE FOR PURPOSES OF HEARING CONSERVATION. EPA Document Number EPA-550/9-73-001 (in press.) 1973.

13. Burns, W. NOISE AND MAN. Philadelphia: Lippencott (1969).
14. Burns, W & Robinson, D. W. (1970). *Hearing and noise in industry*. London: Her Majesty's Stationery Office, 43 pp + 16 Appendices.
15. Kryter, Karl THE EFFECTS OF NOISE ON MAN. New York: Academic Press (1970).
16. Anon. Occupational Safety and Health Standards. Occup. Safety and Health Admin, DOL, Vol. 36, p. 105, 1971.
17. Robinson, D. W. Estimating the Risk of Hearing Loss due to Continuous Noise. In Occupational Hearing Loss, D. W. Robinson, Ed. Academic Press, N. Y., 1971.
18. King, P. F. (1971). Hearing conservation in aircrew and ground support personnel. *Aeromedical aspects of vibration & noise*, Guignard, J. C. & King, P. F. AGARD-ograph 151. . Paris: NATO/AGARD. Part 2, Ch. 10-23, pp 204-257.
19. Anon. Criteria for a Recommended Standard - - - Occupational Exposure to Noise, NIOSH Department of Health, Education and Welfare (1972).
20. Gallo, R & Glogig, A (1964). Permanent threshold shift changes produced by noise exposure and aging. *Amer Ind. Hyg. Assoc. J.* 25, 237-245.
21. Taylor, W. Pearson, J. Mair, A & Burns, W. (December 1965). Study of noise and hearing in jute weaving. *J Acoust Soc Amer.* 38, 113-120.
22. Hinchcliffe, R. (1959). The threshold of hearing as a function of age. *Acoustica*, 9, 303-308.
23. Passchier-Vermeer, W. (April 1968). *Hearing loss: due to exposure to steady-state broadband noise*. Instituut Voor Gezondheidstechniek, Sound & Light Division, Rpt 35, 18 pp.
24. Summar, T. and Fletcher, John, 1965 (see related EPA document²³).
25. Vacated
26. Kell, R. L., J. C. G. Pearson, W. I. Acton, and W. Taylor in *Occupational Hearing Loss*, D. W. Robinson (Ed.), (Academic Press, London, 1971) pp. 179-191.
27. Lebo, C.P. and Oliphant, D. S. Music as a source of acoustic trauma. *Laryngoscope*, 78, 1968.
28. Rintelmann, W. F. and Borus, J. F. Noise-induced hearing loss and rock and roll music. *Arch. Otolaryngol.* 88, 1968.
29. Flugrath, J. M. (November 1969). Modern day rock-and-roll music and damage-risk criteria. *J acoust soc Amer.* 45 (3), 704-711.
30. Lipscomb, David M. High intensity sounds in the recreational environment: a hazard to young ears. *Clin. Pediat.* 8 (2) 63-68 (1969).

31. Lipscomb, David M. Ear damage from exposure to rock and roll music. *Arch. Otol.* 90: 545-555 (1969).
32. Dey, F. L. Auditory fatigue and predicted permanent hearing defects from rock-and-roll music. *New Eng. J. Med.* 282 (9): 467-470 (1970).
33. Fletcher, John L. High-frequency hearing and noise exposure. Presented to the International Congress on Noise as a Public Health Problem, Dubronik, Yugoslavia (1973).
34. Wood, W. Scott and Lipscomb, David M. Maximum available sound-pressure levels from stereo components. *JASA*, 52 (2) 484-487 (1972).
35. Fletcher, J. L. (13 October 1972). *Effects of non-occupational noise exposure on a young population*. Rpt for NIOSH, HEW, HSM 099-71-52. 18 pp.
36. Smitley, Ellen K & Rintelmann, W. F (April 1971). Continuous versus intermittent exposure to rock and roll music: effect upon temporary threshold shift. *Arch Environ Health*, 22, 413-420.
37. Burns, W & Robinson, D. W. (1970). *Hearing and noise in industry*. London: Her Majesty's Stationery Office, 43 pp + 16 Appendices.
38. Burns, W. The relation of Temporary Threshold Shift in Individuals. In OCCUPATIONAL HEARING LOSS, D. W., Robinson, (ed.) London: Academic Press, 1971.
39. Robinson, D. W. & Cook, J. P. (1968). *The quantification of noise exposure*. Teddington, Middlesex, England: National Physical Laboratory draft dated 14 June 1968. 8 pp.
40. Luz, G. A. & Hodge, D. C. (October 1970). Recovery from impulse-noise induced TTN, in monkeys and men: a descriptive model. *J acoust Soc Amer*, 49, (6 part 2), 1770-1777.
41. Ward, W. D. and D. A. Nelson. On the Equal-Energy Hypothesis Relative to Damage-Risk Criteria in the Chinchilla. In Occupational Hearing Loss, D. W. Robinson, Ed. Acad. Press., N. Y., 1971.
42. Eldredge, D. and Covell (see related EPA document ²³).
43. Miller, J. D., Watson, C. S., and Covell, W. (1963) "Deafening Effects of Noise on the Cat," *Acta Oto-Laryngol. Suppl.* 176, 91 pp.
44. Miller, J. D., Rothenberg, S. J. & Elderedge, D. H. (June 1971). Preliminary observations on the effects of exposure to noise for seven days on the hearing and inner ear of the chinchilla. *J. Acoust. Soc Amer*, 50, (4 part 4) 1199-1203.

45. Lipscomb, David M. Theoretical consideration in the rise of high frequency hearing loss in children. Presented to the International Congress on Noise as a Public Health Problem. Dubrovnik, Yugoslavia (1973).
46. Carder, H. M. & Miller, J. D., (September 1972). Temporary threshold shifts from prolonged exposure to noise, *J. Speech & Hearing Res.*, 15 (3), 603-623.
47. Mills, J. H., Gengel, R. W., Watson, C. S. & Miller, J. D., (August 1970). Temporary changes of the auditory system due to exposure to noise for one or two days. *J. Acoust Soc Amer*, 48, (2 part 2), 524-530.
48. Mills, J. H. (1973), "Temporary and Permanent Threshold Shifts Produced by Nine-Day Exposures to Noise," *J. Speech Hear. Res.* (Submitted).
49. Price, G. R. (July 1968). Functional changes in the ear produced by high-intensity sound. I. 5.0 KHz stimulation. *J. Acoust Soc Amer.*, 44 (6), 1541-1545.
50. Peters, E. N. (May 1965). Temporary shifts in auditory thresholds of chinchilla after exposure to noise, *J. Acoust Soc Amer.*, 37, (5), 831-833.
51. Ward, W. D., Glorig, A & Selters, W. (February 1960). Temporary threshold shift in a changing noise level. *J. Acoust Soc Amer*, 32 (2) 235-237.
52. Price, G. R. (September 1971). Functional changes in the ear produced by high-intensity sound. II. 500-Hz stimulation. *J. Acoust Soc Amer*, 51 (2 part 2), 552-558.
53. Poche, L. B., Stockwell, C. W. & Ades, H. W. (November 1968). Cochlear hair-cell damage in guinea pigs after exposure to impulse noise. *J. Acoust Soc Amer*, 46 (4 part 2). 947-951.
54. Trittipoe, W. J. (November 1958). Residual effects of low noise levels on the temporary threshold shift, *J. Acoust Soc. Amer.*, 30, (11), 1017-1019.
55. Ward, W. D. (April 1960). Recovery from high values of temporary threshold shift. *J. acoust. Soc. Amer.*, 32, (4), 497-500.
56. Karlovich, R. S. & Luterman, B. F. (August 1969). Application of the TTS paradigm for assessing sound transmission in the auditory system during speech production. *J. Acoust Soc. Amer*, 47, (2 part 2), 510-517.
57. Kryter, K. D. and G. Garinther (1966). "Auditory Effects of Acoustic Impulses from Firearms," *Acta Oto-Laryngol. Suppl.* 211.
58. Acton, W. I. (1967). A review of hearing damage risk criteria. *Ann Occup Hyg*, 10, 143-153.
59. Coles and Rice (1970) - see related EPA document ²³
60. Coles and Rice (1971) - see related EPA document ²³

61. Coles (1971) - see related EPA document²³
62. Loeb, M & Fletcher, J. L. (August 1968). Impulse duration and temporary threshold shift. *J. acoust Soc. Amer.*, 44, (6), 1524-1528
63. Gjaevenes, K. (1967). Damage-risk criterion for the impulsive noise of "toys". *J acoust Soc Amer*, 42 (1), 268.
64. Coles, R. R. A., Garinther, G. R., Hodge, D. C. & Rice, C. G. (1968) Hazardous exposure to impulse noise. *J acoust Soc Amer*, 43 (2), 336-343.
65. Baughn, W. L. (September 1966). Noise Control - percent of population protected. *International Audiology*, 5 (3), 331-338.
66. Passchier-Vermeer, W. "Steady-State and Fluctuating Noise: Its Effects on the Hearing of People" in Occupational Hearing Loss, D. W. Robinson. Ed. Academic Press, N. Y., 1971.
67. Kryter, Karl. Impairment to hearing from exposure to noise. *JASA*, 53 (5) 1211-1234, 1973.
68. Cohen, Alexander. Some general reactions to Kryter's paper "Impairment to hearing from exposure to noise. *JASA*, 53 (5) 1235-1236 (1973).
69. Davis, Hollowell. Some comments on "Impairment to hearing from exposure to noise by K. D. Kryter. *JASA*, 53 (5) 1237-1238 (1973).
70. Lempeer, Barry L. Technical aspects of Karl Kryter's paper "Impairment to hearing from exposure to noise" with respect to the NIOSH statistics. *JASA*, 53 (5) 1239-1241 (1973)
71. Ward, E. Dixon Comments on "Impairment to hearing from exposure to noise" by K. D. Kryter, *JASA*, 53 (5) 1242-1243 (1973).
72. Kryter Karl, Reply to the critiques of A. Cohen, H. Davis, B. L. Lember, and W. D. Ward of the paper "Impairment to hearing from exposure to noise" *JASA* 53 (5), 1244-1254. (1973).
73. Anon. "Assessment of Occupational Noise Exposure for Hearing Conservation Purposes," ISO Recommendation R1999 pp. 1-11, Switzerland (May 1971).
74. Schmidek, M. Henderson, T & Margolis, B (December 1970). *Evaluation of proposed limits for intermittent noise exposures with temporary threshold shift as a criterion*. Cincinnati: HEW, NIOSH, National Noise Study, 10 pp.
75. Ward, W. D. (1970). Hearing Damage. In: *Transportation Noises: A symposium on acceptability criteria*. Chalupnik, J. D. (ed). Seattle & London: Univ Washington Press, pp. 174-186.

76. Ward, W. D., Glorig, A & Sklar, Diane L (June 1959). Temporary threshold shift produced by intermittent exposure to noise. *J acoust Soc Amer*, 31 (6), 791-794.
77. Cohen, A. & Jackson, E. (1968). Threshold shift in hearing as a function of band-width and mode of noise presentation. *J. Auditory Res*, 8, 401-414.
78. Schmidek M. Margolis, R & Henderson, *Effects on the level of noise interruptions on temporary threshold shift*. Cincinnati: HEW, Public Health Service, NIOSH rpt, 8 pp.
79. Falk, S. A. Combined effects of noise and ototoxic drugs. Environmental Health Perspectives. Experimental Issue Number Two, October, 1972 (USPHS, NIH), 5-22.
80. Ward, W. D. (1966). Temporary threshold shift in males and females. *J acoust Soc Amer*, 40 (2), 478-485.
81. Rosen, S. & Rosen, Helen V. (1971). High frequency studies in school children in nine countries. *The Laryngoscope*, 81, 1007-1013.
82. Rosen, Samuel, Bergman, Moe, Plester, Daniel, El-Mofty, A. and Satti, M. H. Presbycusis study of a relatively noise-free population in the Sudan. *Ann. Otol.* 71:727, 743 (1962).
83. Kryter, Karl Damage-risk Criteria for Hearing. In NOISE REDUCTION, Leo Baranek, ed. New York: McGraw-Hill (1960).
84. Schwartz (1962) - see related EPA document ²³
85. Kupp (1966) - see related EPA document ²³
86. Nowak and Kahl (1969) - see related EPA document ²³
87. Julse and Partsch (1970) - see related EPA document ²³
88. Schneider, E. J., Mutchler, J. E, Hoyle, H. R., Ode, E. H. & Holder, B. B. (1970). The progression of hearing loss from industrial noise exposures. *Amer. Ind. Hygiene Assoc. Jour*, 31, May-June, 368-376.
89. Loeb, M. and Fletcher, John. Temporary threshold shift for "normal" subjects as a function of age and sex. *J. Aud. Res.* 3, 65-72 (1963).
90. Ward, W. Dixon Effect of temporal spacing on temporary threshold shift from impulses. *JASA.* 34, 1230-1232 (1962).
91. Kryter, K. D. et al (1965). *Hazardous exposure to intermittent and steady-state noise*. Rpt of Working Group 46, NAS-NRC Comm on Hearing, Bioacoustics & Biomechanics, ONR Cont No. NONR 2300 (05), 30 pp.

92. Fletcher, H. and Steinberg, J. C. Loudness of Complex sounds. *Phys. Rec.* 24 (1924).
93. Mercer, D. M. A. (September 1967). Noise-damage criterion using A-weighting levels. *J. Acoust Soc. Amer.*, 43, (3), 636-637.
94. Harris, J. D. (May 1965). Pure-tone acuity and the intelligibility of everyday speech. *J. Acoust Soc. Amer.*, 37 (5), 824-830.
95. Kryter, K. D., Williams, C. & Green, D. M. (September 1962). Auditory acuity and the perception of speech. *J. acoust Soc. Amer.*, 34 (9), 1217-1223.
96. Coles et al (1970 - see related EPA document²³)
97. Ward, W. D. (September 1962). Effect of temporal spacing on temporary threshold shift from impulses. *J. acoust Soc. Amer.*, 34 (9), 1230-1232.
98. Ward, W. D. (ed.) PROPOSED DAMAGE-RISK CRITERION FOR IMPULSE NOISE (GUNFIRE). Report of Working Group 57, NAS-NRC Committee on Hearing, Bioacoustics, and Biomechanics (1968).
99. Ward, W. D. Selters, W. & Glorig, A (June 1961). Exploratory studies on temporary threshold shift from impulses. *J. acoust Soc. Amer.*, 33 (6), 781-793.
100. Muirhead, J. C. (July 1960). Hearing loss due to gun blast. *J. acoust Soc. Amer.*, 32, 885.

SECTION 6

MASKING AND SPEECH INTERFERENCE

The one effect of noise of which every person is aware is its interference with the understanding of speech. Technically speaking, such interference is only one aspect of the general phenomenon of "masking" -- an interaction of two acoustic stimuli whereby one of them:

1. Changes the perceived quality of the other.
2. Shifts its apparent location or loudness.
3. Makes it completely inaudible.

Much information has become available over the past 50 years concerning the masking of fairly simple signals such as pure tones, noise bands and nonsense syllables by noises of various spectra, and general laws have been developed that will allow rather accurate prediction of whether or not a given speech sound will be masked by a particular noise. Recent reviews of masking in general have been presented by Jeffress¹ and Scharf². Both Webster^{3, 4} and Kryter⁵ summarize much of the evidence concerning the masking of individual speech sounds by noise.

INTELLIGIBILITY OF SPEECH

Unfortunately, most of this specialized knowledge is often of limited assistance in the prediction of the intelligibility of "ordinary speech" -- speech as it actually occurs in real life. Ordinary speech consists of a complicated sequence of sounds whose overall intensity and spectral distribution are constantly varying. Because of this lack of uniformity, some sounds will be masked by a specific steady noise while others will not. Furthermore, even in a steady noise, the energy in different frequency regions fluctuates from moment to moment; therefore, a sound that might be masked at one instant could be clearly perceptible the next. Finally, it is not usually necessary for the listener to hear all the speech sounds in a sentence because ordinary speech is very redundant -- that is, it contains more information than is necessary for understanding. The listener decodes the speech by a synthesizing process, only partly understood at present, that depends not only on the acoustic cues but also on his knowledge of the language and of the context in which the speech occurs. For example, most people, although actually hearing only "She icked up the baby," would need no additional information in order to know what was actually said. Thus, even though one speech sound was missed completely, the sentence would have been correctly understood, and its intelligibility would be "100%."

For these reasons, the relations between the spectral and intensive characteristics of noise and the intelligibility of ordinary simultaneously-presented speech are rather complicated. Often they must be measured directly instead of being predicted on the basis of results with isolated words, although conversion charts have been constructed to transform scores on tests involving only words to the approximate expected scores for the sentences of ordinary discourse.

Many variables may influence the accuracy of speech communication from talker to listener in an experiment. In addition to the masking noise present at the listener's ear, all the following can be important:

- The characteristics of the talker.
- The test materials.
- The transmission path from talker to listener.
- The spatial locations of the talker, noise source, and listener.
- The noise level at the speaker's ear (if different from that at the listener's, particularly).
- The presence or absence of reverberation.
- The integrity of the listener's auditory system.

The outcome of experiments involving noise and speech is usually measured by the percentage of messages understood, and this percentage is taken as a measure of intelligibility or the "articulation score" of the speech. Other measures are occasionally used; among these are:

- Ratings of the quality or the naturalness of the speech.
- Recognition of the talker.
- Recognition of the personality traits.
- Psychological state of the talker.

MEASUREMENT OF SPEECH—INTERFERENCE

In describing speech interference, the noise concerned can be defined either in terms of its specific spectrum and level or in terms of any number of summarizing schemes. In addition to the average A-weighted sound level, the two most generally-used alternative methods of characterizing noises in respect to their speech-masking abilities are:

- The articulation index (AI).
- The speech interference level (SIL).

Articulation Index

The articulation index, initially developed by French and Steinberg⁶, although extended and somewhat simplified by Kryter⁷, is a very complicated measure that takes into account the fact that certain frequencies in the masking noise are more effective in masking than other frequencies. Determination of the AI involves:

1. Dividing the frequency range in which significant speech energy exists (250 to 7000 Hz) into 20 bands, each of which contributes 1/20 of the total intelligibility of speech.
2. Determining the difference between the average speech level and the average noise level (that is, the signal-to-noise ratio) for each of these bands.
3. Combining these numbers to give a single index.

This AI, by essentially predicting how much masking of specific speech sounds will occur, will therefore predict the intelligibility of “speech” at a given level in a specific noise. Simplified procedures for estimating the AI from measurements of octave-band levels have also been developed⁷. Although the AI is as yet the most accurate measurement to use in predicting the effects of noise on speech intelligibility, it is difficult to use and more difficult for laymen to interpret.

Speech Interference Level

The SIL, which was introduced by Beranek⁸ in 1947 as a simplified substitute for the AI, is an indication of only the average general masking capability of the noise. Contributions to intelligibility by the lowest and highest frequencies are ignored. As originally formulated, it was defined as the average of the octave-band SPLs in the 600-1200, 1200-2400 and 2400-4800-Hz octaves. Since that time, the preferred frequencies for octave bands have been changed. One modern version of the SIL is the average of the SPLs in the three octave bands centered at 500, 1000, and 2000 Hz. So many variations of SIL in terms of the specific octave bands to be averaged have been developed that a shorthand notation is now used. SIL (.5, 1, 2) is the average of the SPLs of the three octave bands centered at 500, 1000 and 2000 Hz; SIL (.25, .5, 1, 2) includes the 250-Hz band in the average, and so on. The original SIL would be SIL (.85, 1.7, 3.4) in this notation. At the present time, the American National Standards Institute is promoting the acceptance of SIL (.5, 1, 2, 4) as providing the best estimate of masking ability of a noise.

The simple A-weighted sound level is also a useful index of the masking ability of a noise. The A-weighting process emphasizes the median frequencies, as do the various SILs. However, in contrast to most SIL schemes, A-weighting does not ignore the lowest frequencies completely.

Experiments have shown that the AI is somewhat more accurate than any of the SILs or dBA (or other similar weighting schemes that were not developed specifically for speech) in predicting the speech-masking ability of a large variety of noises.⁹ Nevertheless, dBA and SIL ratings will continue to be used, because for most noises of importance, the advantage in accuracy of AI determinations does not outweigh the ease of measurement of dBA or SILs.

Noise Level, Vocal Effort, and Distance

Since much speech is spoken at a reasonably constant level, and in “ordinary” surroundings, it is possible to express many of the empirical facts about average speech communication in a

single graph. The basic data come from Beranek¹⁰, and are shown in Table 6-1. These are values of SIL (.85, 1.7, 3.4) that Beranek estimated would just permit reliable conversation out of doors (understanding of 95% or more of the key words in a group of sentences), a situation corresponding to correctly hearing approximately 75% of a list of isolated phonetically-balanced words. Thus, Table 6-1 indicates that speech when spoken at a normal level can only just be heard at a distance of 3 feet when the noise has an SIL (.85, 1.7, 3.4) of 55 dB. As voice level is judged to go from "normal" to "raised", "very loud", and (sustained) "shouting", respectively, Beranek postulates a four-fold increase in vocal output for each step, or a 6-dB increase in acoustic output. If the voice rises 6 dB for each step, then, as a first approximation, the noise can also increase by the same amount without changing the intelligibility of the speech. Therefore, at 3 feet a "raised" voice can be heard through a 61-dB-SIL (.85, 1.7, 3.4) noise, a "very loud" voice is intelligible in 67 dB SIL, and a "shout" will be understood in 73 dB SIL.

The values for other distances in this table are merely expressions of the well-known inverse square law, which is that the sound intensity will drop by a factor of 4 (i.e., the level will drop 6 dB) if one doubles the distance from the source in the free fields (outdoors). If the listener is 6 feet from the talker, therefore, the speech level at his ears will have dropped to 6 dB less than what it was at 3 feet, hence the noise that will permit normal conversation will also be 6 dB lower, or 55-6-49 dB SIL (.85, 1.4, 3.4). A chart can, therefore, be constructed showing the relations of Table 6-1 in graphic form. Further, since it is simpler, for general purposes, to use dBA instead of SIL, a conversion from SIL to dBA is made for the purpose of this graph (Table 6-1).

Although the difference between the SIL and dBA values of any two noises will ordinarily not be the same, since this difference will depend on the exact spectrum of each, attempts have been made to determine an average conversion number for a more or less vaguely-defined "average" noise. Klumpp and Webster¹¹, for example, showed in their sample of 16 shipboard noises that SIL (0.5, 1, 2) values averaged about 10 dB lower than corresponding A-weighted sound levels and about 17 dB lower than C-weighted sound levels. Similarly, Kryter⁵ selected seven different common spectra from the research literature and found that for these noises dBA minus SIL was about 9 dB, dBC minus SIL was 13 dB. For the present purposes, then, it can be assumed that for not-unusual noises, the A-weighted sound levels that will permit conversation can be derived by simply adding 10 dB to the values of Table 6-1, and that the overall (C-weighted) levels will be an additional 5 dB higher, or 15 dB above SIL (.85, 1.7, 3.4) values.

The dashed lines in Figure 6-1 show these converted values. The ordinate is the A-weighted sound level of the noise at the listener's ears. The abscissa is the distance between talker and listener in feet, plotted in a logarithmic fashion. The four dashed lines indicate the highest noise level that will permit near-100 percent understanding of sentences spoken with the effort indicated on each curve, in the outdoor environment. Thus, in a 70 dBA noise, a normal voice can be heard

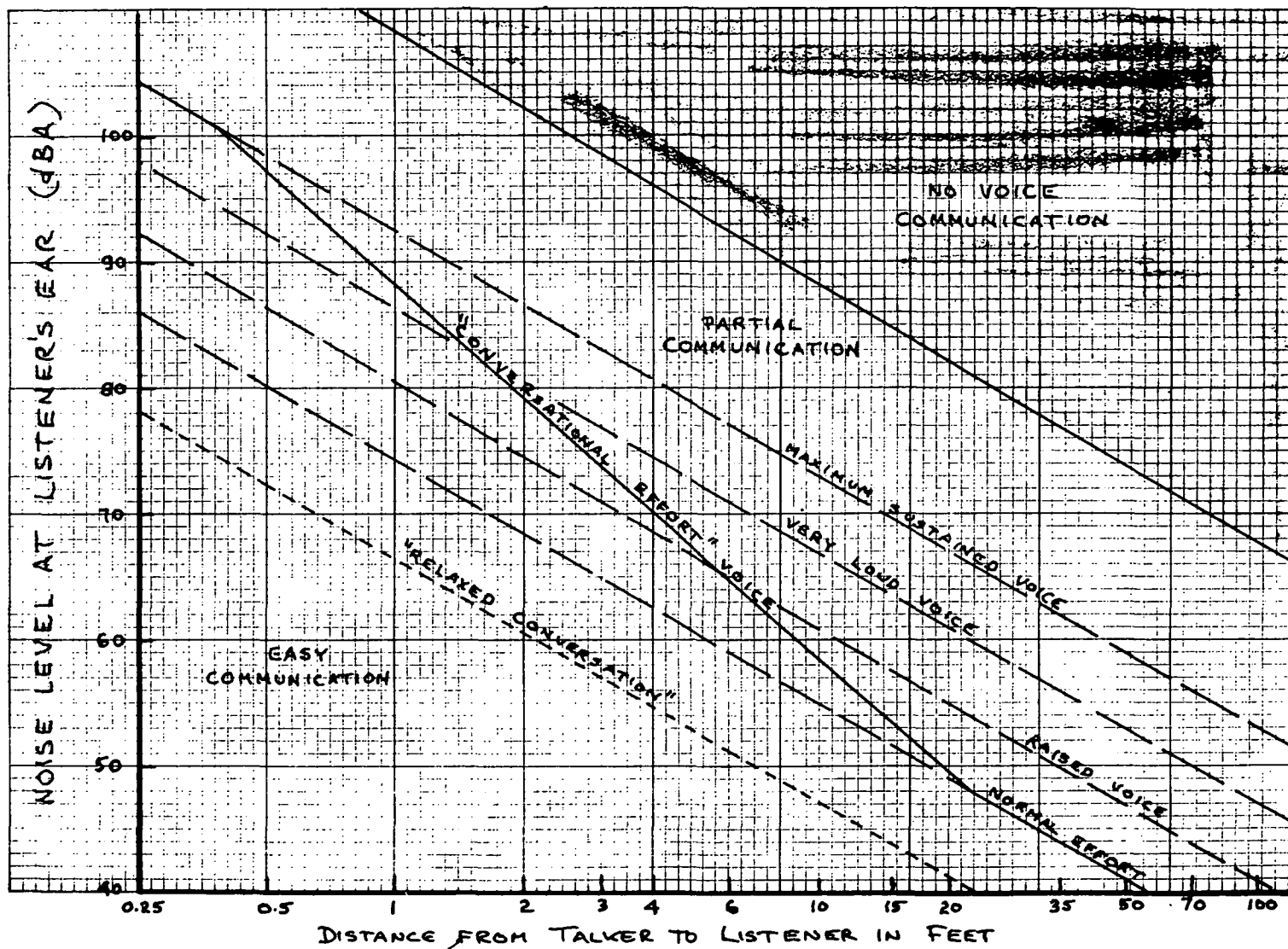


Figure 6-1. Distance at which ordinary speech can be understood as a function of A-weighted sound levels of masking noise in the outdoor environment.

TABLE 6-1

SPEECH INTERFERENCE LEVELS (SIL(.85., 1.7, 3.4))
 FOR OUTDOORS ENVIRONMENTS THAT PERMIT
 BARELY RELIABLE CONVERSATION, OR THE
 CORRECT HEARING OF APPROXIMATELY 75%
 OF PHONETICALLY-BALANCED WORD LISTS, AT
 VARIOUS DISTANCES AND VOICE LEVELS. FROM BERANEK.¹⁰

Distance between talker and listener (ft)	Normal	Voice Level Raised Very Loud SIL (in decibels)		Shouting
0.5	71	77	83	89
1	65	71	77	83
2	59	65	71	77
3	55	61	67	73
4	53	59	65	71
5	51	57	63	69
6	49	55	61	67
12	43	49	55	61

at a distance of only 2 feet, a raised voice at 3.5 feet, a very loud voice at 7 feet, and a shout at 15 feet. The curve farthest to the left indicates that in 70-dBA outdoor noise, speech at the level that is generated when people are engaged in "relaxed conversation" in quiet would be completely understandable only at a distance of 9 inches or so. Beyond 15 feet, progressively more and more of even shouted speech is masked, so that at 70 feet (i.e., at the boundary of the blackened area on the right), a shout may serve to attract a listener's attention, but will convey little other information. Hence, beyond this point, no voice communication is possible unless of course, the speech is amplified by one means or another (cupping the hands, using a megaphone, or employing electronic amplification).

Reception of Indoors Speech

The dashed curves of Figure 6-1 predict fairly accurately how noise will affect the perception of speech in the outdoor environment (field free). However, the criterion of distance between the talker and the listener is not valid to assess the intrusion of the outdoor noise levels on the reception of speech indoors because of the reverberant build up of sound by reflections from the walls of the room. Over the years, various studies have been concerned with specifications of values which could be utilized in the design of rooms. An example of such data are presented in Table 6-2.

The data available in the pertinent literature suggests that, for most instances, a reasonable value for the design of rooms where oral communication is important is somewhere in the range between 40-45 dBA. It is found that a steady state noise level that does not exceed this value will assure a 100 percent sentence intelligibility.¹²

FACTORS IN THE DEGREE OF SPEECH INTERFERENCE

Characteristics of People (Speech, Age, and Hearing)

The contours on Figure 6-1 represent conditions for young adults, speaking the same dialect, when they are in a diffuse noise field. The location of these contours will shift in accordance with many variables. Lower noise levels would be required if the talker has imprecise speech (poor articulation) or if the talker and the listener speak different dialects. Children have less precise speech than do adults¹³, and their relative lack of knowledge of language often makes them less able to "hear" speech when some of the cues in the speech stream are lost. Thus, adequate speech communication with children requires lower noise levels than are required for adults. One's ability to understand partially-masked or distorted speech seems to begin to deteriorate at about age 30 and declines steadily thereafter¹⁴. Generally, the older the listener, the lower the background must be for nearly normal communication. Finally, it is well known that persons with hearing losses require more favorable speech-to-noise ratios than do those

TABLE 6-2

DESIGN OBJECTIVES FOR INDOOR A-WEIGHTED SOUND LEVELS IN
ROOMS WITH VARIOUS USES, AS RECOMMENDED BY AN
ACOUSTICAL ENGINEERING FIRM ON THE BASIS OF
EXPERIENCE WITH ACCEPTABILITY LIMITS EXHIBITED
BY THE USERS OF THE ROOMS. FROM BERANEK ET AL.¹⁶

Type or use of space	Approximate A-weighted sound level (dBA)
Concert halls, opera houses, recital halls	21 to 30
Large auditoriums, large drama theaters, churches (for excellent listening conditions)	Not above 30
Broadcast, television and recording studios	Not above 34
Small auditoriums, small theaters, small churches, music rehearsal rooms, large meeting and conference rooms (for good listening)	Not above 42
Bedrooms, sleeping quarters, hospitals, residences, apartments, hotels, motels (for sleeping, resting, relaxing)	34 to 47
Private or semiprivate offices, small conference rooms, class- rooms, libraries, etc. (for good listening conditions)	38 to 47
Living rooms and similar spaces in dwellings (for conversing or listening to radio and television)	38 to 47
Large offices, reception areas, retail shops and stores, cafe- terias, restaurants, etc. (moderately good listening)	42 to 52
Lobbies, laboratory work spaces, drafting and engineering rooms, general secretarial areas (for fair listening conditions)	47 to 56
Light maintenance shops, office and computer equipment rooms, kitchens, laundries (moderately fair listening conditions)	52 to 61
Shops, garages, power-plant control rooms, etc. (for just- acceptable speech and telephone communication)	56 to 66

with normal hearing. However, little quantitative data exists to indicate how much the curves of Figure 6-1 should be shifted for specific values of the factors above.

Situational Factors

Of course, adequate communication in higher noise levels than those indicated on Figure 6-1 can occur if the possible messages are known to be restricted. Thus, at ball games, it is possible to discriminate the umpire's "ball" and "strike" (assuming that he actually says these words) at much greater distances and in more intense levels of noise than indicated on the chart. This factor accounts for the success of communication in many industrial situations with high levels of noise. Failure may occur, however, when an important but *unpredictable* message must be communicated. For example, firemen in a high-level noise may have little difficulty with standard communications about the use of familiar equipment, but they may encounter grave difficulty communicating about unexpected events that occur at the scene of the fire.

The opportunity to lipread or use facial or body gestures in support of hearing will improve the success of communication in background noise. Almost everyone has some small amount of lipreading skill that they often use without awareness of its contribution to intelligibility.

Spatial variables also may facilitate or impede speech communication in noise. If the source of noise is clearly localized in a position different from that of the talker, speech communication may be possible under noise conditions less favorable than those indicated on Figure 6-1. On the other hand, noise interferes with speech communication more when either is reverberant (involves echoes).

Noise Characteristics

Finally, it must be remembered that the exact characteristics of the noise are also important for predicting speech communication. While the A-weighted noise level is an adequate measure of many noises, some situations and noises demand a more complicated analysis. This is particularly true of noises that consist almost exclusively of either low frequencies or high frequencies—e.g., the rumble of ships' engines or the hiss of compressed air. A chart similar to Figure 6-1, but with an additional correction based on the difference between the C- and A-weighted levels of the noise, has been developed by Bostsford.¹⁵ However, in case of a very unusual noise, it is probably better to calculate the AI if a relatively accurate prediction of speech intelligibility is necessary. A discussion of the use of the various methods of measuring noise to predict speech interference can be found elsewhere.⁵

Figure 6-1 applies only to reasonably steady noises. Intermittent noises and impulses will, of course, mask certain signals only while they are present, and noises fluctuating in level will

provide variable degrees of masking. Again, speech is redundant enough so that an isolated 1-second burst of noise is unlikely to produce much disruption of the communication process; however, this probability grows with both the frequency and the duration of the noise-bursts. If a noise criterion such as "X percent perception of sentences" is adopted, therefore, it will be necessary to specify those patterns of noise that produce this particular degree of intelligibility loss. For example, any noise above 70 dBA in level will interfere with conversation, even with a raised voice. Hence, if a criterion were 90 percent sentence intelligibility, then an 85-dBA noise would meet the criterion, provided it were on only 10 percent of the time.

Acoustic Privacy

It should be pointed out that not all masking is an unmitigated evil. A noise that can be ignored may be able to blot out an annoying one. Indeed, offices can be made too quiet, so that everyone can hear the speech and other sounds produced by everyone else—in which case the speech in question becomes "noise." In a study of workers in noisy workshops, Matsui and Sakamoto¹⁷ found that just as many persons admitted feelings of irritation about noise in the 50-dB environment that served as a control situation ("desk work") as those in a 100-dB environment; in the control case, the irritation was attributed to the rustling of paper.

For "acoustic privacy," therefore, a moderate amount of background noise may be desirable. If an office area has been made too quiet, a low level of noise (recorded sounds of surf or a waterfall would serve as well as the intentionally uninteresting music that is widely employed in this country) may have to be reintroduced so that its level permits ordinary conversation at 10 feet or less but requires raising the voice in order to be heard at greater distances. The "optimum" noise level is seldom if ever complete silence.

SUMMARY—MASKING AND SPEECH INTERFERENCE

Speech interference is one aspect of "masking" — an interaction of two acoustic stimuli whereby one of them changes the perceived quality of the other, shifts its apparent location or loudness, or makes it completely inaudible. Much information is available concerning the masking of fairly simple signals such as pure tones, noise bands and nonsense syllables by noises of various spectra; and general laws have been developed that will allow rather accurate prediction of whether or not a speech sound will be masked by a particular noise.

In describing speech interference, the noise concerned can be defined either in terms of its specific spectrum and level or in terms of any number of summarizing schemes. In addition to the average A-weighted sound level, the two most generally-used alternative methods of characterizing noises in respect to their speech-masking abilities are the articulation index (AI) and the speech interference level (SIL). The AI takes into account the fact that certain frequencies

in the masking noise are more effective in masking than other frequencies. The SIL is more simplified, indicating only the average general masking capability of the noise. Since much speech is spoken at a reasonably constant level, it is possible to express many of the empirical facts about average speech communication in a single graph showing noise level, vocal effort, and distance.

Various factors enter into the degree of speech interference. Speech, age, and hearing of individuals affect communications. Children have less precise speech than adults do. Older listeners are more susceptible to interference from background noise.

Situational factors influence the degree of speech interference. In some contexts, the predictability of the message will decrease speech interference. Nonverbal communication and lipreading have the same effect. Spatial variables may facilitate or impede speech communication in noise. The exact characteristics of noise are important in predicting speech communication.

REFERENCES

1. L. A. Jeffress, "Masking," in "Foundations of Modern Auditory Theory, Vol. 1," J. V. Tobias, Ed., Academic Press, New York (1970).
2. B. Scharf, "Critical Bands," in "Foundations of Modern Auditory Theory, Vol. 1," J. V. Tobias, Ed., Academic Press, New York (1970).
3. J. C. Webster, "Effects of Noise on Speech Intelligibility," in "Noise as a Public Health Hazard," ASHA Reports No. 4, Am. Speech and Hearing Assoc., Washington (1969).
4. J. C. Webster, "The Effects of Noise on Hearing Speech," Proc. Internat. Congress on Noise as a Public Health Problem, Govt. Printing Office (1973).
5. K. D. Kryter, "The Effects of Noise on Man," Academic Press, New York (1970).
6. N. R. French and J. C. Steinberg, "Factors governing the intelligibility of speech," J. Acoust. Soc. Am. 19, 90-119 (1947).
7. K. D. Kryter, "Methods for the calculation and use of the articulation index," J. Acoust. Soc. Am. 34, 1689-1697 (1962).
8. L. L. Beranek, "The design of speech communication systems," Proc. Inst. Radio Engrs. 35, 880-890 (1947).
9. R. G. Klumpp and J. C. Webster, "Physical measurements of equally speech-interfering Navy noises," J. Acoust. Soc. Am. 35, 1328-1338 (1963).
10. L. L. Beranek, "Noise control in office and factory spaces," 15th Annual Mtg. Chem. Eng. Conf., Trans. Bull. 18, 26-33 (1950).
11. T. S. Korn, "Effect of psychological feedback on conversational noise reduction in rooms," J. Acoust. Soc. Am. 26, 793-794 (1954).
12. Kryter, K. D., Specifying Tolerable Limits of Exposure to Noise, Draft Document for World Health Organization, Europe, 1973.
13. S. Eguchi and I. J. Hirsh, "Development of speech sounds in children," Acta Oto-Laryngol. Suppl. 257 (1969).
14. A. Palva and K. Jokinen, "Presbycusis: V. Filtered Speech Test," Acta Oto-Laryngol. 70, 232-241 (1970).
15. J. H. Botsford, "Predicting speech interference and annoyance from A-weighted sound levels," J. Acoust. Soc. Am. 42, 1151 (1967).
16. L. L. Beranek, W. E. Blazier and J. J. Figwer, "Preferred Noise Criterion (PNC) Curves and their Application to Rooms," J. Acoust. Soc. Am. 50, 1223-1228 (1971).
17. Matsui, K. and Sakawato, H., The Understanding of Complaints in Noisy Workshop; *Ergonomics*, 14, 95-102, 1971.

Section 7

ADDITIONAL PHYSIOLOGICAL AND PSYCHOLOGICAL CRITERIA

Acoustic stimulation of the ear can affect many parts of the body and nervous system in addition to the auditory system. These “non-auditory” or “extra-auditory” effects are mediated through at least three neural systems which are not considered to be an integral part of the auditory mechanism:¹

1. The autonomic nervous system controlling general somatic responses and the state of arousal of the body—the glands, viscera, heart, blood vessels, etc.
2. The reticular nervous system which appears to be involved in the state of arousal of the higher brain centers of the central nervous system and with sensory inputs related to pain and pleasure.
3. The cortical and subcortical brain centers concerned with cognition, consciousness, task performance, “thinking,” etc.

It is important, therefore, to consider not only the more overt effects of noise, such as hearing loss and the masking of speech, but the more subtle effects which noise can produce. These non-auditory effects can be merely transitory or, in some cases, long-lasting. They usually take place without conscious knowledge of their occurrence.

PAIN

Tympanic Membrane

There are two general types of aural pain or discomfort. The first type is caused by the stretching of the tympanic membrane tissues in response to large amplitude sound waves. Although there is a fairly wide range of individual variability, especially for high-frequency stimuli,¹ the threshold of pain for normal ears is approximately 135-140 dB SPL. This threshold is essentially independent of frequency,² and it will occur in totally deaf as well as normally hearing people since it is not a function of the ear's sensorineural system. A good indication that this reaction is a function of the tympanic membrane was demonstrated by Ades *et al*,³ who found that people without eardrums report no sensations of pain to sound levels up to 170 dB SPL. At somewhat lower sound pressure levels (120 to 130 dB), one may experience some discomfort or a tickling sensation in the ear canal. Since these levels are considerably above the level of hearing damage risk, aural pain

should not be considered an early warning signal of excessive noise exposure. However, if aural pain should occur in an otherwise normal ear, it should be a clear sign that hazardous noise levels are being experienced.

In special cases, aural pain originating in the eardrum or middle ear may occur in response to sound levels considerably lower than 130 dB SPL. Davis and Silverman⁴ point out that sounds of moderate intensity can produce pain when middle ear tissues are tender from inflammation and the eardrum may be tense with pus. Similarly, contraction of the middle ear muscles (elicited at about 80 to 90 dB SPL) can be painful if these muscles are inflamed.

Inner Ear

A second type of aural discomfort occurs as a result of abnormal function in the cochlea or inner ear. Certain sensorineural disorders, and most frequently noise-induced hearing losses, are accompanied by a condition called auditory recruitment, a term attributed to Fowler.⁵ Recruitment is defined as an abnormal increase in loudness perception, a condition seen in pathological ears. In some cases of sensorineural hearing disorders, the condition is more severe, and it can lead to considerably lower thresholds of aural discomfort or pain. Thus, sound levels of only moderate intensity can occasionally be quite uncomfortable to individuals experiencing auditory recruitment. Davis and Silverman⁴ mention that in special cases of sensorineural hearing disorders with symptoms of diplacusis (a condition in which a tone is perceived as having a different pitch in the two ears) and severe tinnitus, subjects can be unusually vulnerable to noise-induced hearing loss. These cases often display lower thresholds of aural pain that may serve a useful warning function.

Hearing Aids

Another important consideration in the area of aural pain is the effect of noise on hearing aid users. Discomfort associated with exposures to traffic noise, loud music, and even raised voice levels is a common complaint among hearing-impaired people who wear hearing aids. Although many hearing aids have devices which automatically limit output intensity to 120 or 130 dB SPL, the protection offered may not be sufficient for some recruiting ears. In some cases, in order for speech to be loud enough to be intelligible, it borders on (or even exceeds) the listener's threshold of discomfort. Hearing aid users comprise approximately 1 percent of the American population,⁶ and about 50 percent of these are over age 65⁷ and tend to suffer more discomfort from loud sounds than their younger counterparts. Thus, a passing subway train at 95 dBA or a jet flyover at 105 dBA, which might be momentarily annoying to a normal listener, could be excruciating when amplified for a hearing-impaired individual with recruitment.

EFFECTS OF NOISE ON EQUILIBRIUM

Many years ago in Italy, Tullio⁸ demonstrated that pigeons could be made to veer off course by presenting an intense low frequency stimulus during flight. From this it has been concluded that a direct relationship could be found between acoustic stimulation and vestibular (balance sense) effects.⁹

Complaints of nystagmus (rapid involuntary side-to-side eye movements), vertigo (dizziness), and balance problems have been reported under noise conditions in the laboratory as well as in field situations. The levels needed to cause such effects are quite high, typically 130 dB SPL or more.¹⁰ Less intense noise conditions in the range of 120 dB SPL, however, can disturb one's sense of balance, particularly if the noise stimulation is unequal at the two ears. This was demonstrated in a laboratory study in which subjects were required to balance themselves on rails of different widths.¹¹ McCabe and Lawrence¹² offered the suggestion that these effects are due to noise directly stimulating the vestibular sense organs whose receptors are part of the inner ear structure. Recently, Lipscomb and Roettger¹³ observed a high degree of swelling of capillary walls in the region of the vestibular organs of rats exposed to 110 dBA noise for 48 hours. Those effects have been attributed to reduced blood flow to the sensory regions following substantial noise exposures.¹³

Dieroff and Scholtz¹⁴ attempted to test whether or not there exists a significant correlation between hearing loss due to steady industrial noise and vestibular function. They conducted various vestibular tests on 293 men and 51 women with various degrees of noise-induced hearing loss. No significant correlations were found, indicating that habitual exposure to continuous high-intensity noise is dangerous only to the auditory system and not to the vestibular system. These findings were obtained by using vestibular tests when the subjects were no longer in the noise. It would be important also to assess whether continuous stimulation by moderate levels of noise will create measurable vestibular conditions while the subject is still in the noisy environment.

Due to the scarcity of available data in the pertinent literature, many questions regarding the effect of noise exposure on equilibrium remain unanswered.

ORIENTING AND STARTLE REFLEXES (ACOUSTIC)

Man is equipped with an elaborate set of auditory-muscular reflexing capabilities. The orienting portion of these reflexes serves to turn the head and eyes toward a sharply occurring sound source in order to locate its origin. The startle reaction (recorded by Molinie),¹⁵ occurs primarily in order to prepare for action appropriate to a possible dangerous situation signalled by the sound. According to Davis¹⁶ and Galambos, *et al.*,¹⁷ the reflex activity begins to operate even at low levels of sound energy. The presence of these extrinsic acoustic reflexes is detected either by noting behavioral clues or by electrophysiological study of muscle tension and activity. With the advent

of low level sound stimulation that is sufficient in abruptness and information to occasion a startle reflex, there is often little or no noticeable evidence that a person has experienced some degree of startle except with the use of electrical measures.

Response In Children

Human response to sound develops at very early childhood. Youngsters in the first two months of life tend to give an all-or-none response to sound stimuli. At this period, a child will signal having heard the sound by a startle reflex, a gigantic seizure-type of reaction, or by a number of other lesser responses such as the eye-blink, crying, diminution of activity or sudden assumption of a listening attitude. In general, neonates demand a considerable amount of sound prior to giving any of the above-named responses.¹⁸ Some children, later found to have normal hearing sensitivity, do not respond well or consistently to sound stimulation during this period. Most small babies, however, do give some degree of response to auditory stimulation if the sound is raised to between 80 and 100 dB Hearing Level (H_L).

With maturity, human response to sound becomes modified and diversified so that a considerable number of additional behavioral observations can be made.¹⁹ After the first two months, small children begin to respond to sound consistently. The sharp startle reaction is reduced, being reserved only for those times that sound has a disturbing quality.

Adult Response

Landis and Hunt²⁰ have given numerous details regarding the behavioral concomitants of the startle response in mature humans. These manifestations include:

- The eyeblink (if the eyes are open).
- Firm closure of the eyes (if the eyelids are loosely closed).
- Facial grimaces of a characteristic nature.
- Bending of the knees.
- A general inward flexion of the body.

These events occur in something less than 0.5 sec. Other observers have cited:

- Increased neck and shoulder muscle tension tending to draw the head downward.
- Random foot movement.
- An elevation of the arms bringing the hand toward the face with an inward rolling of the forearms.²¹

These sudden body movements are accompanied by a set of physiological reactions:

- Alteration in cardiovascular function.
- Increased endocrine activity.
- Alteration of respiration rate and cessation of gastro-intestinal activity.

Fortunately, these physiological effects are of short duration and the body returns rapidly to its previous state within a few seconds (or minutes) after the onset of the startling stimulation.

According to Landis and Hunt,²⁰ the startle response to sound, such as a nearby gunshot, may undergo various degrees of diminution with repetition of the sound. This lessening of response depends upon several factors, including:

- The responsiveness of the individual.
- Repetition rate of the sound.
- The predictability of occurrence of the sound.

In some persons, there is little decrease in reaction from one impulse to the next. With others, there is a marked reduction in reaction as repetitions occur. The eyeblink and head movement aspects of the startle response never habituate completely. Even experienced marksmen exhibit these responses each time they fire a gun. This assertion was confirmed by Davis and Van Liere²² when they measured electrical indicators of muscle activity. An early response with a latency of about 0.1 second showed little reduction with repetition of the sound. A later measured element in the muscle reaction to sound stimulation which had a 0.8 second latency did diminish significantly with repetition of stimulation.

Variation In Muscular Response

A series of experiments by R. C. Davis and his colleagues²³⁻²⁶ demonstrated that the particular muscular responses to sound and the way in which these responses will influence the performance of a motor task depend in detail on:

1. Pattern of muscular tension or posture, prior to the sound.
2. Movements required by the task.
3. Auditory-muscular reflexes.²⁷

From the standpoint of the interfering characteristics of sudden noises, one of the more important findings was that the magnitude of the muscle-tension reflex in response to sound increases with a rise in resting tension in the muscle itself. (This generalization, of course, would not hold as a muscle approaches its maximum level of tension). Thus, if a person is required to make a movement requiring flexion and if his posture heightened tension in the appropriate flexor muscle, a burst of sound, which ordinarily produces the reflex action of flexion, would speed the performance of the movement. The result of this effect is obvious when one considers that the hand might have been holding a fluid-filled container. Under other conditions, however, the burst of sound could greatly interfere with the required movement. As an example, consider that, as before, the required movement was that of flexion but that the person's posture heightened the resting tension in the opposing extensor. In this case, a burst of sound would result in a greater response in the extensor (because of the higher resting tension) than in the flexor. The consequence would be that the required

and desired flexion activity would be interfered with and delayed. In delicate operations in assembly plants, etc., these effects could greatly affect quality control and workmanship.

The ebb and flow of muscular activity is closely linked to and influenced by the rise and fall of sound in an immensely complex manner. Gross body orientation toward an unexpected source of sound will diminish as the sound becomes familiar and predictable. While some components of the startle response to sharp sounds will diminish with repetition of the stimulus, the exact amount of this reduction depends upon a number of variables. Subtle changes in the musculature in response to sustained sound may persist as long as the sound is present, and the effects will depend in a complicated way on posture, activity, and the characteristics of the sound.

Because of the brief durations involved, there is no concrete evidence that startle and orienting reflexes have a direct bearing upon the general health of humans. Secondly, however, being startled might produce an untoward and uncontrollable muscular reaction which can cause injury in the event an arm is caused to extend into rapidly moving machinery, if a person is involved in precarious work, or if sharp items or volatile liquids are being handled.

INTERNAL MECHANISMS--VEGETATIVE AND STRESS REACTIONS

The degree to which a stimulus, such as noise, poses a threat to health and well-being of an individual depends upon the exposure characteristics involved. If the experience is of very brief duration, as was the case with the previously mentioned reactions in this section, the transient nature of the exposure allows the system to return to a normal or preexposure state. If noise stimulation is sustained or consistently repeated, however, it has been observed that specific changes occur in neurosensory, circulatory, endocrine, sensory and digestive systems. These modifications of a body function may tend to be less transitory.

Noise and The Nature of Stress

As an adjunct to continuous exposure to noise, the keen balances maintained in body physiology can become disrupted.²⁸ This disturbance may be made known at the conscious level as the feeling of annoyance, irritation and fatigue which will be discussed later in this section. It generally holds that the disturbing or stressful characteristics of a sound increase with the loudness level of the sound. There is also a frequency-dependent aspect. Those sounds whose energy is in the frequencies at or above 2000 Hz are usually more distressing than sounds whose spectrum contains mostly low-frequency energy. Because of a great range in human variability with respect to the reaction to sound stimulation, these responses are highly unpredictable. There is an element of wide variation in the same individual from day to day or from moment to moment as well as variability between individuals.

Numerous studies have been undertaken to observe the internal reaction experimental animals undergo when they have been exposed to intense sound for long durations. Some of these studies are cited and discussed in detail in the text by Welch and Welch.²⁹ Some of these results await verification by further research efforts. Some of the data has not been supported by subsequent studies. The trend in the literature appears, however, to indicate that there is a potential for some alteration of body function during and, sometimes, immediately after noise exposure.

It appears that some aspects of noise exposure (noise bursts, startling sounds, etc.) result in a form of automatic response in that one's attitude about the exposure conditions tends to have little or no effect upon the internal, bodily reactivity to the noise stimulation. There is, however, a "stress" component which is related to the degree to which the noise stimulation is aversive.³⁰

One further consideration deserves mention here. There is seldom an instance where a single stressing condition exists. Often, a combination of stressors occur, of which noise may be only one. In many situations, the stressor may give rise to fear or anger responses yielding an entirely different combination of body responses. In that case, the stressor itself may be negligible in its effect, while the reaction to the stressor may be the major stressing agent.

Stress, according to Selye,³⁰ is largely non-specific. That is, there is not a set of specific reaction characteristics in the body for each stressing agent. Rather, Selye and his staff in hundreds of experiments have observed that most stressing agents cause an alarm reaction which consists of three manifestations:

1. Thymico-lymphatic involution (shrinking of the thymus gland which is located immediately over the heart).
2. Gastric ulcers, usually located on the duodenum.
3. Adrenal hypertrophy (swelling of the adrenal glands).

It has been shown at 48 hours exposure to 110 dBA broadband noise stimulation evokes these reactions in experimental animals.³¹ It was concluded from that experiment that intense noise, in the sense of Selye's definition, can be classified as a physiologic stressor.

Short and infrequent periods of stress are usually innocuous by virtue of there being an opportunity for the relevant opposing forces of the body to regain their balance within a brief period after exposure. Long-term stress is regarded as posing a potential danger to the health of an individual, this attitude being largely developed from extensive work on experimental animals. A major question that does not appear to have been resolved is with regard to the point at which a noise becomes a stressing agent in man, and what amount of exposure is necessary to cause long-lasting or permanent physiological changes.

Stress and the Metabolic System

There is little definitive data on the degree to which the preceding observations relate to stress in humans, since much of the experimental work, necessarily, has been conducted using animals. Using the umbrella of “stress theory”, however, a number of observations can be made.

Selye³⁰ has described what he calls the General Adaptation Syndrom (GAS) which occurs in three steps after the onset of the action of a stressing agent:

1. The alarm stage was described earlier as effecting thymus, duodenal and adrenal condition. This stage is one where there is considerable activity in the body’s defensive mechanisms as the system begins to muster its defenses against the stressor.
2. The stage of resistance is that period where the body combats the influence of the stressor. If the stressing agent is relatively weak, it will be overcome during this stage.
3. The stage of exhaustion occurs if the stressor is one of sufficient strength or if the stress takes place over a long enough time to wear out the defenses of the body. In the event the stressor is a severe one, the end result of the exhaustion stage would be a breakdown in body function which could end in death.

Selye points out that during the stage of resistance there occurs a decreased resistance to infection, also perhaps to specific diseases he has called the diseases of adaptation. Among such diseases are some types of gastro-intestinal ulcers, different varieties of blood pressure elevation, and possible forms of arthritis.

It should be observed that there is not unanimous agreement among medical authorities relative to the existence of these diseases of adaptation as defined by Selye. There are those who maintain that each disease has its own specific cause or set of causes.

A wide variety of stressful stimuli activates the pituitary-adrenal system with increased secretion of ACTH (adrenocorticotrophic hormone) and a consequent increase in adreno-cortical activity.³² This includes:

- Trauma.
- Surgery.
- Infection.
- Cold or heat exposure.
- Forced exercise.
- Hemorrhage hypoxia.
- Burns.
- Hypoglycemia (low blood sugar).
- Pain.
- Immobilization.
- Severe psychological trauma.
- Anticipation of physical injury.

The ACTH secretion is accomplished by a neurohumoral (chemically mediated) mechanism³³ between hypothalamic nuclei and the adenohypophysis³⁴ (anterior portion of the pituitary gland). Noise can be considered one of the nonspecific stressors which cause the release of ACTH from the pituitary.

Like other stressful stimuli,³⁵ noise causes a biphasic pattern of ACTH release.³⁶ In the rat, the corticosterone secretion rate doubles after 30 minutes and triples after one hour of exposure to a 130 dB tone of 220 Hz. The high rate occurring after a one hour exposure is maintained over 8 hours, but after 12 hours the secretion rate decreases to values at or below control levels only to rise again to the maximal rate after 24 and 48 hours of repeated exposure.

In the rat, noise exposure of 80 dB (SPL) for 18 days alters adrenal function with a decrease in ascorbic acid content³⁷ in the adrenal, a reflection of ACTH stimulation. A level as low as 68 dB (SPL) for only 30 minutes releases ACTH as measured by a decrease in adrenal ascorbic acid content and by eosinopenia (low numbers of one type of white blood cells), a peripheral glucocorticoid effect.³⁸ Dilation of the capillary bed of zona reticularis (one of the layers of the adrenal gland) and medullar sinusoids (terminal blood channel in the adrenal gland) occurs after 80 dB (SPL). With higher exposures to 102 dB for 4 hours per day for 11 days, these vascular changes worsen and karyopyknosis (shrinking of a cell nucleus) occurs in the cells of the zona fasciculata³⁷ (another layer in the adrenal gland). Other pathological changes include an increase in adrenal weight which can be demonstrated in mice after only 15 minutes daily exposure for 4 weeks to 110 dB sound ranging between 10-20 kHz. Studies in humans are few but a 65 dB sound of 10 kHz has been found to cause a 53 percent increase in plasma 17-hydroxycorticosteroids.⁴⁰

There is indirect evidence that noise-induced adrenal changes are transient, disappearing with cessation of the noise. Eosinopenia, a peripheral glucocorticoid effect, occurs only temporarily after noise,³⁹ and the pathological changes in the adrenal cannot be demonstrated one month after exposure.³⁷ As noted, the general adaptation syndrome of Selye⁴¹ to chronically maintained stress consists of three stages of response. However, adaptation to stress is not a constant finding. Plasma corticosterone levels in rats are persistently elevated during the chronic application of multiple stresses (sound, flashing lights, and cage oscillation).⁴² Likewise, there is no evidence that the hypothalamo-hypophyseal-adrenal axis, (interaction between the hypothalamus, pituitary gland and adrenal glands) adapts to the stress of chronically maintained noise.

Noise also affects the adrenal medulla (the inner portion of the adrenal gland). An increased urinary excretion of epinephrine (a product of the adrenal gland) occurs in the rat in response to high frequency sound (20 kHz) at 100 dB.⁴³ Increased urinary excretion of epinephrine and norepinephrine after exposure to 90 dB (2000 Hz) for 30 minutes is a constant finding in normal humans and in patients with essential hypertension (high blood pressure without known cause) and in those recovered from myocardial infarction⁴⁴ (heart attack).

Variables In Stress Effects

Stress theory, even as presented by its strongest advocates, is admittedly complicated. There are complex interactions between conditioning factors that lead to disease, non-specific reactions to stressing agents and general behavioral concomitants which create an immensely complex pattern. In view of this, the predictability of body response to any given stressor, including noise, is impossible.

Whether there is any adaptation or accommodation to an ongoing stressful condition caused by sound is not well established. Several persons have questioned the ability of the body to adapt to the stressing effect of an on-going stimulus. It can be reasoned that if adaptation were to occur, however, each new presentation of a noise stimulus would reestablish the stressing condition. Therefore, it does not seem likely that the highly variable noise stimulation most people receive can be easily or effectively accommodated.

It is certain that intense sound can serve as a stressor and, at least for some of the more popular experimental animals, can lead to some physiological changes. Additionally, it is plausible that some of the more intense sounds in the environment will act as stressors for people. The conditions under which this might occur are yet unknown. Factors important to consider are:

- The intensity level of the sound stimulus.
- Its characteristics (sudden vs. gradual rising, etc.).
- The amount of fear or misfeasance engendered by the sound.
- The susceptibility of the individual to emotional and physiological reaction.

The concept that stress is universally bad and unhealthy is misleading. At certain periods in life, some stressing agents and stressful situations might be construed as necessary (alerting, orienting, motivating). Thus, although it is plausible that noise can be detrimental as a stressing agent, there is insufficient data to indicate unequivocally that noise as a stressor is sufficiently severe to cause seriously untoward reactions. Most studies of noise-induced stress upon internal body functions have utilized quite high sound intensities. There is however, some evidence that low level noises create internal physiological changes.³⁸

In Czechoslovakia, a study by Kirkova and Kromorova⁴⁵ implied that stress reactions may well become important at high levels. Medical records of 969 workers in 85-to-115-dB areas were compared with those of 689 workers in 70-dB working environments or less. In addition to a higher incidence of hearing loss, the noise exposed group were found to have a higher prevalence of peptic ulcers.⁴⁵

Circulatory System and Vasoconstriction

The effects of noise in the laboratory on gross parameters of the circulatory system—blood pressure, pulse rate, EKG—are apparently negligible, at least at intensities up to 100 dB (SPL).⁴⁶⁻⁴⁸ Although there are reports that a higher incidence of circulatory problems exists in noise-exposed steel workers⁴⁹ and machine shop operators,⁵⁰ it cannot be said with confidence that noise alone caused the circulatory problems in these populations. It has been observed that the slight differences in the men exposed to high levels of sound relative to those less exposed in these European studies could be due to equally small differences in other working conditions such as poor ventilation, heat or light, stress from other sources such as anxiety over job security, and especially personnel selection.⁵¹ This critique is supported by data advanced by Satalov *et al.*⁵² in which men with the greatest hearing loss (and who therefore presumably suffered a greater average noise exposure) had blood pressure figures no higher than those with the most normal hearing, when age was controlled. To settle this controversy, a well-controlled study for long periods of time is needed to observe heart problems in American industrial workers who have been exposed to noise.

Associated with ongoing noise exposure, some have found evidence of constriction of blood vessels which is primarily manifest in the peripheral regions of the body such as fingers, toes, and earlobes.⁵³⁻⁵⁶ The effect has been noted to be proportional to the number of decibels by which the overall SPL exceeds 70 dB, up to 110 dB at least, reaching values that represent changes of as much as 40 percent from resting values. Some observe that vasoconstriction does not completely adapt with time, either on a short-term or long-term basis, and the effects often persist for considerable time after cessation of the noise. Jansen⁵⁵ has suggested that vasoconstriction, with its concomitant effect on the circulatory system in general, will eventually lead to heart trouble. For this statement to be verified, however, there must be considerably more confirmative information as to the lasting (irreversible) effects of noise stimulation upon the cardiovascular system.

As an adjunct to the stress reaction creating a condition of generalized vasoconstriction, observations have been made wherein capillary loops in the cochlea are constricted. This is hypothesized as being another means whereby cochlea damage occurs. Rather than intense sound pressure physically destroying cochlear tissues, these reports indicate a damage mechanism resulting from insufficient oxygen and other nutrients. In brief, the blood supply for the cochlear cells becomes inadequate during intense sound stimulation.⁵⁷⁻⁶¹

Pupillary Dilation

According to Jansen,⁶² noise affects the sympathetic part of what he calls the vegetative nervous system. It is in this realm that he has reported on a number of occasions that eye pupil dilation occurs as one of myriad body reactions to noise exposure. As is the case with cardiovascular effects, the effect is proportional to the intensity of the stimulus in excess of 70 dB SPL, and grows at least to the 110 dB stimulus level. Adaptation over time does not occur.

A neurological basis for pupillary dilation is provided through the complex nerve network for the balance sense. This network, called in part the medial longitudinal fasciculus, sends nervous impulses from the balance mechanism to the cranial nerve which controls pupillary action (CN III—the Oculomotor Nerve). In this context, the pupillary activity caused by high levels of noise may, in fact, be a result of stimulation not of the cochlea but of the vestibular portion of the inner ear which operates the sense of balance.

The significance of this particular physiological reaction is not well understood. It is cited here in the event that future study suggests that a definable and important function in noise reaction is served by pupillary dilation.

Essentially, the reaction to high levels of noise can lead to a condition where the counter relevant forces within the body compete for control, altering the emotions, the general health and stability of human organisms. It remains to be proven whether this condition is as deleterious to health as some have suggested, but there is virtually no support for any notion that this type of exposure is good for one. At least, the results of the noise exposure may not culminate in a definable illness, but there is need to discover whether this exposure adds its stressing effects to the body without a person becoming consciously aware that he is being stressed.

It is not difficult to project some of the information contained in this section into a “dooms-day” prediction. Yet it must be pointed out that the bulk of research on this topic has been conducted with very small nonhuman subjects (rats, guinea pigs, chinchillas). Therefore, the projections to human reactivity cannot be easily made. The most appropriate interpretation of the data is to realize that inordinately great exposure to noise has a potentially deleterious effect upon vital physiological processes and must be avoided if one is to remain free of the types of disturbances such exposure might cause.

Some would state the interpretation even more cautiously, for they hold that the weight of even the nonhuman evidence must be further established. Long-term studies are needed which will ultimately determine whether the alleged devastating side effects of excessive noise exposure are real. To date, the evidence on either side of the argument is incomplete. Man has never before been forced to endure an acoustic environment composed of such frequent and high level sounds as in this age; therefore, his responses to such sound conditions are not fully predictable.

A most important area of investigation is to attempt ways to learn if there is such a thing as a “threshold” of irreversable physiological damage. As stress occurs, does the body return fully to the previous state within a reasonable period after the stressing condition? How many recurrences of noise stress are necessary to bring about some irreversable stress reaction which might lead to any of several disorder conditions? Answers to these and similar questions must be found prior to our full understanding of noise as a stressor.

EFFECTS OF NOISE ON SLEEP

There exists evidence that noise may interfere with sleep. At high noise levels, 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, dreamless stage to a lighter stage of sleep. However, much of what we know about sleep comes from experiments in the laboratory on a few people. Caution must therefore, be exercised in making generalizations about the general population. During a normal night as a person relaxes and enters a stage of drowsiness, the EEG pattern shows a transition from rapid, irregular waves to a regular pattern, known as the alpha rhythm. At this stage, the person is "asleep." As time progresses, the pattern disappears altogether and is replaced by low-voltage, fast irregular waves, known as the beta stage of sleep. During this stage, rapid eye movements can be observed. It is usually considered that during this stage of sleep dreaming occurs.

As time progresses further, the EEG pattern shows quick bursts of longer amplitude, low frequency waves, known as delta waves. Later on, the spindles disappear and are replaced by delta waves of greater magnitude and lower frequency. The two last stages of sleep, spindles and delta waves, are known as the deep stages of sleep.⁶³⁻⁶⁵ The various stages of sleep described above are referred to as 1,2,3 and 4 stages of sleep with the exception of the second stage which is normally called REM (rapid eye movement stage).

Normally, a person will go through the progression described above with occasional reversals. The amount of time spent between deep sleep and lighter stages of sleep is somewhat dependent upon age; however, it is usually considered that all stages of sleep are necessary for good physiological and psychological health.

The effects of acoustic stimulation on sleep depend upon several factors:

1. The nature of the stimulus.
2. The stage of sleep the person is in.
3. Instructions to the subject and his psychophysiological and motivational state.
4. Individual differences, e.g. sex, age, physical condition and psychopathology.⁶⁶

For the purpose of this document we will review the relationship between noise and sleep in terms of each of the factors listed above.

NATURE OF STIMULUS

The likelihood of noise interference with sleep is greatly dependent upon the noise level. Studies have indicated that the effect of noise on sleep becomes increasingly apparent as ambient noise levels exceed about 35 dBA.⁶⁷ Thiessen found that the probability of subjects being awakened by a peak sound level of 40 dBA was 5 percent, increasing to 30 percent at 70 dBA. Including consideration of EEG changes, the probability increases to 10 percent for 40 dBA and 60 percent for 70 dBA.⁶⁸ Karagodina *et al.*,⁶⁹ observed that subjects who slept well (based on psychomotor

activity data) at 35 dBA, complained about sleep disturbance and had difficulty in falling asleep at 40 dBA, and at 50 dBA. These subjects took over an hour to fall asleep initially, with awakening occurring often during the sleep period. These data formed part of the basis for Karagodina's *et al* suggestion of 30-35 dBA as the maximum allowable noise limits for noise inside apartments, with the 30 dBA level applicable to nighttime, when sensitivity to noise is increased.

Grandjean⁷⁰ proposed that noise should not exceed 35 phons during the night in order to preserve the beneficial restorative processes of sleep, although individual differences in tolerance to noise were found to range from 30 to 70 phons. Beland *et al.*⁶⁷ also suggest a maximum allowable steady-state noise level of 35 dBA for sleeping, based on studies of community reaction to aircraft noises.

There seems to be some agreement that moderate noise levels (70-80 dBA, even as low as 48-62 dBA) result in EEG changes in human sleep patterns, manifested especially by an initial depression or interruption of alpha rhythm.⁷¹ Thiessen found that for sound stimuli at 70 dBA, the most likely reaction was to awaken, followed by shifts in sleep stages.⁷² At 50 dBA there was 50 percent chance that no reaction would occur, with the remaining 50 percent about equally divided between the following four levels of responses:

1. Slight change in EEG pattern lasting a few seconds and detectable only on the recording chart.
2. Pattern change lasting up to one minute and usually only detectable on the chart.
3. Sleep level change easily observed by analysis of the magnetic tape record.
4. Awakening.

With 40-45 dBA sound levels there was still a greater than 10 percent probability that a response would result. This response was either a change in sleep stage or awakening.

It is usually reported that subjects who have been deprived of sleep require more intense auditory stimuli in order to awaken than do normally rested persons.⁷³ In addition, if the number of sound peaks increases, the subject will take longer to fall asleep even if the average sound level decreases.

It has also been reported that brief acoustic stimuli of low frequencies (100 Hz) and fast rise time (1 msec) are most effective in eliciting EEG-K-complex in stage 2 of sleep.⁷⁴ These findings have been confirmed by Williams.⁶⁶

Berry and Thiessen compared the effects of impulsive tone bursts with simulated sonic booms and truck noise (with a maximum intensity of 70 dBA).⁷⁵ They observed that frequency of awakening is lower for impulsive noise. Peak level for impulsive noise has apparently no significant effect on the response, although increases in level for truck noise and subsonic jet flyover do increase the frequency of awakenings and shifts in sleep stages.

Mery et al. used artificial crescendos of white noise, aircraft flyovers, and traffic noise as stimuli in a number of experiments. They found that everything else being equal, low density traffic noise are more sleep-disruptive than high density noises.⁷⁶

Other researchers have observed that weak stimuli that are either unexpected or novel may have an effect on sleep.⁷⁷⁻⁷⁸ Furthermore Williams (1973) reports studies by Buendia *et al.*, 1966 and Schect *et al.*, 1968 which suggest that differentiated responses acquired during waking to specific acoustic stimuli persist during sleep in both animals and humans.

The rate or presentation of stimuli has also been found to have a significant effect on sleep.⁷⁹ Schieber *et al.* found that low density traffic sounds (61 dB) are more disruptive of sleep than high density traffic, thus confirming the results of Mery *et al.*⁸⁰

STAGES OF SLEEP

It is found that the effect of noise on sleep is very much dependent upon the stage of sleep. Results of some studies suggest that thresholds for awakening appear to be lower in sleep stage REM for both ordinary noise and sonic booms.⁸¹ Evans *et al.* (1966) were able to elicit relatively complex motor responses to verbal instructions in REM stage of sleep.⁸²

Auditory stimuli presented during stages 3 and 4 generally do not result in complete awakening, but in more than 30 percent of the cases, produce shifts to stage 2.⁷⁵

The amount of accumulated sleep time also affects the probability of awakening, with arousal more likely to occur after longer periods of sleep, no matter what the stage of sleep.^{66,73,79}

MOTIVATION OF SUBJECT

Motivation or familiarity of the subject with the noise source may be a factor in the degree of arousal during sleep.⁸³ The ability of sleepers to discriminate among stimuli of various sorts has been observed especially if the discrimination was learned when the subject was awake.⁷³⁻⁸⁴

In general it is found that effects of motivation on sleep disturbance are somewhat dependent upon the stage of sleep.⁸⁵ These results are confirmed by Zung and Wilson who demonstrated that instructions and financial incentives produce an increase in frequency of stage shifts and awaking following presentations of moderate sound stimuli.⁸⁴

Instructions given to subjects before sleep may influence the effects of noise on sleep. Researchers at the FAA Civil Aeromedical Institute in Oklahoma City employed simulated booms which they did not label as sonic booms to investigate the effects of booms on sleep behavior, moods and performance. They instructed their subjects "to ignore disturbances and attempt to get the best night's sleep possible." They found that the number of responses to booms were smaller than those expected on the basis of the data presented by Lukas and Kryter.⁸⁶

DIFFERENCES OF AGE

A number of studies have indicated that children and young persons are less affected by noise in all stages of sleep than middleaged and older persons.⁸⁷⁻⁹¹ There is no evidence that children are especially sensitive to sleep disturbance by noise.⁹² However, since general sleep disturbance, in the form of nightmares, enuresis, etc., occurs commonly in children aged 4-6 years, it is possible that noise may have some effect on this age group, especially since this age group appears to be particularly disturbed by sudden arousal from sleep stage 4.⁶⁵

Although the sleep of very young children is less disturbed by noise than that of adults or the elderly it has been claimed that babies who have had gestational difficulties or have been brain injured are particularly sensitive to noise.⁹³

DIFFERENCES OF SEX

It has been claimed that women are more sensitive to noise during sleep than men.^{84,89,94} Lukas and Dobb (1972) found that middle aged women are particularly sensitive to subsonic jet aircraft flyovers and simulated sonic booms.⁹⁵

Adaption to Noise

The question of whether or not adaptation to noise during sleep takes place is the subject of considerable debate. Adaptation in this context means whether or not repeated exposure to sound stimuli during sleep will result in progressively less interference with normal sleep. Lukas and Dobbs have indicated that some adaptation does take place in studies of sonic booms during stage 2 of sleep.⁹⁵ Bartus has argued on the other hand that adaptation does not occur.⁸³ Some tests performed by the National Research Council of Canada indicate that awakening response does seem to lessen with time, but there is not adaptation of the average response.⁹⁶

Ando *et al.* found, in a study of women who had moved to Itami City, near Osaka Airport in Japan, during pregnancy, that it was possible that some sort of adaptation occurred in the fetus. 48 percent of the women who had moved to the area in the first 5 months of pregnancy said that their infants slept soundly on exposure to air craft noise after birth. This was true for less than 15 percent of the infants whose mothers had moved in the latter 5 months of pregnancy.⁹⁷

Conclusions

The discussion above indicates that sleeping in noisy surroundings does produce some effects on sleep either in the form of awakening, if the noise is loud enough, or in the form of shifts in the stages of sleep. Usually, however, much of our data comes from laboratory experiments that involve few people, and "responses" are evaluated in terms of physiological measurements such as EEG. Caution must therefore be exercised in drawing conclusions regarding the effect of noise on the

sleep of the general population. Even greater caution must be exercised in making references about the longrange effect of sleep disturbance since there exist very little experimental data regarding these longterm effects. We know, however, that sleep may be interfered with by noise and that some groups (such as the old and middle age and the sick) are particularly sensitive to these effects. Since sleep is thought to be a restorative process during which the organs of the body renew their supply of energy and nutritive elements, noise could be a health hazard.

Further, we also know that survey data indicate that sleep disturbance is often the principal reason given for noise annoyance.⁹⁸ Since it lowers the quality of life, interference with sleep by noise constitutes a health hazard within the frame of reference of the World Health Organization definition of Health.

THE EFFECT OF NOISE ON GENERAL HEALTH AND MENTAL HEALTH

Personal health includes a wide variety of conditions and mental states (see definition of health in Section 1). The complexity of the human body is great, and coupled with the complexity of human mental function, it is extremely difficult to quantify "health effects" in the wake of stimulation by noise. Individual variations from day to day in susceptibility to physical and mental health conditions add a further complicating factor.

It has been said that one person's noise is another's music. Mental set, orientation, personality, general health, and a myriad of other personal factors confound the attempt to fully and comprehensively recognize all of the ramifications of the effect of noise on general health and mental health. In all, there is relatively little known about the effects of noise upon general health and mental health.⁹⁹

Fatigue

Fatigue, in the sense of subjectively described weariness or nervous exhaustion, is so highly individualized that a clear understanding of it is difficult to ascertain. Fatigue, in the medical and physiological sense, is indicated by the occurrence of increased pulse frequency, decrease in pulse pressure, a rise in pulmonary ventilation and slight augmentation of oxygen consumption.¹⁰⁰ In addition, fatigue is described as resulting from the exhaustion of metabolic reserves that leads to a measurable change in the cardiovascular and respiratory systems. Further, blood glucose levels decline and serum cholesterol levels increase. Fatigue does not ordinarily impair the ability to complete tasks, rather, it lowers the motivation to perform.¹⁰¹ (See the discussion on Effects of Noise on Performance—Section 8).

The extent to which noise exposure contributes to fatigue is difficult to assess. In using extremely intense levels of infrasound, aerospace researchers¹⁰¹ have induced symptoms of

extreme fatigue in their subjects.¹⁰² However, the exposure conditions for those subjects were highly atypical.

A study was conducted by the U. S. Public Health Service in 1941, to determine the relationship between fatigue and driving conditions among interstate truck drivers.¹⁰³ The results of various psychological and physiological tests demonstrated that, with increasing hours of driving, there was a gradual and progressive diminution in certain bodily functions. The most consistent changes were found in certain dexterity test results and manual steadiness. Physiologic changes recorded after driving for prolonged periods of time included:

- Heart rate.
- Blood pressure.
- White blood cell counts.

Interestingly, the medical findings of fatigue and the drivers' independent judgement of apparent fatigue correlated quite highly. In that study, no attempt was made to relate any of the observations to noise exposure.

In a more recent study reported by Aston and Janway,¹⁰⁴ truck drivers were subjected to truck vibration. The results of their investigation led Aston and Janway to conclude that vehicle vibration is not intense enough to cause the severe conditions created in laboratory studies of vibration effects on the body. However, they did offer the suggestion that chronic exposure to vibration, especially of very low frequencies (5-7 Hz), could provide sufficient cumulative insult that, coupled with other infective or pulmonary disorders, long-term pulmonary debilitation might occur. (See also Section 10).

General Health Effects

Noise is considered to be a contributor to adverse health influences as well. Numerous conditions have been attributed to noise exposure, such as:

- Nausea.
- Headaches.
- Irritability.
- Instability.
- Argumentativeness.
- Reduction in sexual drive.
- Anxiety.
- Nervousness.
- Insomnia (and its opposite, abnormal somnolence).
- Loss of appetite.
- Other ailments.

These complaints are difficult to assess, not only because of their essentially subjective nature, but also because intense noises are often associated with working conditions that, even without noise, involve stress (including fear) which could account for many of the symptoms with or without the influence of noise. For example, Jansen's study¹⁰⁵ on workers in high-intensity noise gave evidence of higher circulatory problems. Higher incidence of fatigue and irritability leading to social conflicts was also found. By contrast, Felton and Spencer,¹⁰⁶ in a comparison of 50 jet engine testers with 55 control subjects, concluded that noise had nothing to do with morale on the job.

There are some interesting, but difficult to explain, statistics reported by Carosi and Calabro.¹⁰⁷ In a comparison of 330 families in Naples in which either the husband or wife worked in noisy industry (metalwork or industrial weaving) with a control group of 200 non-noise-exposed families matched for age, they found that while 69 percent of the non-noise-exposed families had two or more children, only 24 percent of the noise-exposed families had that many children. If these data were taken at face value, one might conclude that high-level industrial noise exposure reduces human reproductivity or the drive for sexual activity (or both). However, conclusions are premature.

A few attempts have been made to evaluate the health-related aspects of noise stimulation in special environments. For example, Brewer and Briess¹⁰⁸ suggested that non-auditory effects of noise exposure in industry included the development of coughs, hoarseness, lesions, and pains in the throat caused by the strain of shouting above the noise. In another industrial population, Buyniski¹⁰⁹ reported that deaf industrial workers made more trips to the dispensary than did their normal-¹¹² hearing counterparts. Unfortunately, Buyniski did not define the "deafness" of his subjects.

Some have considered noise in a hospital environment to be detrimental to the recovery process of patients.¹¹⁰⁻¹¹² However, this concern has not been verified by data at the present time. Goshen¹¹³ has described as erroneous the conception that because ill health produces discomfort, discomfort can produce ill health. He continued by making the point that sound stimulation, such as that frequently encountered in the hospital environment, might be just as vital in augmenting the recovery of patients as some feel it might be in hampering recovery. Kryter⁹⁹ contended that helpful adaptation to noise would occur very rapidly in an organism which, for some physiological or psychological reason of health, should not be aroused.

Sleep disturbance, the subject of another portion of this chapter, should be mentioned as another possible contributor to the effects of noise on general health. Several authors¹¹⁴⁻¹¹⁷ have stated that sleep interruption or sleep modification due to noise exposure is one of the most harmful conditions noise poses for an individual's health.

Mental Health Effects

One of the most serious charges against noise in the environment has been issued by those who state that noise can adversely affect mental health. A widely-cited report by Abey-Wickrama

*et al*¹¹⁸ stated that aircraft noise contributes to mental illness. In the study, 488 admissions to a psychiatric hospital were divided into two groups. One group consisted of persons who resided in what the researchers classified as a "maximum noise area" (MNA) near London's Heathrow Airport. The other group were residents of the same borough, but they lived outside the MNA. According to rough estimates of the total population represented by the groups, the MNA contained approximately half the number of residents found in the non-MNA. The two groups of psychiatric admissions were equal in number, leading the observers to surmise that the prevalence of mental problems in the MNA was twice that of the non-MNA. Criticisms of technique, control, and inference by the scientific community have been sufficiently great that Herridge¹¹⁹ has indicated that a much more tightly controlled survey is currently underway in the same region of London.

One cannot rule out the possibility that noise exposure not only can eventually produce hearing loss, but also may pose some other health hazard if no attempt is made to reduce individual exposure to noise. Caution must be exercised in interpreting the results of studies in this realm, however, for controls are exceptionally difficult to exercise and quantification of the data is far from easy.

SUMMARY—EFFECTS OF NOISE ON AUTONOMIC NERVOUS SYSTEM FUNCTIONS AND OTHER SYSTEMS

Noise can elicit many different physiological responses. However, no clear evidence exists indicating that the continued activation of these responses leads to irreversible changes and permanent health effects. Sound of sufficient intensity can cause pain to the auditory systems. Except for those persons with poorly designed hearing aids, such intense exposures should not normally be encountered in the non-occupational environment. Noise can also effect the equilibrium of man, but the scarce data available indicates that the intensities required must be quite high or similar to the intensities that produce pain.

Noise-induced orienting reflexes serve to locate the source of a sudden sound and, in combination with the startle reflex, prepare the individual to take appropriate action in the event danger is present. Apart from possibly increasing the chance of an accident in some situations, there are no clear indications that the effects are harmful since these effects are of short duration and do not cause long time body changes.

Noise can interfere with sleep; however, the problem of relating noise exposure level to quality of sleep is difficult. Even noise of a very moderate level can change the patterns of sleep, but the determination of the significance of these changes is still an open question.

Noise exposure may cause fatigue, irritability, or insomnia in some individuals, but the quantitative evidence in this regard is unclear. No firm relationships between noise and these factors can be established at this time.

Noise exposure can be presumed to cause general stress by itself or in conjunction with other stressors. Neither the relationship between noise exposure and stress nor the threshold noise level or duration at which stress may appear has been resolved.

Noise exposure to moderate intensities likely to be found in the environment affects the cardiovascular system in various ways; however, no definite permanent effects on the circulatory system have been demonstrated. Noise of moderate intensities has been found to cause vasoconstriction of the peripheral areas of the body and pupillary dilation. Although several hypotheses exist, there is no evidence at this time that these reactions to noisy environments can lead to harmful consequences over a period of time. Speculations that noise might be a contributory factor to circulatory difficulties and heart diseases are not yet supported by scientific data.

REFERENCES

1. Robinson, D.W. and Dadson, R.S., Threshold of Hearing and Equal-loudness Relations for Pure Tones, and the Loudness Function, *J. Acoust. Soc. Amer.*, 29, 1284-1288, 1957.
2. von Gierke, H.E., Davis, H., Eldredge, D.H. and Hardy, J.D., Aural Pain Produced by Sound, in BENOX. REPORT: AN EXPLORATORY STUDY OF THE BIOLOGICAL EFFECTS OF NOISE prepared by Ades, H.W. et al, ONR Project NR 144079, University of Chicago, 1953.
3. Ades, H.W., Graybiel, A., Morill, S., Tolhurst, G., and Niven, J., Non-auditory effects of High Intensity Sound Stimulation on Deaf Human Subjects, Joint Report No. 5, U. Texas Southwestern Med. School, Dallas Texas and U.S. Naval School of Aviation Medicine, Pensacola, Fla., 1958.
4. Davis, H. and Silverman, S.R., HEARING AND DEAFNESS, Holt, Rinehart and Winston, New York, 1970.
5. Fowler, E.P., Marked Deafened Areas in Normal Ears, *Arch. Otolaryng.*, 8, 151-155, 1928.
6. 18th Annual Facts and Figures, *National Hearing Aid Journal*, 6, Nov. 1972.
7. Minnesota Public Interest Research Group, HEARING AIDS AND THE HEARING AID INDUSTRY IN MINNESOTA, 3036 Univ. Ave. Southeast, Minneapolis, Minn., Nov. 1973.
8. Tullio, P., Sulla Funzione Delle Varie Parti Dell'Orecchio Interno, 1926.
9. Kacker, W.K. and Hinchcliffe, R., Unusual Tullio Phenomena, *J. Laryng. & Otol.*, 84, No. 2, 155-166, 1958.
10. Dickson, E.D.D. and Chadwick, D.L., Observations on Disturbances of Equilibrium and other Symptoms Induced by Jet Engine Noise, *J. Laryngol. Otol.*, 65, 154-165, 1951.
11. Nixon, C.W., Harris, C. and von Gierke, H.E., Rail Test to Evaluate Equilibrium in Low-Level Wideband Noise, *Report AMRL-TR-66-85*, U. S. Air Force Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio, 1966.
12. McCabe, B.F. and Lawrence, M., Effects of Intense Sound on Non-Auditory Labyrinth, *Acta Oto-Laryngol.*, 49, 147-157, 1958.
13. Lipscomb, David M. and Roettger, Ruby L., Capillary Constriction in Cochlear and Vestibular Tissues during Intense Noise Stimulation, *Laryngoscope*, 83, No. 2, 259-263, February, 1973.
14. Dieroff, H.G. and Scholtz, H.J., Zur Frage der Larmbedingten Vestibularisschaden bei begutachteten Larmarbeitern, *Z. Laryngol. Rhiol. Otol.*, 46, 746-757, 1967.
15. Moline, J., Reflexes Oculaires d'origine Auditive, *Rev. Laryng.*, 1, 385-393, 1916.

16. Davis, R.C., Motor Responses to Auditory Stimuli above and below Threshold, *J. Exp. Psychol.*, 40, 107-120.
17. Galambos, Robert, Rosenberg, Philip E. and Glorig, Aram, The Eyeblink Response as a Test for Hearing, *JSHD*, 18, 373-378, 1953.
18. Frisina, D. Robert, MODERN DEVELOPMENTS IN AUDIOLOGY, Measurement of Hearing in Children, James Jerger, (ed.), New York: Academic Press, 1963.
19. Gesell, A. and Armatruda, C., DEVELOPMENTAL DIAGNOSIS, New York: Harper (HOEBER), 1948.
20. Landis, C. and Hunt, W.A., THE STARTLE PATTERN, New York: Farrar and Rinehart, Inc., 1968.
21. Straus H., Landis, C. and Hunt, W.A., Acoustic Motor Reactions Especially the Cochleopalpebral Reflex, *Arch. Oto. Laryng*, 28, 941-945, 1938.
22. Davis, R.C. and Van Liere, D.W., Adaptation of the Muscular Tension Response to Gunfire, *J. Exp. Psychol.*, 39, 114-117, 1949.
23. Davis, R.C., Motor Effects of Strong Auditory Stimuli, *J. Exp. Psychol.*, 38, 257-275, 1948.
24. Davis, R.C., Response to "Meaningful" and "Meaningless" Sounds, *J. Exp. Psychol.*, 38, 744-756, 1948.
25. Davis, R.C., Buchwald, A.M. and Frankman, R.W., Autonomic and Muscular Responses and Their Relation to Simple Stimuli, *Psychol. Nomographs*, 69, No. 405, 1955.
26. Davis, R.C. and Berry, T., Gastrointestinal Reactions to a Response-Contingent Stimulation, *Psychol. Rep.*, 15, 95-113, 1964.
27. Miller, James, Effects of Noise on People, (NTID 300.7), December 31, 1971.
28. Davis, R.C., Buchwald, A.M. and Frankman, R.W., Autonomic and Muscular Responses and Their Relation to Simple Stimuli, *Psychol. Nomographs*, 69, No. 405, 1955.
29. Welch, B.L. and Welch A.S., (eds.), PHYSIOLOGICAL EFFECTS OF NOISE, New York: Plenum Press, 365, 1970.
30. Selye, H., THE STRESS OF LIFE, New York: McGraw-Hill Book Co., 324, 1956.
31. Lipscomb, David M., INDICATORS OF ENVIRONMENTAL NOISE, Indicators of Environmental Noise, William A. Thomas (ed.), New York: Plenum Press, 1972.
32. Sayers G: The adrenal cortex and homeostasis. *Physiol Rev* 30:241-320, 1950.
33. Harris GW: Neural control of the pituitary gland. *Physiol Rev* 28:139-179.
34. McCann SM: Effect of hypothalamic lesions on the adrenal cortical response to stress in the rat. *Am J Physiol* 175:13-20, 1953.
35. Brodish A, Long CNH: Changes in blood ACTH under various experimental conditions studied by means of a cross-circulation technique. *Endocrinology* 59:666-676, 1956.
36. Henkin RI, Knigge KM: Effect of sound on the hypothalamic-pituitary-adrenal axis. *Am J Physiol* 204:710-714, 1963.

37. Osintseva VP: Noise-induced changes in the adrenals. *Gigiena i Sanitariya* 34:147-151, 1969.
38. Geber WF, Anderson TA, Van Dyne V: Physiologic responses of the albino rat to chronic noise stress. *Arch Environmental Health* 12:751-754, 1966.
39. Anthony A, Ackerman E: Effects of noise on the blood eosinophil levels and adrenals of mice. *J Acoust Soc Am* 27:1144-1149, 1955.
40. Arguelles AE, Ibeas D, Ottone JP, Chekherdemian M: Pituitary-adrenal stimulation by sound of different frequencies. *J Clin Endocrinology* 22:846-852, 1962.
41. Selye H: Stress and disease. *Science* 122:625-631, 1955.
42. Rosecrans JA, Watzman N, Buckley JP: The production of hypertension in male albino rats subjected to experimental stress. *Biochemical Pharmacology* 15:1707-1718, 1966.
43. Ogle CW, Lockett M: The urinary changes induced by high pitched sounds (20 kcyc/sec). *J of Endoc* 42:253-260.
44. Arguelles AE, Martinez MA, Pucciarelli E, Disisto MV; Endocrine and metabolic effects of noise in normal, hypertensive and psychotic subjects, in *Physiological Effects of Noise*, edited by Welch BL, Welch AS. New York, Plenum Press, 1970, p. 43-55.
45. Jirkova, H. and Krcmarova, B., Studies of the Influence of Noise on the General Health of Workers in Large Engineering Work, an Attempt at Evaluation (in Polish), *Pracooni Lekarstvi*, Prague, 17, No. 4, 147-148, 1965.
46. Etholm B. and Egenberg, K.E., The Influence of Noise on some Circulatory Functions, *Acta Oto-Laryngol.*, 58, 208-213, 1964.
47. Blazekova, L., Preliminary Results of Investigation of Noise Effect on some Vegetative Functions, *Pracov. Lek.*, 18, 276-279, 1966.
48. Klein, K. and Grubl, M., Uber Hamodynamische Reaktionen unter Akustischen Reizen, Hemodynamic Reactions to Acoustic Stimuli, *Wien. Klin. Wschr.*, 81/40, 705-709, 1969, *Exc. Med.*, 2550, 1970.
49. Jansen, G., Adverse Effects of Noise on Iron and Steel Workers, *Stahl. Eisen.*, 81, 217-220, 1961.
50. Andrinkin, A., Influence of Sound Stimulation on the Development of Hypertension, (cited in Kryter, 1970), *Cor. Et Vasa*, 3, 285-293, 1961.
51. Kryter, K., THE EFFECTS OF NOISE ON MAN, New York: Academic Press, 633, 1970.
52. Satalov, N.N., Ostapkovic, V.E. and Ponomareva, N.I., Sostojanie Sluha i Arterial, 'nogo Davlenija pri Vozdejstvii Intensivnogo Proizvodstvennogo Suma, Hearing and Arterial Blood Pressure in Persons Exposed to Intense Industrial Noise, *Gigiena Truda i Professional'nye (Moscow)*, 13, No. 4, 12-15, April, 1969.
53. Lehman, G. and Tamm, J. Changes of Circulatory Dynamics of Resting Men Under the Effects of Noise, *Intern Z. Agnew Physiol.*, 16, 217-227, 1956.

54. Jansen, G., Zur Neriosen Belasting durch Laim, *Beihefte Zum Zentralblatt fir Arbirts-medizion und Arbitsschutz*, Dr. Dietrich Steinkopff Verlag, Darmstadt, 9, 1967.
55. Jansen, G., Effects of Noise on Physiological State in *Noise as a Public Health Hazard*, W. Ward and J. Fricke, (eds.), (ASHA Reports. 4, Amer. Speech Hearing Assoc., Washington, D.C., 89-98, 1969.
56. Grandjean, E., Physiologische and Psyshologische Wirkungen des Laerms, *Memsch rend Umwelt, Documenta Geigy*, 4, 13-42, 1960.
57. Lawrence, Merle, Effects of Interference with Terminal Blood Supply on Organ of Corti, *Laryngoscope*, 76, No. 8, 1318-1337, 1966.
58. Lawrence, Merle, Circulation in the Capillaries of the Basilar Membrane, *Laryngoscope*, 80, No. 9, 1364-1375, 1970.
59. Lawrence, Merle, Gonzalez, G. and Hawkins, Joseph E., Jr., Some Physiological Factors in Noise-Induced Hearing Loss, *Am. Indust. Hyg. Assn. J.*, 28, 1967.
60. Hawkins, J.E., Jr., The Role of Vasoconstriction in Noise-Induced Hearing Loss, *Ann. OR. and L.*, 80, No. 6, 903-914, February, 1973.
61. Lipscomb, David M. and Roettger, Ruby L., Capillary Constriction in Cochlear and Vestibular Tissues during Intense Noise Stimulation, *Laryngoscope*, 83, No. 2, 259-263, February, 1973.
62. Jansen, Gerd, PHYSIOLOGICAL EFFECTS OF NOISE, Relation Between Temporary Threshold Shift and Peripheral Circulatory Effects of Sound, Welch and Welch, (eds.), New York: Plenum Press, 1970.
63. Dement, W.C. and Klitman, N., Cyclic Variations in EEG During Sleep and their Relation to Eye Movements, Body Mobility and Dreaming *Electroencephal. Clin. Neurophysiol.*, 9, p. 673, 1957.
64. Dement, W. Recent Studies on the Biological Role of Rapid Eye Movement Sleep. *The American Journal of Psychiatry*, 22, p. 404, 1965.
65. Miller, J.D., Effects of Noise on People, U.S. Environmental Protection Agency, *NTID 300.7*, 1971.
66. Williams, H.L., Effects of Noise on Sleep: a Review, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
67. Beland, R.D., Bishop, D.E. and Lafer, S.K., Aircraft Noise Impact-Planning Guidelines for Local Agencies, South Pasadena, Wiley and Ham, *Rept. No. 979-1*, HUD contr. No. H-1675, 36-44, 1972.
68. Thiessen, G.J., Effects of Noise from Passing Trucks on Sleep, Rept. Q1 presented at 77th Mtg. Acoustical Society of America, Philadelphia, April, 1969.
69. Karagodina, I.L., Osipov, G.L. and Shishkin, I.A., Effects of Apartment Noise on Sleep, the Flight Against Noise in Cities, Karagodina, I.L. ed., pg. 38-39 Moscow: 1972.

70. Grandjean, E. Biological Effects of Noise, Congr. Rept. II, 4th International Congress of Acoustics, Copenhagen, August 1962.
71. Andreyeva - Galanina, Y.T., Noise and Noise Disease, *Meditina*, 304, Leningrad: 1972.
72. Thiessen, G.V., Effects of Noise during Sleep, in Physiological Effects of Noise, Welch, B.L. and Walch, A.S. eds., New York: Plenum Press, 1970.
73. Williams, H.L. and Williams, C.L. Nocturnal EEG profiles and performance, *Psychophysiol* 3, 164-175, 1966.
74. Vetter, K. and Hor Vath, S.M., Effects of Audiometrie Parameters on K-Complex of Electroencephalogram, *Psychiatry and Neurology*, 144, 103-109, 1962.
75. Berry, B. and Thiessen, G.J. Effects of Impulsiva Noise on Sleep National Research Council of Canada, *NRC 11597*, 36, 1970.
76. Mery, J., Muzet, A., and Schieber, J.P., Effects du Bruit d'Avions Sur le Sommeil, in *Proceedings of 7th International Congress of Acoustic*, 3, Budapest 509-512, 1971.
77. Oswald, I., Taylor, A.M., and Triesman, M., Discriminative Responses to Stimulation during Human Sleep, *Brain*, 83, 440-553, 1960.
78. Lehmaun, D. and Koukkov, M. Das EEG des Menschen beim Lernen von Neuem und Bekanntem Material, *Anch. Psychiat. Nerveukr.*, 215, 22-32, 1971.
79. Lukas, V.S. and Kryter, K.D. Awakening Effects of Stimulated Sonic Booms and Subsonic Aircraft Noise, in Physiological Effects of Noise, Welch, B.L. and Welch, A.S., eds., 283-293 Plenum Press, New York: 1970.
80. Schieber, J.P., Mery, J. and Muzet A., Etude Analytique en Laboratoire de l' Influence du Bruit Sur le Sommeil, *Rept. of Ceutie d' Etudes Bioclimatiques du CNRS*, Stransbery, France, 1968.
81. Rice, C.G. Sonic Boom Exposure Effects, *J. Sound and Vibration*, 20, 511-517, 1972.
82. Evans, F.V., Gustafson, L.A., O'Connell, D.N., Orue, M.T. and Shon, R.E., Response During Sleep with Intervening Waking Amnesia, *Science*, 152, 666-667, 1966.
83. Bartus, R.T., Hart, F.D. and LeVere, T.E. Bleetioencephalographic and Behavioral Effects of Nocturnally-Occuring Jet Sounds, *Aerosp. Med.*, 384-389, 1972.
84. Wilson, W.P. and Zung, W.W.K. Attention, Discrimination and Arousal during Sleep, *Anch, Gen, Psyshiat.*, 15, 523-528, 1966.
85. Miller, J.D. Effects of Noise on People, U.S. Environmental Protection Agency Document, *NTID 300.7*, 1971.
86. Collins, W.E. and Iampiateo, P.F. Effects on Sleep of Hourly Presentations of Simulated Sonic Booms, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yougoslavia, 1973.
87. Dobbs, M.E. Behavioral Responses to Auditory Stimulation during Sleep, *J. Sound and Vibration*, 20, 467-476, 1972.

88. Kramer, M., Roth, T., Trmdar, J. and Cohen, A. Noise Disturbance and Sleep, Final Report, *FAA-No-70-16*, p. 175, 1971.
89. Lukas, J.S. Awakening Effects of Simulated Sonic Booms and Aircraft Noise on Men and Women, *J. Sound and Vibration*, 20, 457-466, 1972.
90. Lukas, J.S. and Kryter, K.D. Awakening Effects of Simulated Sonic Aircraft Noise on Six Subjects 7 to 72 years of age. *NASA Report No. CR-1599*, Washington, D.C., 1970.
91. Nixon, C.W. and Von Gierke, H.E., Human Response to Sonic Boom in the Laboratory and the Community, *NASA*, 51, 766-782, 1972.
92. Lukas, J.S. and Kryter, K.D. Disturbance of Human Sleep by Subsonic Jet Aircraft Noise and Simulated Sonic Booms, *NASA Report No. CR-1780*, 68, 1971.
93. Murphy, K.P. Differential Diagnosis of Impaired Hearing in Children, *Develop. Med. Child. Neurol.*, 11, 561, 1969.
94. Steinicke, G. Die Wirkungen von Lärm auf den Schlaf des Menschen. Forschungsberichte des Wirtschaftes-v. Verkehr-Sministenium Nordrhein Westfalen No. 416, 1957.
95. Lukas, J.S. and Dobbs, M.E. Effects of Aircraft Noise on the Sleep of Women. *NASA Contractor Report CR-2041*, 1972.
96. Thiessen, G.J. Noise Interference with Sleep, National Research Council of Canada, Ottawa, Canada, 1972.
97. Ando, Y. and Haltori, H. Effects of Intense Noise During Fetal Life upon Postnatal Adaptability, *JASA*, 1128-1130, 1970.
98. Alexandra, A. Les Effects du Bruit Seu le Sommeil, *Nuisances et Environnement* August-September, Paris, France, 1972.
99. Kryter, Karl D. THE EFFECTS OF NOISE ON MAN. New York: Academic Press 1970.
100. Glasser, Otto, MEDICAL PHYSICS, Volume III, Chicago: The Year Book Publishers, Inc., 1960.
101. Morgan, C.T., Cook, J.S., Chapanis, A., and Lund, M.W. HUMAN ENGINEERING GUIDE TO EQUIPMENT DESIGN. New York: McGraw-Hill Book Company, Inc. 1963.
102. Mohr, G.C., Cole, J.N., Guild, E. and Von Gierke, H.E., Effects of Low Frequency and Infrasonic Noise on Man, *AMRL-TR-65-69*, U.S. Air Force Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio 1966.
103. Jones, B.F., Flinn, R.H., and Hammond, E.C. Fatigue and Hours of Service of Interstate Truck Drivers. U.S. Public Health Bulletin No. 2b5. Government Printing Office, Washington, D.C. 1941.
104. Aston, Ray and Janeway, R.N., OVER THE ROAD TO PREVENTABLE DISEASE, Total Body Vibration, compiled by the International Brotherhood of Teamsters, (undated).

105. Jansen, G., Vegetative Larmwirkungen bei Industriearbeitern, *Larmbekämpfung*, 6, 126-128, 1962.
106. Felton, J. and Spencer, C., Morale of Workers Exposed to High Levels of Occupational Noise, *Am. Ind. Hyg. Assoc. J.*, 22, 136-147, 1961.
107. Carosi, L. and Calabro, F., La Prolificita di Coniugi Operai di Industrie Rumorose (Prolificacy of Workers in Noisy Industries), *Folia Medica*, 51, 264-268, 1968.
108. Brewer, D.W. and Briess, F.B. Industrial Noise: laryngeal considerations. *N. Y. State J. Med.*, 60, 1737-1740 1960.
109. Buyniski, E.F. Noise and Employee Health. *Noise Control* 4, (6), 45-46, 1958.
110. Bredengerg, V.C. Quiet Please. *Hosp Prog.* 42, 104-108, 1961.
111. Denzel, H.A. Noise and Health, *Science*, 43, 992, 1963.
112. Minckley, B.B. A study of noise and its relationship to patient discomfort in the recovery room. *Nurs. Res.* 17, 247-250, 1968.
113. Goshen, C.E. Noise, annoyance and progress. *Science*, 144, 487, 1964.
114. Grandjean, E. Biological effects of noise. Paper presented at Fourth International Congress on Acoustics, Copenhagen, 1962.
115. Lehmann, G. Sick people and noise. Max-Planck-Institut fur Arbeitsphysiologie, Dortmund, Germany (undated).
116. Richter, R. Sleep disturbances which we are not aware of caused by traffic noise. EEG Station of the Neurological University Clinic, Basel (undated).
117. Jansen, G. and Schulze, J. Beispiele von Scheafstorungen durch gerausche. *Klin. Wachr.* 3, 132-134, 1964.
118. Abey-Wickrama, I., A'Brook, M.F., Gattoni, F.E.G. and Herridge, C.F., Mental-Hospital Admissions and Aircraft Noise, *Lancet*, 297, 1275-1278, December 13, 1968.
119. Herridge, C.F., Observations of the Effect of Aircraft Noise near Heathrow Airport on Mental Health, International Congress on Noise as a Public Health Problem, Dubrovnik, May 17, 1973.

SECTION 8

EFFECTS OF NOISE ON PERFORMANCE

The effect of noise on the performance of tasks has been studied in the laboratory and in the actual work situation, with somewhat more emphasis on laboratory research. Comprehensive reviews of these studies are available.^{1,2,3,4,5}

It is evident that when a task involves auditory signals, whether speech or nonspeech, noise at any intensity sufficient to mask or interfere with the perception of these signals will interfere with the performance of the task. When mental or motor tasks do not involve auditory signals, the effects of noise on their performance have been difficult to assess.³ In many instances, experiments performed to show effects of noise on working efficiency or productivity have been inconclusive or unreliable. Broadbent, Kryter, and others have pointed out that there has not always been adequate control of all the numerous physical and psychological variables that may significantly influence performance. (Much of the preceding data is from *Effects of Noise on People*, by James Miller, EPA, NTID 300.7).

Viewed as a whole, the literature on noise and performance shows that sometimes noise interferes with performance, sometimes it improves it, and usually it causes no significant changes. A number of general conclusions, however, have emerged:

1. Steady noises without special meaning do not seem to interfere with human performance unless the noise level exceeds about 90 dBA and not consistently even then.¹
2. Intermittent and impulsive noises are more disruptive than steady-state noises.² Even when the sound levels of irregular bursts are below 90 dBA they may sometimes interfere with performance of a task.⁶
3. High-frequency components of noise (above about 2000 Hz) usually produce more interference with performance than low-frequency components of noise.
4. Noise usually does not influence the overall rate of work, but high levels of noise may increase the variability of the work rate. There may be "noise pauses" or gaps in response,⁷ sometimes followed by compensating increases in work rate.

5. Noise is more likely to reduce the accuracy of work than to reduce the total quantity of work ^{7,8}.
6. Complex or demanding tasks are more likely to be adversely influenced by noise than simple tasks.⁹

Noise and State of Arousal

Noise does, therefore, have an effect on performance in some situations, depending on the nature of the stimulus, the task involved, and, as some authors have indicated, the state of the individual affected. In 1955, D.O. Hebb¹⁰ proposed that changes in stimulation not only produce cues for an affected organism, but also activate or arouse areas of the cerebral cortex which are involved in response to these cues. Physiologically, this arousal activity originates in the reticular formation, a portion of the central nervous system, and affects one's psychological state as well as all physiological systems. An individual's level of arousal has a great deal to do with the performance of a difficult task. Too little arousal produces inadequate performance, whereas too much arousal interferes with performance. The optimum is somewhere at the top of an inverted U-shaped curve where performance efficiency would form the vertical axis and level of arousal would form the horizontal one. Thus, noise as an arousing stimulus can enhance, fail to affect or interfere with performance of certain tasks.¹¹

Noise as a Distracting Stimulus

Similarly, noise can act as a distracting stimulus, depending on the meaningfulness of the stimulus and also the psychophysiological state of the individual. To quote Broadbent¹² at the Conference on Noise as a Health Problem in Dubrovnik, Yugoslavia, "Human beings have a limit to the number of features of their surroundings which they can perceive in any limited period of time; and therefore anything which happens in the environment has to compete with other events for our attention." According to Broadbent,¹³ the human sensory system acts as a channel of communication receiving all kinds of information, relevant and useless alike. In order to screen out useless information, such as noise, there appears to be a mental "filter". This filter, however, has the following limits:

- It tends to reject or ignore unchanging signals over a period of time, even though they may be important, as in vigilance tasks.

- An individual's state of arousal, stress or fatigue can hinder the mental filter's ability to discriminate.
- The filter can be overridden by irrelevant stimuli which demand attention because of novelty, intensity or unpredictability.

Thus, distraction can occur if the organism is overloaded with other stimuli, or if it is in an otherwise unfavorable physiological state, or if the stimulus is unusually demanding of attention.

Cumulative Effects

At the same conference Broadbent¹² expanded on his theoretical cause for noise-induced decrements in performance. He suggested that exposure to noise can produce an actual change in the state of the individual that is reflected in failures of selective perception. This change is due to a cumulative effect of noise exposure producing measurable aftereffects in the form of performance decrements. As supporting evidence Broadbent mentioned the following studies:

Wilkinson¹⁴ measured the combined effects of sleeplessness and exposure to 100 dB of white noise. He found that relatively short exposures (30 minutes) tended to create a state of arousal which reduced the negative effect of sleeplessness on performance. These same levels of noise impaired efficiency if an individual was at an optimal state of arousal. Significantly, he found that the previously mentioned combination of noise exposure and sleeplessness had disruptive effects when the task was continued over a prolonged period. Evidently this is not a new phenomenon, since other researchers have found that continuous performance in high noise levels (above 90 dBA) may show adverse effects, sometimes after 1/2 hour's exposure.^{15, 7, 16}

Hartley¹⁷ studied the effect of previous exposure to noise on a visual perception task. He exposed one group of subjects to levels of 95 dBC for 20 minutes and another group to 70 dBC while both were relaxing, reading magazines. Then he exposed both groups to 10 minutes of noise at 25 dBC while the test was administered. The group that had been previously exposed to noise showed significantly greater decrements in performance than those exposed to the quieter level. Thus, a cumulative effect of noise was clearly evident.

Aftereffects

In addition to the cumulative effects of noise on performance, some researchers have reported definite aftereffects. Glass and Singer¹⁸ recently reported on 24 studies done over a period of 5 years in which detrimental aftereffects were noticed on such performance

stimulus to a visual perception task, centrally-located visual signal were more effectively perceived, whereas peripherally-located signals tended to be ignored. The theory resulting from these studies is that noise can cause the organism to become selectively perceptive.

Noise Sensitive Tasks

Some tasks have been described in the literature as particularly sensitive to noise. Among them are tasks of vigilance, information gathering and analytical processes.⁴ Vigilance activities are not repetitive, do not allow for self-pacing, and demand rapid and accurate decisions. Therefore, they are more adversely affected by distraction than many other activities. Authorities tend to agree that noise levels above 90 dB Sound Pressure Level are more disruptive in these cases than levels below 90 dB SPL, and that frequencies above 2000 Hz are more disruptive than lower ones.^{23,24} Interestingly, frequencies above 2000 Hz also make better warning signals since they elicit a shorter reaction time.²⁴

Various experiments have shown the disruptive effects of noise on learning or information gathering. Wakely¹¹ points out that noise may interfere by competing for the limited number of channels available for information input. If the system is already overloaded, an individual must take more time to evaluate the usefulness of the intruding stimulus or run the risk of making errors. When tasks are not self-paced, increased errors will result. Jerison²⁵ found that high levels of noise interfere with short-term memory tasks. Experimenters at the Stanford Research Institute found that noise from sonic booms at 1.2 psf can interfere with the learning of an eye-hand coordination skill without impairing the accuracy of the task.

Special Effects

Some particular types of noise give rise to special effects on task performance. Noise of short or varying duration and impulsive noise tend to produce short residual effects on noise-sensitive tasks. Woodhead²⁷ found that one-second noise bursts can have residual effects on performance of from 15 to 30 seconds. She also found that sonic booms of .8 to 2.5 psf produce residual disruptive effects that are thought to be the result of a startle response (as opposed to the orienting response).

Startle responses from sudden loud noises can conceivably impair safety in such situations as construction work, window washing, use of dangerous machinery and even automobile driving. However, field data and reports of accidents show little tangible evidence of this phenomenon.⁴ Berglund, Rylander and Sorenson²⁸ found that sonic booms of approximately .8 to 45 psf that had tangible effects on task performance had no measurable

tasks as proofreading, difficult graphic puzzles, and competitive response tasks. They concluded that these aftereffects could be produced by noises of high intensity, and especially by noise of low predictability and low controllability.

Glass and Singer¹⁸ also found that perceived controllability over aversive sound affected subsequent performance. Experimental subjects were given a switch to pull in order to provide relief from the noise. Even those who did not pull the switch showed better performance afterward than the noise exposed subjects who did not have that choice. The authors hypothesized from the preceding experiments that unpredictability and uncontrollability lead to a feeling of helplessness and frustration that, in turn, lessens motivation for task performance.

Positive and Neutral Effects

Just as frustrating circumstances in combination with noise can hinder performance, positive motivation can enhance it. Numerous experimenters report that praise, encouragement and monetary rewards can enhance performance in noise. Broadbent and Little¹⁹ report a situation where workers' efficiency improved even before acoustical material was installed, presumably because they were pleased that someone was doing something for them.

As previously mentioned, noise does not always degrade performance. It appears that for the majority of tasks, noise has little if any effect. These are the tracking or controlling tasks where noise levels are fairly continuous and where average, rather than instantaneous, levels of performance are sufficient.⁴ Many mechanical or repetitive tasks found in factory work would fall into this category.

In some situations, noise enhances performance. It appears that moderate levels of noise maintained beneficial arousal levels during monotonous tasks. McGrath²⁰ found that various auditory stimuli at 72 dB improved visual vigilance performance. Also, moderate levels of music or background television have been reported to enhance performance, especially among young people. However, acceptable levels for background stimuli tend to decrease with the aging process, probably because of the gradually decreasing efficiency of the central auditory system⁴.

Occasional studies have been reported where noise exposure produces both positive and negative effects on task performance. Woodhead²¹ showed that the introduction of noise during a memory and calculation task adversely affected the calculation portion. However, when noise was introduced into the calculation phase only, performance was improved. Experiments by Hockey²² showed that sometimes high-priority aspects of a task could be enhanced while low-priority aspects were diminished by the presence of noise. He found that by adding a noise

effects on a tracking test that simulated automobile driving. There is evidence, however, that very intense noise, (above 120 dB SPL) may affect manual dexterity due to disturbances of vestibular function.²⁹

Problems in Evaluation

A very real problem in the evaluation of the effects of noise on performance is the lack of well-controlled field studies. Cohen³⁰ has made inroads in this area by reporting on a 5-year study of medical, attendance, and accident files for approximately 1000 workers in factory situations. Five hundred of these workers were employed in noise levels of 95 dBA or above and 500 in 80 dBA or below. The workers located in the higher noise levels showed significantly greater numbers of job-related accidents, sickness and absenteeism than their counterparts in the quieter jobs. However, the reader is cautioned against drawing definitive conclusions because, as Cohen pointed out, the types of jobs in the noisy and quieter areas could not be equated. For example, possibly the tasks in the noisy areas were inherently more hazardous. More definitive information may be available as records continue to be examined, since hearing conservation measures have been initiated, thereby lowering levels of noise exposure. If accident rates, sickness and absenteeism are diminished it will support the inference that high noise levels were a causative factor.

Cohen² points out an important difficulty in generalizing from the laboratory to real-life situations. He notes that laboratory tasks are novel in nature, thereby causing subjects to be fairly well motivated. Also, the actual noise exposures are comparatively short. By contrast, factory and office workers usually work somewhat below their maximum efficiency and respond to many stimuli besides noise. Thus, there are particular research needs for long-term studies in real-life situations.

SUMMARY—PERFORMANCE AND WORK EFFICIENCY

Continuous noise levels above 90 dBA appear to have potentially detrimental effects on human performance, especially on what have been described as noise-sensitive tasks such as vigilance tasks, information gathering and analytical processes. Effects of noise on more routine tasks appear to much less important, although cumulative degrading effects have been demonstrated by researchers. Noise levels of less than 90 dBA can be disruptive, especially if they have predominantly high frequency components, are intermittent, unexpected, or controllable. The amount of disruption is highly dependent on:

- The type of task.
- The state of the human organism.
- The state of morale and motivation.

Noise does not usually influence the overall rate of work, but high levels of noise may increase the variability of the work rate. There may be “noise pauses” or gaps in response, sometimes followed by compensating increases in work rate. Noise is more likely to reduce the accuracy of work than to reduce the total quantity of work. Complex or demanding tasks are more likely to be adversely effected than are simple tasks. Since laboratory studies represent idealized situations there is a pressing need for field studies in real-life conditions.

REFERENCES

1. Broadbent, D.E., Effects of Noise on Behavior, in HANDBOOK OF NOISE CONTROL, C. M. Harris (Ed.), McGraw-Hill: New York, 1957.
2. Cohen, A., Effects of Noise on Psychological State, in NOISE AS A PUBLIC HEALTH HAZARD, ASHA REPORTS #4, American Speech and Hearing Association, Washington, 1969.
3. Kryter, K.D., EFFECTS OF NOISE ON MAN, Chapter 13, Academic Press, New York: 1970.
4. Guignard, J.C. and King, P.F., Aeromedical Aspects of Vibration and Noise, A. G. A. R. D.ograph #151, NATO Advisory Group for Aerospace Research and Development, Neuilly sur Seine, France: 1972.
5. Gulian, E., Psychological Consequences of Exposure to Noise, Facts and Explanations, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia: 1973.
6. Sanders, A. F., The Influence of Noise on Two Discrimination Tasks, *Ergonomics* 4, 235, 1961.
7. Carpenter, A., Effects of Noise on Performance and Productivity, in CONTROL OF NOISE, Symposium No. 12, 297-310, H. M. Stationary Office: London, 1962.
8. Fornwalt, N.E., Investigation into the Effect of Intermittent Noise of Constant Periodicity vs. Random Periodicity on the Performance of an Industrial Task, Master's thesis, Dept. Industrial Engineering, Texas Tech. Coll., 1965.
9. Boggs, D. H. and Simon, J. R., Differential Effect of Noise on Tasks of Varying Complexity, *J. Applied Psychol.*, 52, 148-153, 1968.
10. Hebb, D. O., Drives and the C. N. S., *Psychological Review*, 62, 243-254, 1955.
11. Wakely, H. C., Noise and Human Behavior, in the *Proceedings of the Symposium On Environmental Noise - Its Human Economic Effects*, 27-34, Chicago Hearing Society, Chicago, 1970.
12. Broadbent, D. E., Factors Increasing and Decreasing the Effects of Noise, paper presented at the International Congress On Noise As A Public Health Problem, Dubrovnik, Yugoslavia, 1973.
13. Broadbent, D. E., PERCEPTION AND COMMUNICATION, Pergmon Press: London, 1958.
14. Wilkinson, R. T., Interaction of Noise with Knowledge of Results and Sleep Deprivation, *J. Experimental Psychology*, 66, 332-337, 1963.

15. Wilkinson, R. T., Some Factors Influencing the Effect of Environmental Stressors Upon Performance, *Psychol. Bull.*, 72, 260-272, 1969.
16. Jerison, H. J., and Wing, S., *Effects of Noise and Fatigue on a Complex Vigilance Task*, WADC Technical Report 57-14, Wright Air Development Center, Wright Patterson AFB, Ohio, 1957.
17. Hartley, L. R., Similar and Opposing Effects of Noise on Performance, paper presented at the International Congress On Noise As A Public Health Problem, Dubrovnik, Yugoslavia, 1973.
18. Glass, D. C. and Singer, J. E., Behavioral Effects and Aftereffects of Noise, paper presented at the International Congress On Noise As A Public Health Problem, Dubrovnik, Yugoslavia, 1973.
19. Broadbent, D. E. and Little, E.A.J., Effects of Noise Reduction in a Work Situation, *Occupational Psychology*, 34, 133, 1960.
20. McGrath, J. J., Irrelevant Stimulation and Vigilance Performance, in VIGILANCE A SYMPOSIUM, Bucknir, D. N. and McGrath, J. J. (eds), McGraw-Hill, New York: 1963.
21. Woodhead, M. M., The Effect of Bursts of Noise on an Arithmetic Task, *American Psychology*, 77, 627-633, 1964.
22. Hockey, G. R. J., Effects of Noise on Human Efficiency and Some Individual Differences, *J. Sound and Vibration*, 20, 299-304, 1972.
23. Broadbent, D. E., Noise, Paced Performance and Vigilance Tasks, *Brit. J. Psychol.*, 44, 295-303, 1953.
24. Broadbent, D. E., Effects of Noises of High and Low Frequency on Behaviour, *Ergonomics*, 1, 21-29, 1957.
25. Jerison, H. J., Paced Performances on a Complex Counting Task under Noise and Fatigue Conditions, *Amer. Psychol.* 9, 399-400, 1954.
26. Lukas, J. S., Peeler, D. J. and Kryter, K. D., Effects of Sonic Booms and Subsonic Jet Flyover Noise on Skeletal Muscle Tension and a Paced Tracing Task, National Aeronautics and Space Administration, Contractor Report NASA, CR-1522, Washington, D. C., 1970.
27. Woodhead, M. M., The Effects of Bursts of Loud Noise on a Continuous Visual Task, *Brit. J. Industrial Med.*, 15, 120-125, 1958.

28. Berglund, K. Rylander, R. and Sorensen, S., Experiments on the Effects of Sonic-Boom Exposure on Humans, *J Acoustical Soc. Amer.*, 51, 790-798, 1972.
29. Harris, C. S., The Effects of High Intensity Noise on Human Performance, Technical Report AMRL-TR-67-119, USAF Aerospace Medical Research Laboratories, Wright-Patterson AFB, Ohio, 1968.
30. Cohen, A., Industrial Noise and Medical, Absence, and Accident Record Data on Exposed Workers, paper presented at the International Congress On Noise As A Public Health Problem, Dubrovnik, Yugoslavia, 1973.

Section 9

INTERACTION OF NOISE AND OTHER CONDITIONS OR INFLUENCES

The preceding chapters have dealt primarily with noise as a single agent as it effects hearing or other physiological or psychological functions. They have also considered mainly the effects of noise on groups or given percentages of the population in what might be considered average conditions. Real life, however, is much more complex than the laboratory, and individuals can be vastly different from the norm. Predictions based on the assumption of normal conditions could miss the mark widely when applied to an individual case or to a group of people with unusual characteristics in common. This chapter will briefly discuss the interactive effects of noise with other agents and conditions that often characterize real life situations.

MEASUREMENT OF EFFECTS

Determination of how other agents or conditions interact with noise in producing a given effect requires three separate experiments, in which is measured:

1. The magnitude (N) of the effect produced by the noise alone.
2. The magnitude (A) of the effect produced by the other agent alone.
3. The magnitude (J) of the effect produced by the joint action of the agent plus the noise.

The specific types of interaction that can occur from a comparison of these three results include the following:

1. Indifferent: the joint effect (J) does not differ significantly from the single effect of either noise or another agent (N or A) whichever is the greater.

$$J \cong N \text{ or } J \cong A$$

2. Additive: the joint effect (J) is approximately equal to the sum of the effect of noise (N) and the effect of the other agent (A).

$$J \cong N + A$$

3. Synergistic: the joint effect (J) is significantly greater than the sum of the other effects (N+A).

$$J > N + A$$

4. Ameliorative: the joint effect (J) is significantly smaller than the larger effect of either noise alone (N) or the other agent alone (A).

$$J < N \text{ or } J < A$$

An enormous number of possible physical and chemical stressors, vitamin or mineral deficiencies, and illnesses exist, all of which could conceivably have some degree of influence—additive, synergistic or ameliorative—on the effects of noise. Furthermore, it is possible that a given agent might have an additive action on one particular effect of noise, a synergistic action on another, and be indifferent as far as a third was concerned. Unfortunately, research in interactive effects has been very sparse. Therefore a brief summary of relevant material is all that can be accomplished at this time.

CHEMICAL AGENTS

Ototoxic Drugs

It is reasonable to expect either an additive or synergistic action from an agent that acts directly on the same physiological elements as noise. For example, agents that are known to be damaging to the hearing mechanism (ototoxic) can be assumed to produce at least an additive effect when combined with noise exposure. Ototoxic drugs—salicylates and quinine, certain diuretics, and aminoglycosidic antibiotics—are known to produce cochlear cell damage and consequent high-frequency hearing loss similar to that produced by noise. There is evidence that a synergistic effect does occur, at least in experimental animals. Quante *et al.*,¹ for example, compared cochlear damage

1. From 90-, 100-, and 110-dB pink noise exposure (see Glossary).
2. From 8 days of kanamycin therapy.
3. From their combination.

Neither the exposure at 90 dB nor the kanamycin therapy produced noticeable changes in the cochlea when administered separately, but animals given the combination showed extensive damage to the outer hair cells. A similar synergistic effect of kanamycin and noise was also shown by Dayal *et al.*² Both studies confirm a similar study by Darrouzet and Sobrinho.³ A similar result was reported by Jauhiainen *et al.*⁴ for neomycin. Sato⁵ has reported previously a synergistic action of noise and quinine, salicylic acid or dihydromycin. This literature has recently been reviewed in greater detail by Falk⁶ and by Haider.⁷

To date there is no definitive data on the interactive effects of ototoxic drugs and noise on humans. There are instances in which a person, during or shortly after a period of medication, definitely suffered a hearing loss when exposed to noise.⁸ It is possible that the noise exposure alone may have been severe enough to produce the same loss in the unmedicated person.

However, it does seem reasonable to advise persons being treated with ototoxic drugs to be particularly careful about noise exposures.

Industrial Chemicals

In an extensive review of industrial hearing loss, Lehnhardt⁹ has summarized the action of various industrial chemicals. Because hearing losses develop in noisy industrial situations in which such substances as carbon disulphide, nitrobenzol, carbon monoxide, trichlorethylene, lead, mercury, arsenic compounds and others are found, there is a possibility that such agents may act additively or synergistically with the noise. Not only hearing damage but also other effects such as cardiovascular problems may be produced.¹⁰ However, as Lehnhardt,⁹ and later, Haider⁷ have pointed out, there still exists no conclusive evidence that the hearing losses in these situations are any greater than would be predicted on the basis of noise exposure alone. It is extremely difficult to match different groups of workers in all respects except the agent in question. In short, then, evidence that exposure to industrial chemicals aggravates hearing losses or non-auditory effects of noise is as yet uncertain.

Vibration

Noise and vibration often occur together, particularly in connection with chain saws, pneumatic hammers and drills. In this case, the possibility of a reciprocally synergistic effect exists. Not only might vibration accentuate the hearing loss produced by the noise, but also the noise could hasten the development of peripheral circulatory problems such as Raynaud's syndrome by inducing vasoconstriction. This condition is one in which the fingers lose their sensitivity, and which is common among operators of pneumatic hammers and drills. The possibility of such an interaction was considered as long as 40 years ago.¹¹

As stated previously, successfully matching groups of workers who differ only in their exposure to one agent is difficult. The most recent attempt to study the interaction of noise and vibration is recounted by Pinter.¹² Large numbers of tractor drivers and chain saw operators exposed to both noise and vibration in the forestry industry were matched, in terms of total estimated cumulative noise exposure, with an equally large number of workers in a furniture industry and a textile mill, respectively. When audiometric results were adjusted for age, the noise plus vibration-exposed populations showed more noise-induced hearing losses than those exposed only to noise. Pinter concludes that vibration enhances the effect of noise on hearing.

Cohen¹³ has pointed out the advantage of measuring the combined effects of noise and other agents using ear protective devices with otherwise equally matched groups. This way, there can be a fairly predictable noise reduction in one group. Although this method has not been used extensively to date, it would seem to be quite helpful in providing future information on the interactive effects of noise and vibration, as well as other agents.

As for effects on other physiological parameters, vibration is usually much more potent than noise. Consequently, most recent studies measure the effects of vibration first alone, and then in combination with noise. A recent study using this technique¹⁴ has yielded negative results—i.e., the addition of noise to vibration (and incidentally to heat stress) produced no significant difference on various performance tasks and physiological measures.

HEALTH CONDITIONS

Mineral and Vitamin Deficiencies

Many people in the world probably suffer from a chronic deficiency in certain minerals or vitamins because of improper diets. Little research has been done, however, on the effect of such deficiencies on susceptibility to noise. Although there is a wealth of literature on the effects of various vitamins and minerals on TTS,⁹ nearly all such experiments involved massive doses of the substance in question, given to presumably otherwise-normal animals. There is a possibility that occasional ameliorative results may in some cases be attributed to an unrecognized deficiency of the substance in the control group.

Research with vitamin A provides an example. Ruedi¹⁵ found that injections of vitamin A produced a decrease in temporary threshold shift. However, a controlled doubleblind study using university students revealed no effect on TTS attributable to the vitamin A, a result later confirmed by Dieroff¹⁷ for noise-induced permanent threshold shifts (NIPTS). A possible explanation of Ruedi's results is that an excess of vitamin A may, in reality, produce no change in susceptibility, whereas a deficiency in vitamin A may actually increase susceptibility to TTS.

Similarly, indication of a slight ameliorative action on TTS for such substances as nicotinic acid, vitamin B₁, hydrochloricpapaverin, nylindrin, thioctic acid and chlorpromazine has recently been reported by Nakamura;¹⁸ for adenosine triphosphate by Faltynek and Vesely;¹⁹ for ephedrine by Stange and Beickert;²⁰ for Hydergine by Plester;²¹ and for destran by Kellerhals *et al.*²² However, considerable effort must still be expended before any of these drugs can be proven generally beneficial.

Illnesses

Whether or not illness affects an individual's susceptibility to various effects of noise is another instance of a reasonable hypothesis with as yet little empirical confirmation. Of course, any condition that increases the amount of energy reaching the cochlea, such as Bell's Palsy, which includes among its symptoms a paralysis of the stapedius muscle, should result in larger TTS's and NIPTS's, and the general consensus is that it does.^{23,24}

The reverse is also true, at least to a limited extent. Dieroff²⁵ showed that in persons with unilateral otosclerosis, which acts much like an earplug in reducing the flow of airborne energy to the cochlea, the “protected” ear has significantly less sensorineural loss. Dohi²⁶ also showed that a chronic perforation of the eardrum reduced the noise-induced hearing losses suffered by industrial workers.

On the other hand, the possibility exists that middle ear diseases which invade the cochlea might cause sufficient changes in the cochlear chemistry and blood supply to increase susceptibility to noise-induced hearing loss. This possibility awaits further exploration. It may, however, account for the fact that when audiometric results of workers are categorized only very broadly, so that all types of “chronic middle ear” problems are thrown into a single group, protection of the affected ear is not always demonstrated.²⁷

Despite the largely inconclusive outcome of this review of interactive effects, it still appears reasonable that both synergistic and ameliorative influences by other agents on the effects of noise will eventually be identified and quantified. Properly planned and executed experiments on the interaction of noise with other stressors is greatly needed if defensible criteria for noise exposure in the presence of such conditions are to be proposed.

SUMMARY—INTERACTION OF NOISE AND OTHER CONDITIONS OR INFLUENCES

Determination of how various agents or conditions interact with noise in producing a given effect requires three separate experiments measuring the effect produced by the noise alone, the effect produced by the other agent alone, and the effect produced by the joint action of the agent and the noise. These results indicate whether the joint effect is indifferent, additive, synergistic, or ameliorative.

Chemical agents may have a joint effect with noise. Ototoxic drugs that are known to be damaging to the hearing mechanism can be assumed to produce at least an additive effect on hearing when combined with noise exposure. There are instances in which individuals using medication temporarily suffer a hearing loss when exposed to noise, but there is no definitive data on the interactive effects of ototoxic drugs and noise on humans. Evidence linking exposure to noise plus industrial chemicals with hearing loss is also inconclusive.

The possibility of a reciprocally synergistic effect exists when noise and vibration occur together. Vibration is usually more potent than noise in effecting physiological parameters. There appears to be consensus that vibration increases the effect of noise on hearing.

Health conditions may interact with noise to produce a hearing loss. Mineral and vitamin deficiencies are one example but little research has been done on the effect of such deficiencies on susceptibility to noise. Another reasonable hypothesis is that illness increases an individual's susceptibility to the adverse effects of noise. However, as with the other hypotheses, conclusive evidence is lacking.

REFERENCES

1. Quante, M., Stupp, H. and Brun, J.P., Ototoxikosen Unter Larmbelastung, *Archiv. fur Klinische und Experimentelle Ohren-, Nasen-, und Kehlkopfheilkunde*, 196, 233, 1970.
2. Dayal, V.S., Kokshanian, A. and Mitchell, D.P., Combined Effects of Noise and Kanamycin, *Ann. Otol.*, 80, 897-902, 1971.
3. Darrouzet, J. and Sobrinho, E.D.L., Inner Ear, Kanamycin, and Acoustic Trauma, Experimental Study, *Rev. Bras. Cirurg.*, 46, 120, 1963.
4. Jauhiainen, T., A. Kohonen and M. Jauhiainen. Combined Effect of Noise and Neomycin on the Cochlea. *Acta Otolaryngol*, 73, 387-390, 1972.
5. Sato, T.A. Study on Experimental Acoustic Trauma in Animals Treated with Specific Poisons for Auditory Organ. *J. Oto-rhino-laryng. Soc. Jap.* 60, Abstr. 31-32, 1957, ref. ZHNO 60, 231, 1958.
6. Falk, S.A. Combined Effects of Noise and Ototoxic Drugs, *Environmental Health Perspectives, Experimental Issue Number Two*, 5-22, Oct. 1972.
7. Haider, M., Influences of Chemical Agents on Hearing Loss, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
8. Darrouzet, J., Essais de Protection de l'Organe de Corti contre l' Ototoxicite Antibiotiques, Etude Experimentelle, *Acta Otolaryng.*, 63, 49-64, 1967.
9. Lehnhardt, E., Die Berufsschaden des Ohres, *Arch. f. Ohr.- Nas. u. Kehlk.- Heilk.*, 185, 11-242, 1965.
10. Zenk, H., Die Begutachtung Cochlearer Schaden Infolge Beruflicher Intoxikationen und Infektionen. *Arbeitsmed., Socialmed., Arbeitshyg.* 2, 358-362, 1967.
11. Temkin, Jakob. Die Schädigung des Ohres durch Lärm und Erschütterung. *Msschr. Ohrenheilk. u. Laryngo-Rhinologie* 67, 257-299, 450-479, 527-553, 705-736, 823-834, 1933.
12. Pinter, I. Hearing of Forest Workers and Tractor Operators, Interaction of Noise with Vibration, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
13. Cohen, A., Industrial Noise and Medical, Absence and Accident Record Data on Exposed Workers, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
14. Grether, W.F., Harris C.S., Ohlbaum, M., Sampson, P.A. and Guignard, J.C., Further Study of Combined Heat, Noise and Vibration Stress, *J. Aerospace Med.* 43, 641-645, 1972.

15. Ruedi, L., Wirkungen des Vitamin A im Menschlichen und tierischen Gehörgang, *Schweiz. Med. Wschr.* 84, 1411-1414, 1954.
16. Ward, W.D. and Glorig, A., The Relation between Vitamin A and Temporary Threshold Shift, *Acta Otolaryng.* 52, 72-78, 1960.
17. Dieroff, H.G., Der Einfluss des Vitamins A auf die Lärmschwerhörigkeit, *HNO (Berlin)*, 10, 323-325, 1962.
18. Nakamura, S., Some of the Basic Problems in Noise Trauma, *Jap. J. Otol.*, 67, 1669-1684, 1964.
19. Faltýnek, L., and Veselý, C., Zur Restitution der Mikrophonpotentiale des Meerschweinchens nach kurzfristiger Lärmbelastung, *Arch. Ohr. Nas. Kehlkopfheilk.*, 184, 109-114, 1964.
20. Stange, G., and Beickert, P., Adaptationverhalten des cortischen Organs nach Ephedrin und Beschallung, *Arch. Ohr. Nas. Kehlkopfheilk.*, 184, 483-495, 1965.
21. Plester, D., Der Einfluss vegetativ wirksamer Pharmaka auf die Adaptation bzw. Hörermüdung, *Archiv. Ohren-Nasen-Kehlkopfheilk.*, 162, 463-487, 1953.
22. Kellerhals, B., Hippert, F. and Pfaltz, C.R., Treatment of Acute Acoustic Trauma with Low Molecular Weight Dextran, *Pract. ORL*, 33, 260-264, 1971.
23. Ward, W.D. Studies on the Aural Reflex, II, Reduction of Temporary Threshold Shift from Intermittent Noise by Reflex Activity; Implications for Damage-Risk Criteria, *J. Acoust. Soc. Am.*, 34, 234-241, 1962.
24. Mills, J.H. and Lilly, D.J. Temporary Threshold Shifts Produced by Pure Tones and by Noise in the Absence of an Acoustic Reflex, *J. Acoust. Soc. Am.* 50, 1556-1558, 1971.
25. Dieroff, H.G., Die Schalleitungsschwerhörigkeit als Lärmschutz, *Z. Laryngol. Rhinol. Otol.*, 43, 690-698, 1964.
26. Dohi, K. Influence of Impaired Tympanic Membrane on Occupational Deafness, *J. Otorhinolaryng. Soc. Jap.* 56: 39, 1953, ref. *ZNHO*, 5, 319, 1954-1955.
27. Mounier-Kuhn, P., Gaillard, J. Martin, H. and Bonnefoy, J., The Influence of the Earlier Condition of the Ear on the Development of Deafness due to Noise Trauma (French), *Acta Oto-Rhino-Laryng. Belg.* 14/2, 176-185, 1960.

Section 10

EFFECTS OF INFRASOUND AND ULTRASOUND

The audio frequency range is generally considered to be 20 to 20,000 Hz. Frequencies below 16 Hz are referred to as infrasonic frequencies. Frequencies above 20,000 Hz are referred to as ultrasonic frequencies.

INFRASOUND

Infrasound occurs in nature at relatively low intensities. Sources of natural infrasonic frequencies are:

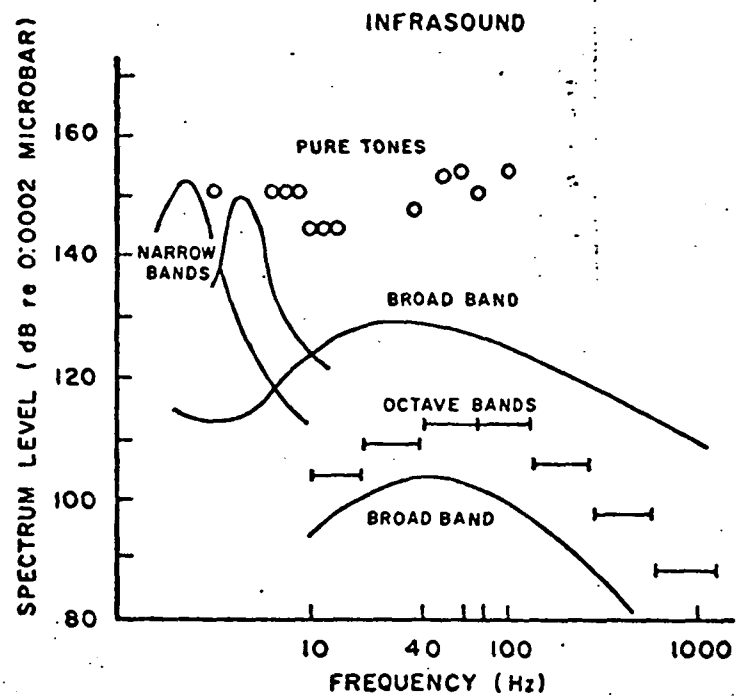
- Earthquakes.
- Volcanic eruptions.^{1,2}
- Winds.
- Air turbulence.
- Thunder.
- Large waterfalls.
- Impact of waves on beaches.³

There are also manmade sources of infrasonic sound such as:

- Air heating and air conditioning systems.
- All transportation systems including jet aircraft.
- High powered propulsion systems utilized in space flights.^{4,5,6}

Man-made infrasound occurs at higher intensity levels than those found in nature. It is therefore conceivable that with the increase in man-made sources, there may exist potential danger to man's health. Stephens and Bryan have reported complaints of people about infrasound, including disorientation, nausea and general feelings of discomfort.⁷ In short, responses generally resemble those seen during whole-body vibration, and are mostly of a non-specific nature, resembling reactions to mild stress or alarm.^{8,9}

Data obtained in comprehensive experiments by Mohr *et al.*, reveal that exposures to high intensity infrasonics (100 db-160 db) for short duration (two minutes) have adverse effects on man.⁸ Results of these studies are summarized in Figure 10-1.



TOLERANCE DATA

EXPOSURE	OBSERVED BEHAVIOR
0 to 50 Hz Up to 145 dB	Chest Wall Vibration, Gag Sensations, Respiratory Rhythm Changes, Post-Exposure Fatigue; Voluntary Tolerance Not Exceeded
50 to 100 Hz Up to 154 dB	Headache, Choking, Coughing, Visual Blurring and Fatigue; Voluntary Tolerance Limit Reached
Discrete Frequencies	Tolerance Limit Symptoms
100 Hz at 153 dB	Mild Nausea, Giddiness, Sub Costal Discomfort, Cutaneous Flushing
60 Hz at 154 dB 73 Hz at 150 dB	Coughing, Severe Substernal Pressure, Choking Respiration, Salivation, Pain On Swallowing, Giddiness

REPRESENTATIVE LOW FREQUENCY AND
INFRASONIC TEST ENVIRONMENTS

MOHR et al
JAMA, 1965

Figure 10-1

Mohr *et al*'s data have been confirmed by Nixon.¹⁰ Whether or not symptoms similar to those described in Figure 10-1 would occur for prolonged exposure to low intensities of infrasound still remains an open question. There is, however, a report by Green and Dunn which shows that there exists a correlation (0.5) between infrasound exposure and disturbance of certain activities, such as increase in absenteeism in school children and unskilled workers and a higher rate of automobile accidents during periods of higher infrasonic exposure.¹¹

A variety of bizarre sensations in the ear have also been reported during exposure to airborne infrasonic waves. These include fluttering or pulsating sensations.¹²

There is some evidence that intense infrasound (120db Sound Pressure Level or above) can stimulate the vestibular system, as can low-frequency vibration, leading to disequilibrium if the stimulation is intense enough; nevertheless, there is no evidence that the hearing organ may be affected by exposures to infrasonic waves encountered in real-life situations.^{8,9} However, Guignard and Coles (1965) have demonstrated that a very high-frequency mechanical vibration may produce a small TTS involving the lower audiometric frequencies and from this it may be inferred that airborne infrasound could possibly also have an effect on hearing.¹³

Various experiments have attempted to shed light on this problem.⁹ Results are presented in Table 10-1. The data contained in Table 10-1 reveals that:

1. Only small, if any, TTS can be observed following exposures to moderate and intense infrasonics.
2. Recovery to pre-exposure hearing levels is rapid when TTS do occur.

The data available suggests that infrasonics do not pose a serious problem to the hearing mechanism when intensities are below 130 db SPL (which is generally the case); however, where high intensities are present (above 130 dB SPL) there may exist a serious hazard.

ULTRASOUND

It will be recalled that ultrasonic frequencies are those above 20,000 Hz. Ultrasonics are produced by a variety of equipment and in industry, such apparatus as:

- High speed drills.
- Cleaning devices.
- Dicing equipment.
- Emulsification and mixing devices.

Research Problems

Ultrasonic waves became recognized as a potential health problem with the advent of jet engines when a number of persons working in the vicinity of jet engines reported symptoms of

TABLE 10-1

INVESTIGATOR	EXPOSURE	HEARING RESPONSE	RECOVERY
Tonndorf (17)	Submarine Diesel Room 10 Hz-20 Hz, No Level Given	Depression of Upper Limits of Hearing as Measured by Number of Seconds a Tuning Fork was Heard - No Conversion to MAP	Recovery in Few Hours Outside of Diesel Room
Mohr, et al (9)	Discrete Tones; Narrow Band Noise in 10 Hz-20 Hz Region. 150 - 154 dB Exposures of About 2 Minutes	No Change in Hearing Sensitivity Reported by Subjects; No TTS Measured About One Hours Post Exposure	
Jerger, et al (6)	Successive 3 Minute Whole Body Exposures, 7-12 Hz; 119 - 144 dB	TTS in 3000 - 6000 Hz Range For 11 of 19 Subjects (TTS of 10 dB - 22 dB)	Recovery Within Hours
Nixon (11)	Pistonphone Coupled to Ear via Earmuff. 18 Hz at 135 dB. Series of 6, 5 Minute Exposures Rapid in Succession	Average TTS of 0 - 15 dB After 30 Minute Exposures	Recovery Within 30 Minutes
Nixon (11)	Pistonphone Coupled to Ear via Earmuff. 14 Hz at 140 dB. Six Individual Exposures of 5, 10, 15, 20, 25 and 30 Minutes	Three Experienced Subjects No TTS in One; Slight TTS in One; 20 - 25 dB TTS in One	Recovery Within 30 Minutes

According to Nixon and Johnson, 1973

TABLE 10-1 (Continued)

INVESTIGATOR	EXPOSURE	HEARING RESPONSE	RECOVERY
Johnson (7)	<p>Ear Only: Pressure Chamber Coupled to Ear via Tuned Hose and Muff</p> <p>171 dB (1-10 Hz) 26 sec, 1s 168 dB (7 Hz) 1 min, 1s 155 dB (7 Hz) 5 min, 2s 140 dB (4,7,12 Hz) 30 min, 1s 140 dB (4,7,12 Hz) 5 min, 8s 135 dB (.6, 1.6, 2.9 Hz) 5 min, 12s 126 dB (.6, 1.6, 2.9 Hz) 16 min, 11s</p> <p>Whole Body: All Exposures, 2s: 8 min at 8 Hz at SPL's of 120, 126, 132, 138 8 min at 1,2,4,6,8,10 Hz at 144 dB 8 min at 12,16,20 Hz at 135 dB to 142 dB</p>	<p>No TTS No TTS No TTS 14-17 dB TTS 8 dB TTS for 1 Subject No TTS No TTS</p> <p>No TTS No TTS No TTS</p>	<p>Recovery Within 30 Minutes Recovery Within 30 Minutes</p>

excessive fatigue, nausea, headache and even vomiting.^{14,15} These responses resemble those found during stress. The problem, however, is difficult to study because of two factors:

1. Ultrasonic waves are highly absorbed by air and, therefore, are of significance only near a source.
2. Airborne ultrasonics from ordinary sources are often accompanied by broadband noise and by sub-harmonics, both of which fall into the audible range.¹⁶

For the reasons just stated, it was thought that the effects reported by various personnel working near jet engines were due to stimulation of the vestibular system by intense acoustic stimulation, and the matter did not receive much attention.^{15,17} However, consideration of the subject was revived in the mid-50's by Crawford.¹⁸

Physiological Effects

In man, there have been reports of blood sugar level decrease following exposure to ultrasonics¹⁹; however, there are also reports of increased blood sugar level.²⁰ There are also reports of electrolyte balance changes in the tissues of the nervous system.²¹ A major problem with these studies is that neither the sound levels nor the frequencies utilized in these experiments are mentioned.

In a study by Batolska, it is cautioned that some of the effects that have been attributed to exposure to ultrasonic waves are similar to those produced by potential toxic agents that often are found in working places.²²

In work by Grigoreva, no significant physiological changes were found in subjects exposed to sound ranging between 110 dB and 115 dB SPL for 1 hour at 20,000 Hz.^{23,24} Parrack, on the other hand, has shown a mild warming of the surface of the body following exposure to 159 dB, and a loss of equilibrium and dizziness has been shown following exposures to a 20-KHz tone at levels of 160 to 165 dB.²⁵

A number of studies designed to assess the effects of ultrasonics on the hearing mechanism are reported in a review paper by Acton,¹⁶ as follows:

"An investigation to determine if the noise from industrial ultrasonic devices caused auditory effects was described by Acton and Carson (1967). The hearing threshold levels of 16 subjects (31 ears) were measured in the frequency range 2 to 12 KHz before and after exposure to the noise over a working day. No significant temporary threshold shifts were detected (Figure 2). On the assumption that if a noise exposure is not severe enough to cause a temporary threshold shift, then it cannot produce permanent damage, it was concluded that hearing damage due to exposure to the noise from industrial ultrasonic devices is unlikely. A parallel retrospective investigation by KNIGHT (1968) on a group of 18 young normal subjects using ultrasonic devices showed

a median hearing level within 5 dB of that of a matched control group of hospital staff except, at 4 KHz where the departure was 7 dB. It was concluded that it would have been difficult to attribute this exposure solely to ultrasonic radiation. In addition, no abnormal vestibular function test (caloric test) results were found.

“Some temporary threshold shifts have been reported as a result of exposures to ultrasound under laboratory conditions.....(Parrack, 1966, Dobroserdov, 1967, Smith 1967).

“The exposures used by Dobroserdov were at high audible frequencies, and those by Smith contained high audible frequency noise. The results due to Parrack are interesting in that he exposed subjects to discrete frequencies mainly in the ultrasonic region, and measured temporary threshold shifts at sub-harmonics of one half of the fundamental and occasionally at lower sub-harmonic frequencies as a result of 5 minute exposures to discrete frequencies in the range 17 to 37 KHz at levels of 148 to 154 dB. Sub-harmonic distortion products have been reported in the cochlear-microphic potentials of guinea pigs (Dallos and Linnel, 1966a) and have also been monitored in the sound field in front of the eardrum using a probe-tube microphone (Dallos and Linnel, 1966b). They were believed to result from non-linear amplitude distortion of the ear drum, and they appeared at a magnitude of the same order as that of the fundamental. This observation may help to explain Parrack’s findings.”

The discussion above reveals that exposure to high levels of ultrasound (above 105 dB SPL) may have some effects on man; however, it is important to recognize that a hazard also arises from exposure to the high levels of components in the audible range that often accompany ultrasonic waves. At levels below 105 dB SPL there does not appear to be significant danger.

SUMMARY—INFRASOUND AND ULTRASOUND

Frequencies below 16 Hz are referred to as infrasonic frequencies. Sources of infrasonic frequencies include earthquakes, winds, thunder, and jet aircraft. Man-made infrasound occurs at higher intensity levels than those found in nature. Complaints associated with infrasound resemble mild stress reactions and bizarre auditory sensations, such as pulsating and fluttering. It does not appear, however, that exposure to infrasound, at intensities below 130 dB SPL, present a serious health hazard.

Ultrasonic frequencies are those above 20,000 Hz. They are produced by a variety of industrial equipment and jet engines, the effects of exposure to high intensity ultrasound (above 105 dB SPL) also resemble those observed during stress. However, there are experimental difficulties in assessing the effects of ultrasound. Since:

1. Ultrasonic waves are highly absorbed by air.
2. Ultrasonic waves are often accompanied by broadband noise and by sub-harmonics.

At levels below 105 dB SPL there have been no observed adverse effects.

REFERENCES

1. Cook, R.K. Radiation of Sound by Earthquakes, 5th International Congress on Acoustics, Liege, Sept. 1965.
2. Hinde, B.J. and Graunt, D.I., Microseisms, *Contemporary Physics*, 8, 267, 1967.
3. Fehr, V., Measurements of Infrasound from Artificial and Natural Sources of Low Frequency Sound, Proceedings of Fall Meeting of British Acoustical Society, 71-105, Nov. 1971.
4. Stephens, R.W.B., Sources of Low Frequency Sound, Proceedings of Fall Meeting of British Acoustical Society, 71-105, Nov. 1971.
5. Stephens, R.W.B., Very Low Frequency Vibrations and Their Mechanical and Biological Effects. Seventh International Congress on Acoustics, 26-G-1, Budapest 1971.
6. Tempest, W. Low Frequency Noise on Road Vehicles. Proceedings of Fall Meeting of British Acoustical Society, 71-106, Nov. 1971.
7. Stephens, R.W.B. and Bryan, M.E. Annoyance Effects Due to Low Frequency Sound, Proceedings of Fall Meeting of British Acoustical Society, 71-109, Nov. 1971.
8. Mohr, G.C., Cole, J.N., Guild, E.C. and Von Gierke, M.E., Effects of Low Frequency and Infrasonic Noise on Man, *Aerospace Medicine*, 36, 817-824, 1965.
9. Nixon, W. and Johnson, D.L. Infrasound and Hearing, paper presented at the International Congress on Noise as a Public Health Problem, Dubrovnik, Yugoslavia, 1973.
10. Nixon, C.W., Some Effects of Noise on Man. Proceedings of 1971 Intersociety Energy Conversion Engineering Conference, Boston, Mass., August 1971.
11. Green, J.E. and Dunn, F. Correlation of Naturally Occurring Infrasonics and Selected Human Behavior, *JASA*, 44, 1456, 1968.
12. Yeowart, N.S., Bryan, M.E., and Tempest, W., The Monaural M.A.P. Threshold of Hearing at Frequencies from 1.5 to 100 c/s. *J. Sound and Vibration*, 6, 335-342, 1967.
13. Guignard, J.C. and Coles, R.R.A., Fifth International Congress on Acoustics, Liege, Sept. 1965.
14. Davis, H., Biological and Psychological Effects of Ultrasonics, *JASA*, 20, 605, 1948.
15. Parrack, H., Ultrasound and Industrial Medicine, *Ind. Med. and Surgery*, 21, 156, 1952.
16. Acton, W.I., The Effects of Airborne Ultrasound and Near-Ultrasound. Paper presented at the International Congress on Noise as a Health Problem, Dubrovnik, Yugoslavia, 1973.
17. Davis, H., Parrack, H.D., and Eldredge, D.H., Hazards of Intense Sound and Ultrasound, *Annals of Otolaryngology*, 58, 732, 1949.

18. Crawford, A.E., *Ultrasonic Engineering*, Butterworth, London: 1955.
19. Asbel, F.F., The Effects of Ultrasound and High Frequency Noise on Blood Sugar Level, *Occupational Safety and Health Abstracts*, 4, 104, 1966.
20. Byalko, N. Certain Biochemical Abnormalities in Workers Exposed to High Frequency Noise, *Excerpta Medica*, 17, 570, 1964.
21. Angelusheff, F.D., Ultrasonics, Resonance and Deafness, *Revue de Laryng, d' Otol., et de Rhinol.*, July 1957.
22. Batolska, A., Occupational Disorders Due to Ultrasound, Work of the Scientific Research Institute of Labor Protection and Occupational Diseases, Sofia, 19, 63-69, 1969.
23. Grigoreva, V.M., Ultrasound and the Question of Occupational Hazards, abstracted in *Ultrasonics*, 4, 214, 1966.
24. Grigoreva, V.M., Effect of Ultrasonic Vibrations on Personnel Working with Ultrasonic Equipment, *Soviet Physics, Acoustics*, 11, 426, 1966.
25. Parrack, H.D., Physiological and Psychological Effects of Noise, Proceedings of Second Annual National Noise Abatement Symposium, Chicago, Ill., 1951.

SECTION 11

EFFECTS OF NOISE ON WILDLIFE AND OTHER ANIMALS

In recent years the effect of noise on wildlife and other animals has become a matter of serious concern for several reasons. As our American civilization proliferates, we find that areas previously considered tranquil and remote are now being exposed to various kinds and amounts of noise. The effects that increased noise levels have on wildlife in these areas are practically unknown. (Much of the following material can be found in *Effects of Noise on Wildlife and Other Animals*, prepared by John Fletcher, et al., EPA NTID 300.5, 1971).

This section will present an overview of the documented and suspected effects of noise on animals. Laboratory animals will be discussed briefly, insofar as their reaction to noise is of interest in assessing the effects on wildlife and farm animals. (Of course the primary reason for studying the effect of noise on laboratory animals has been to throw light on the human reaction). Noise exposures of farm animals will be discussed briefly with respect to possible changes in size, weight, reproductivity, and behavior. Effects of noise on wildlife will be dealt with throughout, although this area is probably the most complex and least documented of the three.

Noise produces the same general categories of effects on animals as it does on humans. For purposes of this document, these categories will be classified as auditory, masking, behavioral, and physiological. The actual effects, although they are somewhat more basic, are in many ways analogous to human life. Reduction of sensitivity in animals may create a particular hardship for those animals that rely on auditory signals for staking out territory, courtship and mating behavior, and locating both prey and predators. Masking of signals can also inhibit these activities in a similar way. Behavioral effects may include panicking and crowding in severe cases, with aversive reactions being more common. Disruption of breeding and nesting habits are occasional consequences of noise exposure, along with possible changes in migratory patterns. Documented physiological changes have been observed almost entirely in laboratory animals. They consist of the general pattern of response to stress including changes in blood pressure and chemistry, hormonal changes and changes in reproductivity.

EFFECT ON HEARING

In assessing the effects of noise on the auditory system of animals, it is important to determine what the particular animal in question can hear. Although the auditory range of most birds and

reptiles lies within the human range,^{2,3} some animals, such as dogs, bats, and rodents, possess hearing sensitivity which we would consider ultrasonic. Sewell⁴ (1970) reported that certain rodents both emit and respond to frequencies up to 40,000 Hz, and even up to 80,000 Hz in special cases. Various procedures have been devised to elicit auditory responses from animals. The Preyer or ear-twitch reflex is a reliable but not very sensitive test of hearing.⁵ Many laboratory animals have been conditioned quite well to respond behaviorally to auditory stimuli. Their cochlear and neural activity in response to sound can be monitored electronically, and also, they can be sacrificed and examined histologically to observe the condition of the auditory mechanism.

Poche, Stockwell and Ades⁶ found that guinea pigs exposed to impulsive noise averaging 153 dB Sound Pressure Level, 1 to 5 seconds apart over a 45 minute period showed histological damage in a narrow band midway along the organ of Corti. Similarly, Majeau-Chargois, Berlin, and Whitehouse⁷ studied the effect on guinea pigs of 1000 simulated sonic booms at approximately 130 dB, at the rate of one boom per second. Although the Preyer reflex did not reveal any changes in hearing sensitivity, histological examination showed considerable loss of sensory cells in the inner ear.

Benitez, Eldredge, and Templer⁸ studied the effects of narrowband noise on chinchillas. They found a temporary threshold shift of 48 dB, with eventual behavioral recovery in response to 48 to 72 hours of an octave-band noise centered at 500 Hz at 95 dB SPL. Similarly, Miller, Rothenberg, and Eldredge⁹ obtained TTSs of 50 dB during 7 days of exposure to a 300-600 Hz octave-band at 100 dB SPL. Although behavioral recovery was nearly complete, histological examination revealed that sensory cells were lost.

In examining the effects of broad-band noise, Miller, Watson, and Covell¹⁰ exposed cats to noise of 115 dB for 15 minutes with a resulting permanent threshold shift of 5.6 dB, and then for 8 hours with a resulting permanent threshold of as much as 40.6 dB. The same exposure broken up into small doses produced considerably less hearing loss.

By exposing guinea pigs to loud music peaking occasionally as high as 122 dB on an irregular schedule, Lipscomb¹¹ found extensive cochlear cell damage. In a similar series of studies, octave-band noise of 110 dB for 8-hour exposure periods was found to create widespread damage throughout the cochlea, regardless of the center-frequency of the noise bands, when guinea pigs were used.¹² This condition was slightly less widespread in the case of chinchillas.¹³

As expected, the extent of noise-induced hearing loss in animals depends upon the intensity, spectrum, and duration of the stimulus and on the pattern of exposure and individual susceptibility. A table of damage-risk contours for various animals would be in order at this point, but to date, this topic has not been as thoroughly explored for animals as it has for humans.

MASKING

Masking of auditory signals can interfere with some animals' communication of necessary information, such as danger, distress, warnings about territorial boundaries, recognition of a mate or of young, etc.

Much of the research on the effects of masking has been to evaluate the effectiveness of commercial units which produce jamming or other aversive signals to repel unwanted animals. Some animals are more resistant to masking than others. Griffin, McCue, and Grinnell¹⁴ showed that bats resist jamming by orienting themselves so that noise and signal are received from different angles. Potash¹⁵ reported that Japanese quail responded to an increase in ambient noise levels from 36 to 63 dBA by increasing the frequency of their separation calls, (i.e., the number of calls in time), thereby improving the signal to noise ratio. However, rabbits, deer, and some species of birds have been repelled by a commercial jamming signal which produces signals of 2,000 and 4,000 Hz which are amplitude and frequency modulated.¹⁶

BEHAVIORAL CHANGES

Behavioral changes are perhaps the most observable effects of noise on wild animals. It seems that many animals learn to differentiate among acoustic stimuli. Deer have been observed grazing close to the runway of a busy heliport,¹⁷ whereas other deer have been noticeably scarce at the first crack of a rifle during the hunting season.¹⁸ Birds have also been seen to react in an adaptive way. Starlings have been repelled by tape-recorded starling distress-calls only to reinfest the area after cessation of the signal.¹⁹ A study by Thompson, Grant, Pearson, and Corner²⁰ showed, by telemetric monitoring of heart rate, that starlings reacted differently to various aversive and neutral stimuli, and habituation to the stimuli occurred at various rates. In order to effectively scare birds, the Committee on the Problem of Noise²¹ reported that a noise level of 85 dB SPL at the bird's ear was required. Since birds seem to adapt quickly, the Committee reported that the signals should be used sporadically throughout the day.

More serious aversive behavior has been observed in some animals. Greaves and Rowe²² found that wild Norway rats and house mice, when exposed to pulsed ultrasound, displayed aversion to the sonic field and did not re-enter the testing ground after exposure. Cutkomp²³ reported that ultrasonic pulses at 65 dB SPL produced aversive behavior in certain species of moths, as well as reduced longevity. Of greater concern are effects reported by Shaw²⁴ who found that adult condors were very sensitive to noise and abandoned their nests when disturbed by blasting, sonic booms, or even traffic noise. As reported by Bell²⁵ and Henkin²⁶ the most harmful effects attributed to sonic booms were mass hatching failures of sooty terns in Florida, where 50 years of breeding success were followed by a 99 percent failure of terns' eggs to hatch in 1969. It is thought that extremely low-altitude supersonic flights over the area may have driven the birds off their nests and damaged the uncovered eggs.

PHYSIOLOGICAL REACTIONS

As stated earlier, the physiological response to noise follows the general pattern of response to stress, which can be an extremely difficult parameter to measure. Undoubtedly, susceptibility to different stressors is variable among animals, as are stressful conditions. Wild animals, of necessity, are more sensitive to a variety of environmental stimuli than most domestic animals²⁷. However, an animal raised under conditions that protect it from stress can become extremely susceptible to disease under even mildly stressful situations.¹ The actual significance of physiological response to stress for an individual animal is not adequately understood.

It must be noted that most of the physiological effects described are the result of relatively brief exposures to very high noise levels. These exposures could be considered acute, and the chances for duplication in real-life situations are fairly slim. Levels cited are sometimes as high as 160 dB, with most in excess of 100 dB, considerably above what we would normally find around airfields, industries, highways or other intrusions of people into the natural habitat of animals. Fletcher et al.¹ point out the difficulty in generalizing from high level, acute exposures to the more realistic low level, chronic ones, as well as the difficulty in generalizing from laboratory animals to wild animals in their natural habitat.

Laboratory experiments have shown that exposure to a 120 Hz tone at 100 dB SPL for intervals of 5 minutes per day for 15 days produced higher adrenal weights and ascorbic acid values and lower blood glutathione levels in experimental rats as opposed to their controls²⁸. Hrubes and Benes²⁹ found that white rats repeatedly subjected to 95 dB noise levels developed increased uremic catecholamines, increased free fatty acid in blood plasma, and increased suprarenal size. Friedman, Byers and Brown³⁰ exposed rats and rabbits to white noise of 102 dB SPL continuously for 3 and 10 weeks, respectively, with a randomly interspersed 200 Hz tone at 114 dB SPL. Although few differences were noted in the rats, the rabbits showed significantly more aortic atherosclerosis and a higher cholesterol content than their controls, along with deposits of fat in the irises of the eyes. The authors concluded that auditory stress can produce changes in the organism's handling of fat.

Although experimental results are not always consistent, auditory stress can also cause changes in reproductive glands and functions. Anthony and Harclerode³¹ reported no significant changes in the sexual behavior of male guinea pigs exposed to 300-48000 Hz band of noise at 139-144 dB SPL for 20 minutes out of each 30 minute period, daily for 12 weeks. (Of course, the animals could have been deafened fairly quickly by such intense exposures, thereby preventing changes which might otherwise have occurred). Experiments by Zondek and Isachar³² found considerably more effect of auditory stress on female rats and rabbits than in the males. Exposure to a stimulus of approximately 100 dB at 4000 Hz for one minute out of every 10, continuously for 9 days, produced enlargement of the ovaries, persistent estrus and follicle hematoma. Exposure to a similar stress caused a significant reduction in both male and female fertility in white rats.

Laboratory studies have also shown that auditory stress can sometimes produce harmful effects in pregnancy. Ishii and Yokobori³³ found that female mice exposed to white noise at the 90, 100, and 110 phon levels for 5 days during pregnancy produced more malformed, still-born, and smaller young than did their controls. More serious effects were found by Ward, Barletta and Kaye,³⁴ who exposed female mice to a 320-580 Hz stimulus at 82-85 dB SPL for 60-75 percent of each hour for 5 hour periods at different stages during pregnancy. Although moderate noise levels were used, 40 percent of the litters were resorbed when exposure occurred during certain periods of pregnancy, and 100 percent of the litters were resorbed when exposure occurred during more critical periods. The authors felt that these effects were due to decreased uterine and placental blood flow, as the result of stress.

Interesting results have been obtained by Anthony, Ackerman, and Lloyd in their study of adreno-cortical activation in rats, mice and guinea pigs. The authors found that these animals could adapt successfully to fairly high levels of noise, but that when audiogenic stress occurred in combination with another stress, such as restriction of food, the animal's life span could be decreased. These findings, along with those which show changes in animals' ability to handle fat, could provide important implications for wildlife, especially during the lean months of late winter.

As mentioned previously, there is little direct information on the physiological response of wildlife to noise. The study of Thompson, Grant, Pearson and Corner²⁰ showed changes in the heart rate of birds by telemetric monitoring, although the long term consequences of this type of stress are still unknown. Studies of fish exposed to noise are not conclusive. A report of the FAO Fisheries³⁶ shows that fish respond to the noise of fishing vessels by diving and by changing direction. The same report states that low frequency noise appears to be more frightening than high frequency noise. Fish kills resulting from underwater explosions are thought to be due to pressure waves rather than acoustic stimulation. A number of studies of the effect of sonic boom on fish egg hatchability failed to show any adverse results.³⁷

FARM ANIMALS

Possible effects of noise on farm animals include changes in:

- Milk production
- Egg hatchability
- Mating behavior
- The animal's size and weight

It appears that some animals are more sensitive to meaningful sound stimuli, such as distress signals.³⁸ However, the majority of studies of the effects of noise on farm mammals have produced negligible results.^{39,40,41,42} Bond⁴³ did find a mild reaction to noise in dairy and beef cattle; however, reactions to low subsonic aircraft noise exceeded the reactions to sonic booms. Furthermore, the same reactions were elicited in response to flying paper, strange persons, or other moving objects.

The reaction of poultry is a slightly different matter. Although noise seems to have little effect on the hatchability of eggs or the quality of chicks hatched,⁴⁴ it does appear to affect the hen's inclination to incubate her eggs. Stadelman⁴⁴ exposed hens to aircraft noise of approximately 120 dB at intervals of 8 dB.

SUMMARY – EFFECTS OF NOISE ON WILDLIFE AND OTHER ANIMALS

Noise produces the same general types of effects on animals as it does on humans, namely: auditory, masking of communication, behavioral, and physiological.

As previously mentioned, the most observable effects of noise on farm and wild animals seem to be behavioral. Clearly, noise of sufficient intensity or noise of aversive character can disrupt normal patterns of animal existence. Exploratory behavior can be curtailed, avoidance behavior can limit access to food and shelter, and breeding habits can be disrupted. Hearing loss and the masking of auditory signals, as mentioned before, can further complicate an animal's efforts to recognize its young, detect and locate prey, and evade predators. Competition for food and space in an "ecological niche" results in complex interrelationships and, hence, a complex balance.

Many laboratory studies have indicated temporary and permanent noise-induced threshold shifts. However, damage-risk criteria for various species have not yet been developed. Masking of auditory signals has been demonstrated by commercial jamming signals, which are amplitude and frequency modulated.

Physiological effects of noise exposure, such as changes in blood pressure and chemistry, hormonal balance, and reproductivity, have been demonstrated in laboratory animals and, to some extent, in farm animals. But these effects are understandably difficult to assess in wildlife. Also, the amount of physiological and behavioral adaptation that occurs in response to noise stimuli is as yet unknown.

Considerable research needs to be accomplished before more definitive criteria can be developed. The basic needs are:

1. More thorough investigations to determine the point at which various species incur hearing loss.
2. Studies to determine the effects on animals of low-level, chronic noise exposures.
3. Comprehensive studies on the effects of noise on animals in their natural habitats. Such variables as the extent of aversive reactions, physiological changes, and predator-prey relationships should be examined.

Until more information exists, judgments of environmental impact must be made on existing information, however incomplete.

REFERENCES

1. Memphis State University, Effects of Noise on Wildlife and Other Animals, U.S. Environmental Protection Agency, Document, NTID 300.5, 1971.
2. Konishi, Masakazu, Comparative Neurophysiological Studies of Hearing and Vocalizations in Songbirds, *Zeitschrift fuer Vergleichende Physiologie*, 67, 363-381, 1970.
3. Manley, Geoffrey, Comparative Studies of Auditory Physiology in Reptiles, *Zeitschrift fuer Vergleichende Physiologie*, 67, 363-381, 1970.
4. Sewell, G.D., Ultrasonic Signals from Rodents, *Ultrasonics*, 8, 26-30, 1970.
5. Memphis State University, Effects of Noise on Wildlife and Other Animals, U.S. Environmental Protection Agency, Document, NTID 300.5, 1971.
6. Poche, L.B., Stockwell, C.W. and Ades, H., Cochlear Hair Cell Damage in Guinea Pigs after Exposure to Impulse Noise, *The Journal of the Acoustical Society of America*, 46, 947-951, 1969.
7. Majeau-Chargois, D.A., Berlin, C.I. and Whitehouse, G.D., Sonic Boom Effects on the Organ of Corti, *The Laryngoscope*, 80, 620-630, 1970.
8. Benitez, L.D., Eldridge, D.H. and Templer, J.W., Electrophysiological Correlates of Behavioral Temporary Threshold Shifts in Chinchilla, Paper presented at the 80th meeting of the Acoustical Society of America, Houston, November 1970.
9. Miller, J.D., Rothenberg, S.J. and Eldredge, D.H., Preliminary Observations on the Effects of Exposure to Noise for Seven Days on the Hearing and Inner Ear of the Chinchilla, *The Journal of the Acoustical Society of America*, 50, 1119-1203, 1971.
10. Miller, J.D., Watson, C.S. and Covell, W.P., Deafening Effects of Noise on the Cat, *Acta Otolaryngologica, Suppl.*, 176, 91, 1963.
11. Lipscomb, D.M., Ear Damage from Exposure to Rock and Roll Music, *Arch. Otol.*, 90, 545-555, 1969.
12. Lipscomb, D.M., Noise Exposure and Its Effects, *Scand., Audiol.*, 1, No. 3, 119-124, September 1972.
13. Lipscomb, D.M., Theoretical Considerations in the Apparent Rise of High Frequency Hearing Loss in Young Persons, Presented to the International Congress of Noise as a Public Health Problem, Dubrovnik, Yugoslavia, May 15, 1973.
14. Griffin, D.R., McCue, J.J.G. and Grinnel, A.D., The Resistance of Bats to Jamming, *Journal of Experimental Zoology*, 152, 229-250, 1963.
15. Potash, L.M., A Signal Detection Problem and Possible Solution in Japanese Quail, *Animal Behavior*, 20, 1972.
16. Crummett, J.G., Acoustic Information Denial as a Means for Vertebrate Pest Control, Paper presented at the 80th meeting of the Acoustical Society of America, Houston, November 1970.

17. Fletcher, J., Agricultural and Recreational Use Noise, Statement at U. S. Environmental Protection Agency Public Hearings on Noise Abatement and Control, Denver, Colorado, V, 45, 1971.
18. Michener, R., Agricultural and Recreational Use Noise, Statement at U.S. Environmental Protection Agency Public Hearings on Noise Abatement and Control, Denver, Colorado, V, 42-46, 1971.
19. Block, B.C., Williamsport, Pennsylvania Tries Starling Control With Distress Calls, *Pest Control*, 34, 24-30, 1966.
20. Thompson, R. D., Grant, C. V., Pearson, E. W. and Corner, G. W., Differential Heart Rate Response of Starlings to Sound Stimuli of Biological Origin, *The Journal of Wildlife Management*, 32, 888-893, 1968.
21. Committee on the Problem of Noise, Final Report, Presented to Parliament July, 1963, London: Her Majesty's Stationery Office, Cmnd. 2056, 19s bd. net.
22. Greaves, J. H. and Rowe, F. P., Responses of Confined Rodent Populations to an Ultrasound Generator, *Journal of Wildlife Management*, 33, 409-417, 1969.
23. Cutkomp, L. K., Effects of Ultrasonic Energy on Storage Insects, Agriculture Department Cooperative State Research Service, Minnesota, 1969.
24. Shaw, E. W., California Condor, Library of Congress Legislative Reference Service, SK351, 70-127, 1950.
25. Bell, W. B., Animal Response to Sonic Boom, Paper presented at the 80th meeting of the Acoustical Society of America, Houston, November 1970.
26. Henkin, H., The Death of Birds, *Environment*, 11, S1, 1969.
27. Goetz, B., Agricultural and Recreational Use Noise, Statement at U. S. Environmental Protection Agency Public Hearings on Noise Abatement and Control, Denver, Colorado, V, 91-96, 1971.
28. Jurtshuk, P., Weltman, A. S. and Sackler, A. M., Biochemical Responses of Rats to Auditory Stress, *Science*, 129, 1424-1425, 1959.
29. Hrubes, V. and Benes, V., The Influence of Repeated Noise Stress on Rats, *Acta Biologica et Medica Germanica*, 15, 592-596, 1965.
30. Friedman, M., Byers, S. O. and Brown, A. E., Plasma Lipid Responses of Rats and Rabbits to an Auditory Stimulus, *American Journal of Physiology*, 212, 1174-1178, 1967.
31. Anthony, A. and Harclerode, J. E., Noise Stress in Laboratory Rodents, II. Effects of Chronic Noise Exposures on Sexual Performance and Reproductive Function of Guinea Pigs, *Journal of the Acoustical Society of America*, 31, 1437-1440, 1959.
32. Zondek, B. and Isachar, T., Effect of Audiogenic Stimulation on Genital Function and Reproduction, *Acta Endocrinologica*, 45, 227-234, 1964.

33. Ishii, H. and Yokobori, K., Experimental Studies on Teratogenic Activity of Noise Stimulation, *Gunma Journal of Medical Sciences*, 9, 153-167, 1960.
34. Ward, C. O., Barletta, M. A. and Kaye, T., Teratogenic Effects of Audiogenic Stress in Albino Mice, *Journal of Pharmaceutical Sciences*, 59, 1661-1662, 1970.
35. Anthony, A., Ackerman, E. and Lloyd, J. A., Noise Stress in Laboratory Rodents, I, Behavioral and Endocrine Response of Mice, Rats, and Guinea Pigs, *Journal of the Acoustical Society of America*, 31, 1430-1437, 1959.
36. F A O Fisheries Report No. 76, Report on a Meeting for Consultations on Underwater Noise, Food and Agriculture Organization of the United Nations, April 1970.
37. Rucker, R. R., Effect of Sonic Boom on Fish, Federal Aviation Administration Systems Research and Development Service, Report No. FAA-RD-73-29, 1973.
38. Bugard, P., Henry, M., Bernard, C. and Labie, C., Aspects Neuro-Endocriniens et Metaboliques de l'Agression Souore, *Revue de Pathologie Gienerale et de Physiologie Clinique*, 60, 1683-1707, 1960.
39. Bond, J., Winchester, C. F., Campbell, L. E. and Webb, J. C., Effects of Loud Sounds on the Physiology and Behavior of Swine, U. S. Department of Agriculture, Agricultural Research Service Technical Bulletin, No. 1280, 1963.
40. Bond, J., Effects of Noise on the Physiological and Behavior of Farm-raised Animals, *PHYSIOLOGICAL EFFECTS OF NOISE*, Welch and Welch, (eds.), New York: Plenum Press, 295-306, 1970.
41. Parker, J. B. and Bayley, N. D., Investigation on Effects of Aircraft Sound on Milk Production of Dairy Cattle, 1957-1958, United States Department of Agriculture, Agriculture Research Service, Animal Husbandry Research Division, 1960.
42. Casady, R. B. and Lehmann, R. P., Responses of Farm Animals to Sonic Booms, Sonic Boom Experiments at Edwards Air Force Base, Annex H., U. S. Department of Agriculture, Agriculture Research Service, Animal Husbandry Research Division, Beltsville, Maryland, September 20, 1966.
43. Bond, J., Responses of Man and Lower Animals to Acoustical Stimuli, U. S. Department of Agriculture, Agricultural Research Service, Animal and Poultry Husbandry Research Branch, Beltsville, Maryland, October 1, 1956.
44. Stadelman, W. J., The Effects of Sounds of Varying Intensity on Hatchability of Chicken Egg, *Poultry Science*, 37, 166-169, 1958.
45. Jeannoutot, D. W. and Adams, J. L., Progesterone Versus Treatment by High Intensity Sound as Methods of Controlling Broodiness in Broad Breasted Bronze Turkeys, *Poultry Science*, 40, 512-521, 1961.

Section 12

EFFECT OF NOISE ON STRUCTURES

Airborne noise normally encountered in real life does not normally carry sufficient energy to cause damage to most structures. The major exceptions to this general statement come from the sonic boom, which produces sudden and considerable changes in atmospheric pressure and from low frequency sound produced by large rocket-engine and certain types of construction equipment. Most of our data on the effects of noise on structures comes from studies of sonic booms generated by super-sonic aircraft, or from studies of structures located near low frequency sound sources.

In the preparation of this document, a review has been made of the effects of sonic booms on structure and the effects of noise induced vibrations.

SONIC BOOMS

Attempts have been made to clarify two issues within the constraints of currently available literature. These issues are summarized in the following questions:

1. What are the over-pressures produced by sonic booms generated by present military and commercial aircraft and how does the pressure vary with time?
2. What are the effects of these levels on physical structures?

Nature of Sonic Booms

The passage of an aircraft at speed greater than the local speed of sound in the atmosphere generates an impulsive noise called a sonic boom. The boom is observable at ground level as a succession of two sharp bangs, separated by a short time interval. Different parts of an aircraft radiate strong pressure waves in the air that grow into shocks known as leading shock and trailing shock. These two shocks form cones in the atmosphere that intersect the surface of the earth in hyperbolas. These interactions trace out a path called the "boom carpet." The length of the boom carpet may be thousands of miles.

Since it is often thought that sonic booms occur only as a supersonic aircraft passes the speed of sound, it should be emphasized that sonic booms occur at all times that a super sonic aircraft travels at faster speed than the speed of sound.

At the surface of the earth, the passage of a sonic boom is registered as an abrupt increase in pressure called the over-pressure, followed by a decrease in pressure below that of atmospheric pressure, thence a return to ambient or atmospheric pressure.

The intensity of a sonic boom is determined by the airplane characteristics and atmospheric conditions.^{1,2} The over-pressure for a supersonic bomber or an SST is typically around 100 newtons/m² (or about 2 lb/ft²) at the center of the boom carpet when cruising in level flight at an altitude of 60,000 ft and at a speed of Mach 2. In this example, the width of the boom carpet would be around 90 nautical miles, and the interval between shocks would be about 300 msec.³

Although a sonic boom is heard as two sharp bangs, most of the energy carried by a sonic boom is contained in a very low frequency range (often below 5 Hz).

Effects of Sonic Booms

The impulse from a sonic boom may set the components of a structure into vibration. Further, if the natural frequency of a structural component matches that of the impulse, the response of this component is greatly increased.³

Further, reflections from rigid surfaces present on the ground⁴ and/or focusing effects can also amplify the intensity of the wave.^{5,6} The point is that, because of possible changes in the impulse intensity from factors cited above, the response of a particular structure to sonic booms may be unpredictable. However, the response of a large collection of structures, such as various buildings in a community, can be fairly well predicted in a statistical sense.

Much of what we know about the effects of sonic booms on structures comes from studies conducted by the Air Force in the United States and some studies in Sweden, Britain and France over the last 10 years.

Field studies carried out in the United States involved sonic boom effects in three cities: St. Louis, Oklahoma City and Chicago. Each of these cities was subjected to systematic over-flights in a period ranging between 1961 and 1965. From these studies it appears that structures most susceptible to damage by sonic booms are secondary structural components such as windows and plaster.⁷ The over pressures tested were of the order of 50 to 150 newtons/m.²⁸ These results have been confirmed in some British studies.⁹

In a study by Parrot, data indicated that window glass can sustain air pressures up to 1000 newtons/m² without any damage.¹⁰ These results have been confirmed by ICAO Sonic Boom Panel.¹¹ These findings imply that a supersonic aircraft under normal conditions is not likely to give rise to over-pressures at ground level greater than 1000 newtons/m² and would not, therefore, cause serious damage to most structures. Caution must be exercised, however, in reaching such conclusions, since it is known that some atmospheric effects and/or factors, such as those cited previously, could lead to a magnification of boom over-pressures which could have serious effects on some structures. This could be particularly critical when some structures are already weakened because of some imperfection (such as misaligned windows) which renders the structure

more susceptible to unusual changes in pressures, even though the changes are small. An example of an unanticipated problem can be found in an incident which occurred in 1959 in Ottawa, Canada. In this instance, a supersonic aircraft was maneuvering at low altitude at speed below that of sound, when it accidentally went supersonic for a brief time. In the process, it caused damage estimated at \$300,000 to the window glass of Ottawa new airport terminal.¹² However, by and large, the effect of sonic boom can be accurately predicted, on a statistical basis.¹³

The results of some of the studies discussed are summarized in Table 12-1.

There has been only very scarce data on the effects of sonic booms on historical monuments and archeological structures; however, these structures are usually old and have sustained some damage from various environmental conditions, such as high winds, temperature and humidity fluctuations. It is, therefore, possible that repeated sonic booms may be an additional factor which, when added to the other environmental factors, could accelerate the "aging" of these structures. An answer to this question must, however, await further research on the long range effects of sonic booms.

NOISE INDUCED VIBRATIONS

High intensity, low frequency acoustical energy has been observed to set structural components, such as windows, light aluminum, or other flat materials, into sympathetic vibratory motions. As it is difficult to determine the transition between noise and vibration, many damaging effects may be the result of a complex interreaction between these two factors.

Effects on Materials

Measurable effects of noise on structure, while not common in most environmental situations, do occur in special circumstances. The heavy concentration of construction equipment in certain urban areas may produce a combination of vibratory energy transmission through the soil, supporting structures, and the air, which could conceivably affect fragile structures. Little research, however, has been accomplished to identify such effects. The launches of Saturn Rockets from Cape Kennedy have provided some data. From experimental and theoretical calculations of window glass breakage, one percent of the windows excited to the critical frequency of 30 Hz at 130 dB SPL (re. 0002 dynes/cm²) would be expected to break, and at 147 dB, 90 percent of the windows would be predicted to break. These effects occur only at certain frequencies, and would not appear if the excitation were at some higher frequencies until the sound pressure level was increased considerably.

Possible seismic motion from the sound of rocket launches has been measured and found negligible even at distances of 400 ft. from the launching site.

TABLE 12-1

SONIC BOOM DAMAGE DATA IN THREE CITIES*

Boom Dates Location	Metropol- itan Popu- lation	Total Super- sonic Over Flights	Maxi- mum Peak Over Pres- sure	No. of Com- plaints	No. Com- plaints Filed	No. Claims Paid	Value Claims Pd. in \$
St. Louis 1961-1962	2,600,000	150	86	5,000	1,624	825	58,648
Oklahoma City 1964	512,000	1,253	58	15,452	4,901	289	123,061
Chicago 1965	6,221,000	49	86	7,116	2,964	1,442	114,763
Total	9,333,000	1,452	84+	27,568	9,489	2,556	296,472

*This table is based on Table 1 of United States Environmental Protection Agency
Publication NTID 300.12, 1971.

+Average for the three cities

Effects on Humans

Vibration of buildings produced either by impulse noise, such as those associated with sonic booms, or certain types of construction equipment or low frequency noise from aircraft, rocket launches, construction equipment, heavy trucks or trains can produce reactions such as startle, discomfort, or interference with activities in humans. These effects have been recognized, and criteria has been proposed for human exposure.^{15,16}

SONIC FATIGUE

Sonic fatigue is a well known and well documented phenomenon. Fatigue, in general, occurs in ductile materials, such as metals, when subjected to repeated stresses of sufficient magnitude. Noise of high intensities can cause such stresses through sympathetic vibrations. These repeated stresses, in turn, produce failure in the material below its normal design load. The design engineer must take such effects into account when designing structures, such as aircraft and rockets, that may be subject to intense noise. However, the intensities encountered in most environmental noises are relatively low; therefore, in most instances, sonic fatigue will not be a problem, since the noise intensities must be above 140 dB SPL for sonic fatigue to occur.

EFFECT OF NOISE ON MATERIALS

Summary

The three general types of effects of noise on material are: sonic boom effects, noise induced vibration, and sonic fatigue. These are secondary effects of noise on the health and welfare of man. Sound can also excite buildings to vibrate, which can cause direct effects on man.

The effects caused by sonic booms are the most significant from an environmental standpoint. Sonic booms of sufficient intensity not only can break windows, but can damage building structures as well. Nevertheless, as with noise in general, the intensity of sonic booms can be controlled to levels that are completely innocuous with respect to material or structures.

Noise induced vibration can cause noticeable effects on community windows near large rocket launch sites. Construction may also cause such effects, but such relationships are poorly defined at this time.

Sonic fatigue is a very real problem where material is used near intense sound sources. However, such considerations are normally the responsibility of a design engineer and do not cause environmental problems.

Building vibrations excited by impulse noise such as sonic booms or from low frequency noise from aircraft or rockets can result in human reactions such as startle, discomfort or interference with some tasks. These effects occur primarily in the infrasound range and point toward the close relationship between sound and vibration. Criteria for human exposure to vibration are available but not discussed in this report.

SUMMARY—EFFECTS OF NOISE ON STRUCTURES

Airborne sound normally encountered in real life does not usually carry sufficient energy to cause damage to most structures. The major exceptions to this are sonic booms produced by supersonic aircraft, low frequency sound produced by rocket engines and some construction equipment, and sonic fatigue.

From an environmental point of view, the most significant effects are those caused by sonic booms on the secondary components of structures. These effects include the breaking of windows and cracking of plaster. Effects such as these have led to the speculation that historical monuments and archeological structures may age more rapidly when exposed to repeated sonic booms.

REFERENCES

1. Garrick, E. D. Atmospheric Effects of Sonic Boom, *NASA document SP-180*, 1969.
2. Maglieri, D. J. Sonic Boom Ground Pressure Measurements for Flights at Altitudes in Excess of 70,000 feet and at Mach Numbers up to 3.0, *NASA document SP-180*, 19-27, 1968.
3. National Bureau of Standards, The Effects of Sonic Boom and Similar Impulsive Noise on Structures, V. S. Environmental Protection Agency, 1971.
4. Slutsky, S. and Arnold, L. Coupled Elastic and Acoustic Response of Room Interiors to Sonic Booms, *NASA document SP-155*, 227-240, 1970.
5. Pierce, A. D., Maximum Overpressures of Sonic Boom Near the Cusps of Caustics, paper presented at Purdue Noise Control Conference, Purdue University, 1971.
6. Warren, C. H. E. Recent Sonic Bang Studies in the United Kingdom, paper presented at Sonic Boom Symposium 11, Houston, Texas, 1970.
7. Wiggins, J. H. Jr., Effects of Sonic Boom on Structural Behavior, *Material Research and Standards*, 7, 235-245, 1967.
8. McKinlye, R. W., Response of Glass in Windows to Sonic Booms, *Material Research and Standards*, 4, 594-600, 1964.
9. Warren, C. H. E. Recent Sonic Bang Studies in the United Kingdom, *JASA*, 51.
10. Parrott, T. L., Experimental Studies of Glass Breakage due to Sonic Booms *sound*, 1, 18-21, 1972.
11. ICAO Sonic Boom Panel Report doc 6649, SBP/11, 1970 reprinted in *Noise Control 1971*.
12. Ramsey, A. W. Damage to Ottawa Air Terminal Building produced by a sonic boom, *Material Research and Standards*, 4, 612, 1964.
13. Hershey, R. L., & Wiggins, J. H. Jr., Statistical Prediction Model for Glass Breakage from Nominal Sonic Booms Loads, FAA report No. FAA RD-73-79, 1973.
14. Sinex, C. H., Effects of Saturn Vehicle Launch Noise on Window Glass, TR-99-1, Dec 15, 1965, Safety Office, John F. Kennedy Space Center.
15. Von Gierke, H. E., on Noise and Vibration Exposure Criteria, *Archives of Environmental Health*, 11, 327-339, Sept. 1965.
16. ISO guide for the Evaluation of Human Exposure to Whole-Body Vibration, *ISO/DIS* 2631. 1972.

GLOSSARY

The following explanations of terms are provided to assist the reader in understanding some terms used in this publication:

A-WEIGHTED SOUND LEVEL—The ear does not respond equally to frequencies, but is less efficient at low and high frequencies than it is at medium or speech range frequencies. Thus, to obtain a single number representing the sound level of a noise containing a wide range of frequencies in a manner representative of the ear's response, it is necessary to reduce, or weight, the effects of the low and high frequencies with respect to the medium frequencies. The resultant sound level is said to be A-weighted, and the units are dB. A popular method of indicating the units, dBA, is used in this *Digest*. The A-weighted sound level is also called the noise level. Sound level meters have an A-weighting network for measuring A-weighted sound level.

ABSCISSA—The horizontal axis on a chart or graph.

ACOUSTICS—(1) The science of sound, including the generation, transmission, and effects of sound waves, both audible and inaudible. (2) The physical qualities of a room or other enclosure (such as size, shape, amount of sound absorption, and amount of noise) which determine the audibility and perception of speech and music.

ACOUSTIC REFLEX—The involuntary contraction of the muscles (stapedius and/or tensor tympani) of the middle ear in response to acoustic or mechanical stimuli.

ACOUSTIC TRAUMA—Damage to the hearing mechanism caused by a sudden burst of intense noise, or by blast. *Note:* The term usually implies a single traumatic event.

AIRBORNE SOUND—Sound that reaches the point of interest by propagation through air.

AIR CONDUCTION (AC)—The process by which sound is normally conducted to the inner ear through the air in the external auditory meatus and the structures of the middle ear.

AMBIENT NOISE (RESIDUAL NOISE; BACKGROUND NOISE)—Noise of a measurable intensity that is normally present.

ARTICULATION INDEX (AI)—A numerically calculated measure of the intelligibility of transmitted or processed speech. It takes into account the limitations of the transmission path and the background noise. The articulation index can range in magnitude between 0 and 1.0. If the AI is less than 0.1, speech intelligibility is generally low. If it is above 0.6, speech intelligibility is generally high.

AUDIBLE RANGE (OF FREQUENCY) (AUDIO-FREQUENCY RANGE)—The frequency range 16 Hz to 20,000 Hz (20 kHz). *Note:* This is conventionally taken to be the normal frequency range of human hearing.

AUDIOGRAM—A chart, table, or graph showing hearing threshold level as a function of frequency.

AUDIOMETER—An instrument for measuring the threshold or sensitivity of hearing.

AUDIOMETRY—The measurement of hearing.

AUDITORY TRAUMA—Damage to the hearing mechanism resulting in some degree of permanent or temporary hearing loss. *Note:* Auditory trauma may be caused by agents other than noise, e.g., head injury; burns; sudden or excessive changes of atmospheric pressure (cf. acoustic trauma).

AURAL—Of or pertaining to the ear or hearing.

BACKGROUND NOISE—The total of all noise in a system or situation, independent of the presence of the desired signal. In acoustical measurements, strictly speaking, the term “background noise” means electrical noise in the measurement system. However, in popular usage the term “background noise” is also used with the same meaning as “residual noise.”

BAND CENTER FREQUENCY—The designated (geometric) mean frequency of a band of noise or other signal. For example, 1000 Hz is the band center frequency for the octave band that extends from 707 Hz to 1414 Hz, or for the third-octave band that extends from 891 Hz to 1123 Hz.

BAND PRESSURE (OR POWER) LEVEL—The pressure (or power) level for the sound contained within a specified frequency band. The band may be specified either by its lower and upper cut-off frequencies, or by its geometric center frequency. The width of the band is often indicated by a prefatory modifier; e.g., octave band, third-octave band, 10-Hz band.

BASELINE AUDIOGRAM—An audiogram obtained on testing after a prescribed period of quiet (at least 12 hours).

BONE CONDUCTION (BC)—The process by which sound is transmitted to the inner ear through the bones of the skull (cf. air conduction).

BOOM CARPET—The area on the ground underneath an aircraft flying at supersonic speeds that is hit by a sonic boom of specified magnitude.

BROADBAND NOISE—Noise whose energy is distributed over a broad range of frequency (generally speaking, more than one octave).

C-WEIGHTED SOUND LEVEL (dBC)—A quantity, in decibels, read from a standard sound-level meter that is switched to the weighting network labeled “C”. The C-weighting network weights the frequencies between 70 Hz and 4000 Hz uniformly, but below and above these limits frequencies are slightly discriminated against. Generally, C-weighted measurements are essentially the same as overall sound-pressure levels, which require no discrimination at any frequency.

CENTRAL HEARING LOSS—Hearing loss resulting from injury or disease involving the auditory pathways or the auditory center of the brain or from a psychoneurotic disorder. *Note:* Central hearing loss can occur in the absence of any damage or deficiency in the peripheral hearing mechanism.

COCHLEA—A spirally wound tube, resembling a snail shell, which forms part of the inner ear and contains the end organ of hearing.

COMMUNITY NOISE EQUIVALENT LEVEL—Community Noise Equivalent Level (CNEL) is a scale which takes account of all the A-weighted acoustic energy received at a point, from all noise events causing noise levels above some prescribed value. Weighting factors are included which place greater importance upon noise events occurring during the evening hours (7:00 p.m. to 10:00 p.m.) and even greater importance upon noise events at night (10:00 p.m. to 6:00 a.m.).

COMPOSITE NOISE RATING—Composite noise rating (CNR) is a scale which takes account of the totality of all aircraft operations at an airport in quantifying the total aircraft noise environment. It was the earliest method for evaluating compatible land use around airports and is still in wide use by the Department of Defense in predicting noise environments around military airfields.

Basically, to calculate a CNR value one begins with a measure of the maximum noise magnitude from each aircraft flyby and adds weighting factors which sum the cumulative effect of all flights. The scale used to describe individual noise events is perceived noise level (in PNdB); the term accounting for number of flights is $10 \log_{10} N$ (where N is the number of flight operations), and each night operation counts as much as 10 daytime operations. Very approximately, the noise exposure level at a point expressed in the CNR scale will be numerically 35-37 dB *higher* than if expressed in the CNEL scale.

CONDUCTIVE HEARING LOSS (CONDUCTIVE DEAFNESS)—Hearing loss resulting from a lesion in the air-conduction mechanism of the ear.

CONTINUOUS NOISE—On-going noise, the intensity of which remains at a measurable level (which may vary) without interruption over an indefinite period or a specified period of time. Loosely, nonimpulsive noise.

CYCLES PER SECOND—A measure of frequency numerically equivalent to Hertz.

DAMAGE RISK CRITERION (DRC)—A graphical or other expression of sound levels above which a designated or a general population incurs a specified risk of noise-induced hearing loss.

DEAFNESS—100 percent impairment of hearing associated with an otological condition. *Note:* This is defined for medicological and cognate purposes in terms of the hearing threshold level for speech or the average hearing threshold level for pure tones of 500, 1000, and 2000 Hz in excess of 92 dB.

DECIBEL—The decibel (abbreviated "dB") is a measure, on a logarithmic scale, of the magnitude of a particular quantity (such as sound pressure, sound power, and intensity) with respect to a standard reference value. (0.0002 microbars for sound pressure and 10^{-12} watt for sound power).

DOSEMETER (NOISE DOSIMETER)—An instrument which registers the occurrence and cumulative duration of noise exceeding a predetermined level at a chosen point in the environment or on a person.

EAR DEFENDER (EAR PROTECTOR)—A device inserted into or placed over the ear in order to attenuate air-conducted sounds.

EARMUFF—An ear defender that encloses the entire outer ear (pinna). *Note:* Earmuffs are customarily mounted as a pair on a headband or in a helmet.

EARPLUG—An ear defender, having specified or standard acoustic characteristics, which upon insertion occludes the external auditory meatus. *Note:* Earplugs should be properly designed, made of suitable material, and correctly fitted to insure that they are acoustically effective and do not harm the ear.

EFFECTIVE PERCEIVED NOISE LEVEL (EPNL)—A physical measure designed to estimate the effective “noisiness” of a single noise event, usually an aircraft flyover; it is derived from instantaneous Perceived Noise Level (PNL) values by applying corrections for pure tones and for the duration of the noise.

FENCE—(Slang.) An arbitrary hearing level, greater than OdB, below which no hearing impairment is deemed to have occurred (“low fence”) or at which complete (100%) hearing impairment is deemed to have occurred (“high fence”).

FILTER—A device that transmits certain frequency components of the signal (sound or electrical) incident upon it, and rejects other frequency components of the incident signal.

FLUCTUATING NOISE—Continuous noise whose level varies appreciably (more than ± 5 dB) with time.

FREE SOUND FIELD (FREE FIELD)—In practice, a sound field in which the effects of spatial boundaries or obstacles are negligible.

FREQUENCY—The number of times per second that the sine-wave of sound repeats itself, or that the sine-wave of a vibrating object repeats itself. Now expressed in Hertz (Hz), formerly in cycles per second (cps).

HAIR CELL—Sensory cells in the cochlea which transform the acoustically derived disturbance of the cochlea into a nerve impulse.

HANDICAP (HEARING HANDICAP)—The occupational and social difficulty experienced by a person who has a hearing loss.

HARD OF HEARING—Having more than zero but less than 100 percent impairment of hearing for everyday speech or for pure tones of 500, 1000, and 2000 Hz. *Note:* This is defined, according to various standards, in terms of an elevated hearing threshold level of which the elevation is less than that defining deafness.

HEARING CONSERVATION (HEARING CONSERVATION PROGRAM)—Those measures which are taken to reduce the risk of noise-induced hearing loss.

HEARING DISABILITY—Hearing handicap prejudicing employment at full wages.

HEARING IMPAIRMENT—Hearing loss exceeding a designated criterion (commonly 25 dB, averaged from the threshold levels at 500, 1000 and 2000 Hz).

HEARING LEVEL—The difference in sound pressure level between the threshold sound for a person for the average for a group and the reference sound pressure level defining a standard audiometric threshold.

HEARING LOSS—Impairment of auditory sensitivity: an elevation of a hearing threshold level with respect to the standard reference zero.

HEARING THRESHOLD LEVEL—The amount by which the threshold of hearing for an ear exceeds a standard audiometric reference zero. Units: decibels.

HEARING THRESHOLD LEVEL FOR SPEECH—An estimate of the amount of socially significant hearing loss in decibels. *Note:* This is measured by speech audiometry or estimated by averaging the hearing threshold level for pure tones of 500, 1000, and 2000 Hz.

HERTZ—Unit of measurement of frequency, numerically equal to cycles per second.

IMPULSE NOISE (IMPULSIVE NOISE)—Noise of short duration (typically, less than one second) especially of high intensity, abrupt onset and rapid decay, and often rapidly changing spectral composition. *Note:* Impulse noise is characteristically associated with such sources as explosions, impacts, the discharge of firearms, the passage of supersonic aircraft (sonic boom), and many industrial processes.

INDUSTRIAL DEAFNESS—Syn. occupational hearing loss.

INFRASONIC—Having a frequency below the audible range for man (customarily deemed to cut off at 16 Hz).

INTERMITTENT NOISE—Fluctuating noise whose level falls once or more times to very low or unmeasurable values during an exposure.

INTERRUPTED NOISE—Syn. Intermittent noise (deprecated).

INVERSE-SQUARE LAW—The inverse-square law describes that acoustic situation where the mean-square pressure changes in inverse proportion to the square of the distance from the source. Under this condition the sound-pressure level decreases 6 decibels with each doubling of distance from the source. See also spherical divergence.

L₁₀ LEVEL—The sound level exceeded 10 percent of the time. Corresponds to peaks of noise in the time history of environmental noise in a particular setting.

L₅₀ LEVEL—The sound level exceeded 50 percent of the time. Corresponds to the average level of noise in a particular setting, over time.

L₉₀ LEVEL—The sound level exceeded 90 percent of the time. Corresponds to the residual noise level.

LEVEL—The value of a quantity in decibels. The level of an acoustical quantity (sound pressure or sound power), in decibels, is 10 times the logarithm (base 10) of the ratio of the quantity to a reference quantity of the same physical kind.

LOUDNESS—The judgment of intensity of a sound by a human being. Loudness depends primarily upon the sound pressure of the stimulus. Over much of the loudness range it takes about a threefold increase in sound pressure (approx. 10 dB) to produce a doubling of loudness.

LOUDNESS LEVEL—The loudness level of a sound, in phons, is numerically equal to the median sound pressure level, in decibels, relative to 0.0002 microbar, of a free progressive wave of frequency 1000 Hz presented to listeners facing the source, which in a number of trials is judged by the listeners to be equally loud.

MASKING—The action of bringing one sound (audible when heard alone) to inaudibility or to unintelligibility by the introduction of another sound. It is most marked when the masked sound is of higher frequency than the masking sound.

MICRO BAR—A microbar is a unit of pressure, equal to one dyne per square centimeter.

MICROPHONE—An electroacoustic transducer that responds to sound waves and delivers essentially equivalent electric waves.

MIDDLE EAR—A small cavity next to the ear drum in which is located the ossicular chain and associated structures.

MIXED HEARING LOSS—Hearing loss due to a combination of conductive and sensorineural deficit.

NARROW-BAND NOISE—A relative term describing the pass-band of a filter or the spectral distribution of a noise. *Note:* The term commonly implies a bandwidth of 1/3 octave or less (cf. Broad-band noise).

NOISE—Disturbing, harmful, or unwanted sound.

NOISE EXPOSURE—The total effective acoustic stimulation reaching the ear or the person over a specified period of time (e.g., a work shift, a day, a working life, or a lifetime).

NOISE EXPOSURE FORECAST—Noise exposure forecast (NEF) is a scale (analogous to CNEL and CNR) which has been used by the federal government in land use planning guides for use in connection with airports.

In the NEF scale, the basic measure of magnitude for individual noise events is the effective perceived noise level (EPNL), in units of EPNdB. This magnitude measure includes the effect of duration per event. The terms accounting for number of flights and for weighting by time period are the same as in the CNR scale. Very approximately, the noise exposure level at a point expressed in the NEF scale will be numerically about 33 dB *lower* than if expressed in the CNEL scale.

NOISE HAZARD (HAZARDOUS NOISE)—Acoustic stimulation of the ear which is likely to produce noise-induced permanent threshold shift in some fraction of a population.

NOISE-INDUCED HEARING LOSS (NIHL)—A sensorineural hearing loss caused by acoustic stimulation.

NOISE-INDUCED PERMANENT THRESHOLD SHIFT (NIPTS)—Permanent threshold shift caused by noise exposure.

NOISE-INDUCED TEMPORARY THRESHOLD SHIFT (NITTS)—Temporary threshold shift caused by noise exposure.

NOISE LEVEL—(Slang.) An averaged sound level (weighted sound pressure level). *Note:* The weighting must be specified.

NOISE LIMIT (NOISE EMISSION STANDARD)—A graphical, tabular, or other numerical expression of the permissible amount of noise which may be produced by a practical source (e.g., a vehicle or an appliance) or which may invade a specified point in a living or working environment (e.g., in a workplace or residence) in prescribed conditions of measurement.

NOISE AND NUMBER INDEX (NNI)—A measure based on Perceived Noise Level, and with weighting factors added to account for the number of noise events, and used (in some European countries) for rating the noise environment near airports.

NOISE POLLUTION LEVEL (L_{NP} or NPL)—A measure of the total community noise, postulated to be applicable to both traffic noise and aircraft noise. It is computed from the “energy average” of the noise level and the standard deviation of the time-varying noise level.

NOISE RATING (NR) NUMBERS (CONTOURS)—An empirically established set of standard values of octave-band sound pressure level, expressed as functions of octave-band center frequency, intended as general noise limits for the protection of populations from hazardous noise, speech interference and community disturbance. *Note:* The NR number is numerically equal to the sound pressure level in decibels at the intersection of the so designated NR contour with the ordinate at 1000 Hz.

NOISE SUSCEPTIBILITY—A predisposition to noise-induced hearing loss, particularly of an individual compared with the average.

NON-ORGANIC HEARING LOSS (NOHL)—That portion of a hearing loss for which no otological or organic cause can be found. Hearing loss other than conductive or sensorineural.

NONSTEADY NOISE—Noise whose level varies substantially or significantly with time (e.g., aircraft flyover noise). (Syn: fluctuating noise.)

NORMAL HEARING—The standardized range of auditory sensitivity of a specified population of healthy, otologically normal people determined in prescribed conditions of testing. (Deprecated.)

NORMAL THRESHOLD OF HEARING—Syn. Standard audiometric threshold.

OCCUPATIONAL HEARING LOSS—A permanent hearing loss sustained in the course of following an occupation or employment. *Note:* While noise is usually presumed to be the cause, other causes are possible (e.g., head injury).

OCTAVE—An octave is the interval between two sounds having a basic frequency ratio of two. For example, there are 8 octaves on the keyboard of a standard piano.

OCTAVE BAND—All of the components, in a sound spectrum, whose frequencies are between two sine wave components separated by an octave.

OCTAVE-BAND SOUND PRESSURE LEVEL—The integrated sound pressure level of only those sine-wave components in a specified octave band, for a noise or sound having a wide spectrum.

ORDINATE—The vertical axis on a chart of graph.

ORGAN OF CORTI—The end organ of hearing made up of hair cells and their associated and supportive structures.

OTOLOGICALLY NORMAL—Enjoying normal health and freedom from all clinical manifestations and history of ear disease or injury; and having a patent (waxfree) external auditory meatus.

PEAK SOUND PRESSURE—The absolute maximum value (magnitude) of the instantaneous sound pressure occurring in a specified period of time.

PERCEIVED NOISE LEVEL (PNL)—A quantity expressed in decibels that provides a subjective assessment of the perceived “noisiness” of aircraft noise. The units of Perceived Noise Level are Perceived Noise Decibels, PNdB.

PERCENT HANDICAP—Syn. Percent impairment of hearing.

PERCENT IMPAIRMENT OF HEARING (OVERALL) (PIHO)—The estimated percentage by which a person’s hearing is impaired, based upon audiometric determinations of the hearing threshold level at 500, 1000 and 2000 Hz (cf. Percent impairment of hearing for speech).

PERCENT IMPAIRMENT OF HEARING FOR SPEECH (PIHS)—An estimate of the percentage by which a person’s hearing is impaired, particularly at the frequencies (500, 1000, and 2000 Hz) deemed important for the perception of speech. *Note:* The scale 0 to 100 percent is arbitrarily set to correspond linearly with a standard range of values of hearing threshold level for speech in decibels (more than one standard has been used). The percent impairment of hearing increases by approximately 1.5 percent for each decibel of elevation of the estimated hearing threshold level for speech (average of 500, 1000 and 2000 Hz) in the standard ranges.

PERCEPTIVE HEARING LOSS—Syn. Sensorineural hearing loss. (Obs.)

PERMANENT HEARING LOSS—Hearing loss deemed to be irrecoverable.

PERMANENT THRESHOLD SHIFT (PTS)—That component of threshold shift which shows no progressive reduction with the passage of time when the putative cause has been removed.

PERSISTENT THRESHOLD SHIFT—Threshold shift remaining at least 48 hours after exposure of the affected ear to noise.

PHON—The unit of measurement for loudness *level*. $\text{Phon} = 40 + \log_2 \text{sone}$.

PINK NOISE—Noise where level decreases with increasing frequency to yield constant energy per octave of bandwidth.

PITCH—A listener's perception of the frequency of a pure tone; the higher the frequency, the higher the pitch.

PNdB—See perceived noise level.

PRESBYCUSIS—The decline in hearing acuity that normally occurs as a person grows older.

PURE TONE—A sound wave whose waveform is that of a sine-wave.

RECRUITMENT—The unusually great increase in loudness with rising sound levels.

RESONANCE—The relatively large amplitude of vibration produced when the frequency of some source of sound or vibration "matches" or synchronizes with the natural frequency of vibration of some object, component, or system.

RISK—That percentage of a population whose hearing level, as a result of a given influence, exceeds the specified value, minus that percentage whose hearing level would have exceeded the specified value in the absence of that influence, other factors remaining the same. *Note:* The influence may be noise, age, disease, or a combination of factors.

SEMI-INSERT EAR DEFENDER—An ear defender which, supported by a headband, occludes the external auditory meatus at the entrance to the ear canal.

SENSORINEURAL HEARING LOSS—Hearing loss resulting from a lesion of the cochlear end-organ (organ of Corti) or its nerve supply.

SHORT-LIVED NOISE—Noise of measurable intensity lasting without interruption (although the level may vary) for more than half one second but less than one minute (cf. Continuous noise; impulsive noise).

SOCIOCUSIS—Elevation of hearing threshold level resulting from or ascribed to non-occupational noise exposure associated with the general social environment and exclusive of elevation associated with aging.

SONE—The unit of measurement for loudness. One sone is the loudness of a sound whose level is 40 phons.

SONIC BOOM—The pressure transient produced at an observing point by a vehicle that is moving past (or over) it faster than the speed of sound.

SOUND—See acoustics (1).

SOUND LEVEL (NOISE LEVEL)—The weighted sound pressure level obtained by use of a sound level meter having a standard frequency-filter for attenuating part of the sound spectrum.

SOUND LEVEL METER—An instrument, comprising a microphone, an amplifier, an output meter, and frequency-weighting networks, that is used for the measurement of noise and sound levels in a specified manner.

SOUND POWER—Of a source of sound, the total amount of acoustical energy radiated into the atmospheric air per unit time.

SOUND POWER LEVEL—The level of sound power, averaged over a period of time, the reference being 10^{-12} watts.

SOUND PRESSURE—(1) The minute fluctuations in atmospheric pressure which accompany the passage of a sound wave; the pressure fluctuations on the tympanic membrane are transmitted to the inner ear and give rise to the sensation of audible sound. (2) For a steady sound, the value of the sound pressure averaged over a period of time. (3) Sound pressure is usually measured (a) in dynes per square centimeter (dyn/cm^2), or (b) in newtons per square meter (N/m^2). $1 \text{ N}/\text{m}^2 = 10 \text{ dyn}/\text{cm}^2$ 10^{-5} times the atmospheric pressure.

SOUND PRESSURE LEVEL (SPL)—20 times the logarithm to the base 10 of the ratio of the sound pressure in question to the standard reference pressure of $0.00002 \text{ N}/\text{m}^2$. Units: decibels (dB).

SPECTRUM—Of a sound wave, the description of its resolution into components, each of different frequency and (usually) different amplitude and phase.

SPEECH AUDIOMETRY—A technique in which speech signals are used to test a person's aural capacity to perceive speech in prescribed conditions of testing.

SPEECH DISCRIMINATION—The ability to distinguish and understand speech signals.

SPEECH-INTERFERENCE LEVEL (SIL)—A calculated quantity providing a guide to the interfering effect of a noise on reception of speech communication. The speech-interference level is the arithmetic average of the octave-band sound-pressure levels of the interfering noise in the most important part of the speech frequency range. The levels in the three octave-frequency bands centered at 500, 1000, and 2000 Hz are commonly averaged to determine the speech-interference level. Numerically, the magnitudes of aircraft sounds in the Speech-Interference Level scale are approximately 18 to 22 dB less than the same sounds in the Perceived Noise Level scale in PNdB, depending on the spectrum of the sound.

SPEED (VELOCITY) OF SOUND IN AIR—The speed of sound in air is 344 m/sec or 1128 ft/sec at 78°F .

SPL—See sound pressure level.

STANDARD—(1) A prescribed method of measuring acoustical quantities. Standards in this sense are promulgated by professional and scientific societies like ANSI, SAE, ISO, etc., as well as by other groups. (2) In the sense used in Federal environmental statutes, a standard is a specific statement of permitted environmental conditions.

STANDARD AUDIOMETRIC THRESHOLD—A standardized set of values of sound pressure level as a function of frequency serving as the reference zero for determinations of hearing threshold level by pure-tone audiometry.

STAPEDIUS REFLEX (STAPEDIAL REFLEX)—(Likewise, tensor tympani reflex.) The reflex response of the stapedius (likewise, tensor tympani) muscle to acoustic or mechanical stimulation. Commonly, synonymous with acoustic reflex.

STEADY NOISE (STEADY-STATE NOISE)—Noise whose level varies negligibly within a given period of time.

TEMPORARY THRESHOLD SHIFT (TTS)—That component of threshold shift which shows a progressive reduction with the passage of time after the apparent cause has been removed.

THRESHOLD OF HEARING (AUDIBILITY)—The minimum effective sound pressure level of an acoustic signal capable of exciting the sensation of hearing in a specified proportion of trials in prescribed conditions of listening.

THRESHOLD OF FEELING (TICKLE)—The minimum effective sound pressure level of an auditory signal capable of exciting a sensation of feeling or tickle in the ear which is distinct from the sensation of hearing.

THRESHOLD OF PAIN (AURAL PAIN)—The minimum effective sound pressure level of an auditory signal at the external auditory meatus which is capable of eliciting pain in the ear as distinct from sensations of feeling, tickle, or discomfort.

THRESHOLD SHIFT—An elevation of the threshold of hearing of an ear at a specified frequency. Units: Decibels.

TINNITUS—Ringing in the ear or noise sensed in the head. Onset may be due to noise exposure and persist after a causative noise has ceased, or occur in the absence of acoustical stimulation (in which case it may indicate a lesion of the auditory system).

TONE—A sound of definite pitch. A pure tone has a sinusoidal waveform.

TTS—See temporary threshold shift.

ULTRASONIC—Pertaining to sound frequencies above the audible sound spectrum (in general, higher than 20,000 Hz).

VASOCONSTRICTION—The diminution of the caliber of vessels, arteris and arterioles.

VESTIBULAR MECHANISM (SYSTEM)—The sensory mechanism which has to do with balance, locomotion, orientation, acceleration and deceleration.

WEIGHTING (FREQUENCY WEIGHTING)—The selective modification of the values of a complex signal or function for purposes of analysis or evaluation, in accordance with prescribed or standardized rules or formulae. *Note:* This may be done by computation or by the use of specified weighting networks inserted into electronic instrumentation so as to transform input signals.

APPENDIX A

Some Source References — Acoustics and Noise

GOOD INTRODUCTORY ARTICLES ON POTENTIAL SOLUTIONS

Mecklin, John M., *It's Time to Turn Down All That Noise*, Fortune, October, 1969.

Beranek, L.L., *Noise*, Scientific American, December, 1966.

GENERAL INTEREST BOOKS

**Report to the President and Congress on Noise*, NRC 500.1, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, December 31, 1971.

Bragdon, Clifford, *Noise Pollution: The Unquiet Crisis*, University of Pennsylvania Press, Philadelphia, 1972.

Berland, Theodore, *The Fight for Quiet*, Englewood Cliffs, Prentice-Hall, 1970.

Still, Henry, *In Quest of Quiet*, Harrisburg, Pa. Stackpole Books, 1970.

Baron, Robert Alex, *The Tyranny of Noise*, New York, St. Martin's Press, 1970.

Burns, William, *Noise and Man*, Philadelphia, Pa., Lippincott, 1969.

EFFECTS OF NOISE ON PEOPLE

**Effects of Noise on People*, NTID 300.7, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

Proceedings, Conference on *Noise as a Public Health Hazard*, June, 1968, American Speech and Hearing Association, 9030 Old Georgetown Road, Washington, D.C. 20014 (\$5.00).

Proceedings, American Association for the Advancement of Science *International Symposium on Extra-Auditory Physiological Effects of Audible Sound*, Boston, Massachusetts, December, 1969. Obtain from Plenum Press, 227 West 17th Street, New York 10011 (\$15.00).

Kryter, K., *Effects of Noise on Man*, Academic Press, 1970 (\$19.50).

Stevens, S. S. and Warshofsky, Fred, *Sound and Hearing*, Time-Life Books (Life Science Library Series), New York, 1970.

LEGISLATION

**Laws and Regulatory Schemes for Noise Abatement*, NTID 300.4, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

**State and Municipal Non-occupational Noise Programs*, NTID 300.8, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

Hildebrand, James L. (ed.), *Noise Pollution and the Law*, William S. Hein & Co., Inc., Law Book Publishers, Buffalo, New York, 1970.

Working Paper for the Noise Legislation Workshop, The National Symposium on State Environmental Legislation sponsored by the Council of State Governments, Washington, March 16-18, 1972. (Obtain from EPA, Office of Noise Abatement and Control, Washington, D.C. 20460.)

The Noise Around Us: Findings and Recommendations, Report of the Panel on Noise Abatement to the Commerce Technical Advisory Board, U.S. Department of Commerce, September 1970 (Obtained from U.S. Government Printing Office, Washington, D.C. 20402—\$.50). Full report of the committee available as COM-71-00147, from National Technical Information Service, Springfield, Virginia 22151—\$6.00.

A Report to the 1971 Legislature on the Subject of Noise, Pursuant to Assembly Concurrent Resolution 165, 1970, California Department of Public Health, 2151 Berkeley Way, Berkeley, California. (Released March 22, 1971)

Toward a Quieter City, report of the Mayor's Task Force on Noise, City of New York, 1970. (Obtain from N.Y. Board of Trade, 295 Fifth Avenue, New York City, \$1.50)

Transportation Noise Pollution: Control and Abatement. NASA Langley Research Center and Old Dominion University, 1970 (obtain from Dr. Gene Golia, Old Dominion University, P.O. Box 6173, Norfolk, Virginia 23508.)

A Brief Study of a Rational Approach to Legislative Control of Noise, National Research Council of Canada, NRC 10577, Ottawa, 1968.

OF SPECIAL INTEREST TO DESIGNERS, ARCHITECTS AND URBAN PLANNERS

Beranek, Leo L. (ed.), *Noise and Vibration Control*, McGraw-Hill Book Co., New York, 1971.

**The Effect of Sonic Boom and Similar Impulsive Noise on Structures*, NTID 300.12, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

**Community Noise*, NTID 300.3; U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

**Transportation Noise & Noise from Equipment Powered by Internal Combustion Engines*, NTID 300.13, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, Technical Document, December 31, 1971.

**Noise from Construction Equipment & Operations, Building Equipment, & Home Appliances*, NTID 300.1, U.S. Environmental Pro-

tection Agency, Office of Noise Abatement and Control, December 31, 1971.

**Fundamentals of Noise: Measurement, Rating Schemes, & Standards*, NTID 300.15, U.S. Environmental Protection Agency, Office of Noise Abatement and Control, December 31, 1971.

Department of Housing and Urban Development: 1. Circular 1390.2, Subject: *Noise Abatement and Control*; Departmental Policy, Implementation Responsibilities, and Standards, 1971. 2. *Noise Assessment Guidelines*, August 1971, in furtherance of Section 4a of the above mentioned Circular, available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, price 70 cents, Stock Number 2300-1194.

Berendt, R. D., Winzer, G. E. and Burroughs, C. B., *A Guide to Airborne, Impact and Structure-borne Noise Control in Multi-family Dwellings*, FHA Report FT-TS-24, January, 1968.

Meyer, Harold B. and Goodfriend, Lewis, *Acoustics for the Architect*, Reinhold Publishing Co., New York, 1957.

Solutions to Noise Control Problems in the Construction of Houses, Apartments, Motels and Hotels, AIA Files No. 39-E, Owens-Corning Fiberglass Corporation, Toledo, Ohio, 1963.

Building Code Section on Noise Insulation Requirements in Multifamily Dwellings, Local Law No. 76 for 1968, City of New York.

Proceedings, Conference on *Noise as a Public Health Hazard*, June, 1968, Amer. Speech & Hearing Assoc., 9030 Old Georgetown Road, Washington, D.C. 20014. (Especially see McGrath, Dorn, "City Planning and Noise.")

Land Use Planning with Respect to Aircraft Noise, October 1964. Can be obtained from

*These reports are available from the National Technical Information Service, 5258 Port Royal Road, Springfield, Virginia 22151; and from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. They will not be available from the EPA directly.

the Federal Aviation Administration; the National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia 22151; and the U.S. Air Force, refer to AFM 86-5, TM 5-365, NAVDOCKS P-98.

Harris, C. H., ed., *Handbook of Noise Control*, McGraw-Hill Book Co., 1957 (includes chapters on community noise and city planning, anti-noise ordinances, and noise control requirements in building codes).

PERIODICALS

Noise/News, published bi-monthly by the Institute of Noise Control Engineering. For information contact Circulation Department, P.O. Box 1758, Poughkeepsie, N.Y. 12601. (This is a new newsletter dedicated to publication of news items related to the scientific and engineering aspect of noise, its control, and its effects on people.)

Sound and Vibration, published monthly. For information contact Sound and Vibration, 27101 E. Oviatt Road, Bay Village, Ohio 44140.

TVASNAC 'Quotes.' Town-Village Aircraft Safety & Noise Abatement Committee Newsletter, published monthly. For information contact Editor, TVASNAC Quotes, 196 Central Avenue, Lawrence, N.Y. 11559.



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