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Temporary Auditory Threshold Shift in Guinea Pigs Caused By High Speed Dental Drill Noise

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TEMPORARY AUDITORY THRESHOLD SHIFT IN GUINEA PIGS CAUSED BY HIGH SPEED DENTAL DRILL NOISE

by

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DEDICATION

To my parents, Sidney and Florence Waldman, for their encouragement, support and understanding.

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VITA

The author, Donald Jerome Waldman, the son of Sidney Waldman and Florence Waldman, was born on March 13, 1953, in Los Angeles, California.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

REVIEW OF THE LITERATURE

Different sounds affect people in different ways. Quiet music often can be relaxing. Loud noises are usually bothersome and many can cause mental and physical disorders such as nervousness, indigestion, and headache. Noise can decrease the ability to concentrate, increase accident proneness and reduce overall efficiency (Rapp, 1960). Very loud noises such as explosions or continuous exposure to high noise levels, will cause damage to the hearing mechanism (Rosenblith and Stevens, 1953).

Robin (1960), Sockwell (1971) and Burns (1973) list the following variables to be considered in determining the possibility of hearing loss:

1. Intensity or loudness, measured in decibels. The maximum noise level (or sound pressure level) found to have a damaging effect of a permanent nature (i.e., injurious level) differs slightly with different frequencies; high-pitched noise is more injurious than low pitched noise. The borderline between innocuous and injurious levels differs with different authors in different countries. In the U.S. and Japan it is 90 db at 2,000-3,200 cps and 85 db at 3,200-6,400 cps.
2. Length of exposure. This factor must be taken in connection with the other four, and especially with the susceptibility of the person exposed. As a general rule, the longer the exposure, the more likelihood of a damaging effect.

3. Continuity of exposure. A certain continuity of a noise at each exposure is necessary if damage is to be produced. This is a different mechanism from the "explosive" damage of a very loud single noise. Prolonged noise at injurious levels are more likely to cause damage if rest periods or intervals are not given. The ear has a remarkable ability to adapt itself to tolerate noise if the nerve-endings are allowed rest-periods. The length of rest periods necessary to obtain a return of normal hearing will differ with various factors (especially the susceptibility one), and would have to be determined in each case.
4. Frequency of the noise. The cochlea is more vulnerable in those parts receptive to frequencies between 2,500 and 6,000 cps. Exposure to such noise causes a characteristic loss in the audiometer readings near the 4,096 cps level.
5. Susceptibility. This varies tremendously. There are no certain reasons for this, though some relatively minor ones are known. These are: a. Age. As a rule, older persons are more susceptible than young. b. Health. Debilitated and tired persons are more susceptible. c. Persons with certain ear conditions, such as otosclerosis and nerve deafness. d. Sex. Research by Glorig (1959) seems to indicate less loss in women in comparative circumstances.

Robin (1960) reports that the degree of hearing loss caused by

noise varies enormously, depending on the factors mentioned above. The audiograph usually shows a dip of about 15-20 db somewhere between the 2,048 and 4,096 cps frequencies. In some patients there may be a gradual progressive slope down to the 8,192 cps frequency. With increasing exposure, the loss may progress even to about 50 db at 4,096 cps and 80 db at 8,192 cps.

Age and existent ear damage or disease can accelerate hearing loss. Davis and Silverman (1970) have tabulated the percentage risk of developing a hearing handicap for various ages and exposure levels of noise.

The injurious quality of noise is greatly affected by resonance and reflection. It is (damped) by absorbent coverings of walls, barriers, etc. The distance from the source of noise has a marked effect on the sound pressure level. This is reduced at the rate of 6 db per doubling of the distance from the noise (Glorig, 1959).

Pain is not an adequate indicator of danger to hearing. Absence of auditory pain should not be construed to mean absence of injury to hearing (Davis, et al., 1958). Pain is produced in the ear when noise levels are of the order of 130 db; noise-induced hearing loss, however, may be produced by prolonged exposure to considerably lower noise levels. Pain is not a necessary accompaniment of noise-induced hearing loss.

The most important function of hearing in civilized life is the hearing of every day human speech. It is the loss of this function that causes disability in the medicolegal as well as in the social sense.

Davis, et al., (1958) discusses types of hearing loss. Impairments

of hearing are divided into several classes, depending on the part of the auditory system that is affected, the nature of the impairment or its cause. Impairment of the external and the middle ear prevent normal conduction of sound to the sense organ, and these conditions are grouped together as conductive hearing loss. Hearing loss due to impairment of the cochlea or auditory nerve is termed sensory or nerve deafness. A combination of causes of hearing impairment can occur and is called a "mixed" type of hearing loss. For example, an ear may have some presbycusis, some persistent noise-induced hearing loss, and an additional temporary threshold shift all at the same time.

Acute loss of hearing may be produced by a single blast or explosion, or by a brief exposure to an extremely intense noise. The injury to the sense organ in the inner ear produced in this way may be different from the injury produced by longer, repeated exposures to less intense noise. The term "acoustic trauma" is appropriate for the acute injury from a single blast. The single sudden event distinguishes acoustic trauma from noise-induced hearing loss. The distinction is important because the prognosis for recovery is very different. Acoustic trauma may cause any degree of injury to hearing, but recovery, either partial or complete, extends over a period of weeks or months, wheareas in the hearing loss that is induced gradually by repeated noise exposure the improvement is substantially complete within a few days after removal from the noise.

Certain types of sense-organ hearing loss, such as are found in Meniere's disease, may indicate an abnormal sensitivity to noise-induced hearing loss. Other types of hearing loss, such as presbycusis, may not

affect susceptibility and may simply elevate the threshold still more. Conductive hearing loss in general tends to protect against noise-induced hearing loss rather than to increase the susceptibility to it. Continued hazardous noise-exposure may therefore be quite permissible for some men with some types of hearing loss, but strongly contraindicated for others (Davis, et al., 1958).

Corso (1959) in a study to determine the normal thresholds of hearing for pure tones for an age-stratified sample of subjects drawn from a population exposed to minimal levels of industrial noise, tested the hearing of 500 males and females, 18-49 years old. The results of this study showed that women have more sensitive hearing than men. For both men and women, there is a decrease in hearing sensitivity with increasing age and a progressive spreading of the loss from the higher to the lower frequencies. Men are more affected than women, with the hearing loss occurring at an earlier age and producing a greater degree of auditory impairment.

Exposure to noise of sufficient intensity for long enough periods of time can produce detrimental changes in the inner ear and seriously decrease the ability to hear. Some of these changes are temporary and last for minutes, hours, or days after the termination of the noise. After recovery from the temporary effects, there may be residual permanent effects on the ear and hearing that persist throughout the remainder of life. The changes in hearing that follow sufficiently strong exposure to noise are complicated. They include distortions of the clarity and quality of auditory experience as well as losses in the ability to detect

sound. These changes can range from only slight impairment to nearly total deafness (Miller, 1974).

The primary site of injury is found to be in the receptor organ of the inner ear. The outer ear, eardrum, and middle ear are almost never damaged by exposure to intense noise. The eardrum, however, can be ruptured by extremely intense noise and blasts (von Gierke, 1965). Excess exposure to noise can result in the destruction of hair cells and collapse or total destruction of sections of the organ of Corti. In addition, auditory neurons may degenerate (Miller, 1974).

Evidence from human cases and animal experiments suggests that the loss of sensory cells must be quite extensive in the upper part of the cochlea (that part which is important for the perception of low frequency sounds) before this damage is reflected as a change in threshold. In the lower part of the cochlea (that part which is important for the perception of high-frequency sounds) losses of sensory cells over a few millimeters are sometimes reflected as changes in hearing (Bredberg, 1968).

The mechanism by which overexposure to noise damages the auditory receptor is not well understood. Very intense noise can mechanically damage the organ of Corti. Thus, loud impulses such as those associated with explosions and firing of weapons can result in vibrations of the organ of Corti that are so severe that some of it is simply torn apart. Other very severe exposures to noise may cause structural damage that leads to rapid "breakdown" of the processes necessary to maintain the life of the cells of the organ of Corti. Such an injury is, as mentioned above,

an "acoustic trauma."

Overexposure to noise of lower levels for prolonged periods of time also results in the degeneration of the hair cells and accessory structures of the organ of Corti. Such injuries are called "noise-induced cochlear injuries." Many theories have been proposed to explain noise-induced cochlear injuries. One notion is that constant overexposure forces the cells to work at too high a metabolic rate for too long a period of time. As a result the metabolic processes essential for cellular life become exhausted or poisoned, and this leads to the death of the cells. In a sense, the receptor cells can die from overwork.

No matter what theory is eventually found to be correct, certain facts are established beyond doubt (Miller, 1974). Excessive exposure to noise leads to the destruction of the primary auditory receptor cells, the hair cells. There can be other injuries to the organ of Corti that can range from mild distortion of its structure to collapse or complete degeneration. The auditory neurons may also degenerate. All of these are highly specialized. Once these cells are destroyed, they do not regenerate and cannot be stimulated to regenerate; they are lost forever.

The structural changes caused by exposure to noise can be seen clearly in surface preparations of the guinea pig cochlea. Exposure to 140 db for only 30 seconds produces gross distortion of the outer hair cells in the basal turn of the cochlea and tears them off the basilar membrane. One hundred and thirty decibels for 20 minutes produces a slightly less dramatic effect, and 72 hours exposure at 120 db produces

only just visible damage. When the animals are killed a year after exposure to 120 db, patchy loss of the hair cells is seen with retrograde degeneration of the neurons. Noise below 90 db seems to produce no morphological damage even after long exposure (Lancet, 1975). It is of course difficult to relate these results in guinea pigs directly to man since there may be considerable species variability.

von Krammer (1968) reports that acoustic trauma results from a lower intensity sound irritant. The injury is not associated with pain. Chronic auditory lesions are irreversible, and localize on the basilar membrane at the point of highest resonance of the traumatic frequency. Zones above 4,000 cps seem to be particularly vulnerable. Auditory lesions may remain undetected for years, says von Krammer, since an individual may experience a 28 per cent loss of hearing (30 db) before becoming aware of a problem in oral communication.

Schuknecht (1974) says it appears that moderate intensities of acoustic stimulation incite metabolic activity leading to exhaustion of enzymes and glycogen stores, diminished oxygen tension, decreased energy output, and reversible alterations in organelles of the sensory cells and nerve endings. The functional manifestation is auditory fatigue or temporary threshold shift. More intense stimulation results in irreversible morphological alterations and permanent hearing loss.

Davis (1953) exposed guinea pigs to intense tones of frequencies ranging from 185-8,000 cps at sound pressures from 138-148 db. Each frequency caused damage at specific localized regions of the cochlea. No single tone, at the intensities and durations employed, injured all of

the cochlea.

Miller (1963) found that, although in cats (as in man) moderate exposures to white noise produced a maximum temporary threshold shift (TTS) at 4 kc, when he increased the severity of the exposure until permanent losses developed, the locus of maximum loss was found at 2 kc, an octave lower.

Stockwell and Harlow (1969) exposed guinea pigs to pure tones of 125, 500, 1,000, 2,000, and 4,000 Hz at intensities of 130 and 150 db sound pressure level (SPL) for a period of one or four hours. Each cochlea was prepared histologically and a cochleogram of the sensory cell population was constructed to show the pattern of hair cell loss. The radial distribution of damage was related to exposure frequency. Lower frequencies produced proportionally greater damage in distal hair cell rows than did higher frequencies. Hair cell damage caused by exposures at 150 db was severe and over wide areas, extending from the supposed site of maximum stimulation primarily toward the base. Exposures to 130 db caused damage which was more selective than has been reported previously. Lesions produced by a 4,000 Hz tone appeared near the stimulation maximum for that frequency, but lesions caused by lower frequencies tended to appear progressively nearer the base with respect to stimulation maxima. The existence of multiple peaks of damage was a prominent feature.

Hamernik and Henderson (1974) exposed chinchillas and guinea pigs to high intensity impulse noise and afterwards examined the integrity of the organ of Corti. In histological preparations the greatest effect was

seen in loss of outer hair cells, with fewer inner hair cells affected. In evoked response audiometry tests a consistent finding has been the growth of a temporary threshold shift to a maximum as much as 14 hours after exposure before recovery begins. This phenomenon has been observed by others (Sitler, 1972 and Luz, 1971).

Spoendlin and Brun (1973) exposed guinea pigs to noise at intensities between 110 db and 140 db for exposure times of 30 seconds to one week. They then examined the cochleas of the animals and found that the degeneration of damaged elements is not immediate, but proceeds and reaches its final and permanent status only after a longer period of time. They conclude that direct mechanical destruction as well as metabolic exhaustion are competing factors in acoustic traumatic damage of the cochlea. Direct mechanical damage is usually irreversible and appears immediately after relatively short exposures at high intensities above a certain level whereas metabolically induced damage is partly reversible, occurs after long exposures with moderate intensities and develops more slowly over a longer period of post-exposure time. They delineate three zones of intensity of acoustic traumatic effects: up to 90 db, practically no damage is produced; from 90 to 130 db permanent acoustic traumatic damage mainly of the metabolic type occurs, and above 130 db severe irreversible structural damage occurs. They found that exposure time and intensity do not seem to be equally responsible for structural damage. At higher intensity levels, exposure intensity is by far more decisive on the extent of damage than is exposure time. Thus, equal total energy does not seem to produce always the same amount of damage.

Smitley (1971) explains temporary threshold shift (TTS) to be a reduction in hearing sensitivity (threshold) resulting from noise exposure provided that thresholds return to preexposure levels with time (minutes, hours or days) after cessation of the noise exposure.

Miller (1963) defines a threshold shift as the postexposure threshold, expressed in decibels, minus the preexposure threshold. If a threshold shift is measured at a time that exceeds a few seconds after the cessation of the exposure and if this threshold shift declines to zero over time, the animal is said to have suffered a temporary threshold shift. If threshold shifts are measured that are stable and persist over a period of several weeks, then these threshold shifts are said to be persistent threshold shifts (PTS) and permanent injury to the auditory mechanism is inferred.

The primary measure of hearing loss is the hearing threshold level. The hearing threshold level is the level of a tone that can just be detected. The greater the hearing threshold level, the greater the degree of hearing loss or partial deafness. An increase in a hearing threshold level that results from exposure to noise is called a "threshold shift."

Some threshold shifts are temporary and they diminish as the ear recovers after the termination of the noise. Frequently repeated exposures can produce temporary threshold shifts that are chronic, though recoverable when the exposures cease. When a threshold shift is a mixture of temporary and permanent components, it is a compound threshold shift. When the temporary components of a compound threshold shift have disappeared

(that is, the ear has recovered as much as it ever will), the remaining threshold shift is permanent. Permanent threshold shifts persist throughout the remainder of life.

Temporary threshold shifts can vary in magnitude from a change in hearing sensitivity of a few decibels restricted to a narrow region of frequencies to shifts of such extent and magnitude that the ear is temporarily, for all practical purposes, deaf. After cessation of an exposure, the time for hearing sensitivity to return to near-normal values can vary from a few hours to two or three weeks. In spite of efforts in many laboratories, the laws of temporary threshold shifts have not yet been completely determined. There are large numbers of variables that need to be explored. Also, there are probably several different underlying processes which need explication before the laws of noise-induced temporary threshold shifts will be completely understood (Miller, 1974).

Nonetheless, certain generalizations seem to be correct (Ward, 1963). Noises with energy concentrations between about 2,000 and 6,000 Hz probably produce greater temporary threshold shifts than noises concentrated elsewhere in the audible range. In general, A-weighted sound levels must exceed 60-80 db before a typical person will experience temporary threshold shifts even for exposures that last as long as 8-24 hours. All other things being equal, the greater the intensity level above 60-80 db and the longer the time in noise, the greater the temporary threshold shift. However, exposure durations beyond 8-24 hours may not produce further increases in the magnitude of the shifts (Mills, et al., 1970). Another interesting property of temporary threshold shifts is

that such shifts are usually greatest for test tones 1/2-1 octave above the frequency region in which the noise that produces the shift has the greatest concentration of energy. It should also be noted that under certain conditions, contractions of the muscles of the middle ear can offer significant protection from exposure to intense sound. Finally, there is less temporary shift when an exposure has frequent interruptions than when an exposure is continuous.

Acquired hearing damage can be aggravated by frequent periods of temporary threshold shift. The rest periods between exposures are significant in reducing the temporary threshold shift. This will affect individuals differently in relation to their personal susceptibility (Davis, et al., 1958).

Miller (1963) investigated the aural effects of exposure to intense noise by behavioral measurements of the auditory sensitivity of cats before and after such exposures and by histological examination of their cochleas. With sound pressure levels of 105-115 db, the maximum TTS is at 4 kc and the TTS is greater above 4 kc than below it. Miller hypothesizes that (a) the qualitative characteristics and the underlying processes of noise-induced TTS are the same for cat and man, and (b) a given exposure will produce more TTS in cat than in man. However, in order to produce equivalent TTS in cat and man, the noise level must be 18 db higher than that for cat.

Kylin (1959) studied the temporary threshold shift of hearing in 220 persons 15 minutes after cessation of exposure for 2 hours to various

types of octave-band filtered white noise with sound pressure levels ranging from 75 to 115 db. As regards the bands 75-150, 150-300, 600-1,200, and 2,400-4,800 cps, the power to produce TTS was least at 75-150 cps and increased with each octave band up to 2,400-4,800 cps. At the latter band the effect was approximately similar to that at 4,800-10,000 cps. The TTS increased with rising sound pressure level within the respective octave bands. The affected auditory region increased with the sound pressure. The threshold shifts primarily appeared at frequencies situated within the octave band used for exposure to noise and within the next highest bands. Frequencies below the region used for exposure were only exceptionally influenced.

Glorig (1962) found that at 4,000 Hz, the TTS measured 2 min. after exposure (TTS_2) to a steady noise for 8 hours was equal to the PTS after 10 years of working in the same industrial noise environment.

Ward and associates (1959) conducted a study based upon the concept of TTS and its relation to permanent threshold shift (PTS). They assumed that if a noise fails to produce a TTS, it cannot produce a permanent loss.

On the other hand, Sataloff, et al., (1965) found no apparent relationship between PTS and TTS for any frequency between 2,000 and 8,000 Hz among workers exposed to about 90 db sound pressure level over a ten year period.

Nixon and Glorig (1961) did a study of male workers in three levels of noise. They found that the amount of threshold shift at 4,000 Hz from these occupational exposures shows no further increase after about 10 years

of exposure although the threshold shifts for lower frequencies continue to increase.

Ward (1965) reports that there is some evidence that the average TTS produced in normal ears by a day's work performed in a given noise environment is monotonically related to the average permanent losses produced after many years of daily exposure.

Temporary threshold shift grows linearly with the logarithm of time, reports Ward (1963), although he gives no physiological mechanism shown to be responsible for this empirical relation. He does, however, review evidence bearing on this relation. He also states that more TTS was produced by high frequencies than by low, and that maximum fatigue was usually produced at a frequency about half an octave above the exposure frequencies.

It is true that under certain conditions one can consistently observe less fatigue after exposure at a given level than after exposure at a lower level. This effect is probably due to a shift in mode of vibration of the stapes, caused by strong contraction of the middle-ear muscles (Ward, 1962).

In most experiments concerned with the effects of noise upon auditory sensitivity, the duration of the noise exposure ranges from a few minutes to a few hours. Over this range of durations the relation between temporary threshold shift and exposure duration is linear when duration is plotted on a logarthmic scale (Ward, 1959 and Kylin, 1960). For very short or for very long exposures, however, this log-time relation may not

predict the obtained data (Mills, 1970). For example, when the duration of the exposure is shortened sufficiently, the TTS predicted by the log-time relation becomes infinitely large.

An exponential relation between TTS and duration of exposure has been proposed (Keeler, 1968 and Botsford, 1968). Critical to this approach is the assumption that TTS reaches a maximum or asymptote and that this maximum is not exceeded as the exposure is prolonged indefinitely.

An asymptotic TTS has been measured by Carder and Miller (1969). After exposing trained chinchillas to noise for 2-21 days, they reported that after 24-48 hours of exposure the growth of TTS was asymptotic. Furthermore, the time necessary to reach an asumptote, an index of the time constant of TTS, was independent of the level of the exposure, the spectrum of the exposure, and the test-tone frequency at which the TTS was measured. They interpret this as reflecting a physiological equilibrium state.

Melnick (1974), also using chinchillas, investigated TTS resulting from 16 hours of continuous exposure to an octave band of noise, 300 to 600 Hz, at octave band levels of 80, 85, 90 and 95 db. Ten subjects were tested at each level. Thresholds were at frequencies ranging from 125 to 8,000 Hz during and after the noise exposure. Group data indicate that the 16 hour exposure period was not long enough to clearly establish asymptotic levels of TTS. As a group, these subjects recovered to within 5 db of preexposure threshold measures by 58 hours postexposure. Individual subject variability was notable in the pattern of TTS development.

Mills (1970) in an experiment with a human subject, concludes that

the time for TTS to reach asymptote for human listeners of noise at 92.5 db for 29.5 hours is somewhere between 4 and 12 hours. Recovery from this TTS to 0 db TTS required about 6 days. Mills observes that a striking aspect of recovery from asymptotic TTS is the long time required for complete recovery of the threshold even though the initial magnitude of the TTS was small, 27.5 db. When TTSs of similar magnitudes are produced by exposures of very much less than 95 db, recovery proceeds much more rapidly than it does from asymptotic TTS. This recovery data seems to disprove an earlier generalization (Ward, 1959) that recovery from a temporary threshold shift measured two minutes after exposure (TTS_2) depends only on its magnitude regardless of how that TTS_2 was produced.

Mills (1970) presents two hypothesis in a discussion on asymptotic TTS. One is that if the TTS audiogram measured after 12 hrs. of continuous exposure to a sound of fixed level and spectrum truly represents an asymptote, then that TTS audiogram is an upper bound on the noise-induced permanent threshold shift (NIPTS) audiogram that could be produced by that sound no matter what the schedule of exposure. This hypothesis is necessarily correct if TTS does reach a true asymptote in about 12 hrs. and if threshold shifts either remain constant or diminish after the cessation of an exposure.

The other hypothesis is that it is possible that the asymptotic TTS audiogram will be exactly matched for a given schedule of exposure to a particular noise by the asymptotic NIPTS audiogram. This would be particularly likely if the fatigue processes that produce TTS are the same processes that produce destruction of the sensory cells and the organ of Corti;

but this possibility is unproven. Evidence that NIPTS is an asymptote at least for test tones at 4 KHz is given by Nixon and Glorig (1961) and Taylor (1965).

One of the enigmas about hearing loss produced by noise exposure is that people with apparently similar histories of experience with noise do not necessarily develop similar hearing losses. Much of the interest in investigations of TTS has resulted because measures of TTS were thought to be a good means for predicting susceptibility to hearing loss from noise exposure. Thus far, the data produced by investigations of TTS have not been fruitful in predicting noise-induced permanent threshold shift (Melnick, 1974).

One of the first researchers to present evidence that susceptibility is not unitary was Theilgard (1949). On several different occasions he exposed 7 listeners to 4 different pure tones (of 500, 1,000, 2,000 and 4,000 cps) at 100 db and found only slight consistency of findings in given ears from frequency to frequency. Furthermore, he tested both ears of 4 of his subjects, and found appreciable differences. He concludes: "It would thus be quite illusory to characterize the degree of 'auditory fatigue' of a single individual by referring to the magnitude of the effect recorded at a single frequency. Some results using the same test twice show that there is a more-than-negligible variability from day to day."

Kryter (1962) in an article on audio analgesia, came to a conclusion about noise exposure in general, that is, to avoid any damage risk to hearing, a person should not be exposed to more noise or sound in one

day than that which would cause, on the average, a temporary threshold shift of more than 20 db at any frequency when measured two minutes after exposure (TTS_2).

Ward (1958) says that when TTS_2 exceeds about 50 db some subjects may experience a permanent elevation in their threshold of hearing. The amount of this elevation, e.g., permanent hearing loss, is usually much less than the initial loss. The amount of recovery is greatest within the first 24 hrs., but some recovery may be found even a month after the exposure (Davis, 1950).

On the other hand, when the measured hearing loss or threshold shift is less than 50-60 db, it is nearly always found that the person's hearing returns to normal within 24 hrs. after exposure. When the threshold shift is less than 30 db, the subject recovers from his fatigue usually within 2 to 4 hrs, at the most within 24 hrs. (Ward, 1959). This finding is in disagreement with that of Mills (1970).

The specification of TTS_2 less than or equal to 20 db in the average subject after exposure to noise is recommended with the view in mind that: (a) if recovery from auditory fatigue is complete within 24 hrs., the exposure causing that fatigue can be repeated every 24 hrs. without leading to a permanent loss; (b) the range of individual differences with respect to susceptibility to auditory fatigue is such that when the average person sustains a TTS_2 of 20 db, the most tender-eared person will show a TTS_2 of less than 50 db (Kryter, 1962). This is supported by the experiments of Davis (1950) and Ward (1959).

Ward (1959) found that for brief durations (from 1/2 sec. up to about 1 min.), an exposure followed by an equally long period of relative silence causes much less auditory fatigue than one would predict. In order to predict the effects of intermittent broad-band sound, he developed the formula $TTS_2 = 1.06R(S-91)$ (log T/1.7), where R is the "on" fraction, S the over-all sound pressure level of the noise, and T the exposure duration in minutes, including the off periods.

Slow recovery from noise-induced threshold shifts probably holds whenever the exposure is severe either in terms of the total duration or in terms of the amount of threshold shift present a few minutes after the termination of the noise. Recovery from temporary threshold shift appears to be very slow when the initial threshold shift exceeds 35-45 db (Ward, 1960), when the exposure lasts as long as about 12 hrs. (Mills, 1970). For example, it has been shown that exposure to a noise with an A-weighted sound level of about 80 db for two days results in small temporary threshold shifts that do not disappear for several days (Mills, 1970).

Noise induced permanent threshold shifts can accumulate as exposures are repeated on a near-daily basis over a period of many years as was shown in a study of jute weavers by Taylor (1965). He found that as the exposures are repeated year after year, the ear becomes less and less able to recover from the temporary threshold shift present at the end of each day. Also, the amount of TTS present at the end of each day's work increases. As the exposures are repeated, the noise-induced temporary threshold shifts become permanent or nearly so.

Kryter (1966) states that a TTS_2 of no more than 10 db at 1,000 cps or below, no more than 15 db at 2,000 cps, and/or no more than 20 db at 3,000 cps or above will be deemed an acceptable general daily amount of temporary threshold shift in young adults with normal hearing. He postulates that TTS_2 will rank the various kinds of exposures encountered in a single-day's assignment in the same manner as would TTS measured at any later time after exposure. This assumption is supported by evidence that TTS's maintain their rank order during recovery, and by evidence that recovery from TTS_2 does not depend on how the TTS_2 was produced (Ward, 1960). Kryter also postulates that the NIPTS (Noise-induced permanent threshold shift) eventually produced after many years of habitual exposure, 8 hrs. per day, is about numerically equal to the TTS_2 at 1,000 cps produced in young normal ears by an 8 hr. exposure to the same noise; the NIPTS at 2,000 cps produced after many years of habitual exposure, 8 hrs. per day, is about 5 db less than the TTS_2 at 2,000 cps produced in young normal ears by an 8 hr. exposure to the same noise; while the NIPTS at 4,000 cps eventually produced after many years of habitual exposure, 8 hrs. per day, is about 3 db greater than the TTS_2 at 4,000 cps produced in young normal ears by an 8 hr. exposure to the same noise. Work by others supports this postulation (Glorig, 1961; Kylin, 1960; Nixon, 1961). Kryter makes a broad generalization that mean $NIPTS_{10\ yr.}$ (NIPTS after 10 years) in a given population has a linear regression on TTS_2 with slope one and intercept zero. If this generalization is correct for all test frequencies and all classes of exposures, then $NIPTS_{10\ yr.}$ would, on

the average, be equal to TTS_2.

Kryter (1973) believes that the presently accepted medical definitions of the American Academy of Opthalmology and Otolaryngology (AAOO) of the relations between pure-tone hearing levels and the understanding of everyday speech underestimate hearing impairment for speech as measured by laboratory clinical tests, and as experienced by people in real life.

Fox (1972) in a discussion on occupational hearing loss, reviews the procedure set by the American Medical Association used to convert threshold readings of hearing impaired persons into percentage of hearing impairment. If the average hearing level at 500, 1,000 and 2,000 Hz is 25 db or less, usually no impairment exists in the ability to hear everyday speech under everyday conditions. On the other extreme if the average hearing threshold at 500, 1,000 and 2,000 Hz is over 92 db the impairment for everyday speech is considered to be total. If the average of the three frequencies 500, 1,000 and 2,000 Hz exceeds 25 db an allowance of 1 1/2 percent for each db is made up to a maximum of 100 percent. For determining binaural hearing impairment the AMA formula specifically recommends that the percentage of hearing of the better ear is multiplied by five, the resultant figure is added to the percentage of impairment in the poorer ear and the sum is divided by six. The final percentage represents the binaural hearing impairment.

Ewersen (1973) reports a statistically clear relationship between exposure time and severity of hearing loss after testing 1,108 patients who had a hearing loss as a result of working in noisy industrial environments

for up to 50 years. These environments averaged a continuous noise level above 90 db. Ewertsen states that it is generally accepted that continuous noise of 85-90 db or more is hazardous to hearing. As with other senses, hearing undergoes a deterioration with age. In a person with a noise-induced hearing loss, this loss will be added to that due to aging. It is, therefore, usual for people with occupational hearing losses to get along quite well through their thirties and forties, until they come to the age of 50 years, when they begin to feel their hearing handicap more and more. This means that the noise wears out the reserves 10-20 years earlier than we would have expected due to aging.

In 1972, the National Institute of Occupational Safety and Health (NIOSH) in the United States published a "Criteria for a Recommended Standard... Occupational Exposure to Noise." This document stated that there was still risk of noise induced hearing loss at 90 dbA, and that the current standards should be lowered to 85 dbA.

Martin (1975) did a study of the hearing of workers in a steel mill, the results of which indicate that there is increased risk of noise induced hearing loss at noise exposure levels between 85 and 90 dbA. This risk increase ranges from 4.0% to 22.5% for those subjects 50 to 65 years of age. This is 4.0% to 22.5% above the normal 10% impairment due to presbycusis. Length of exposure to noise ranged from one to six hours per eight hour shift.

Larsen (1953) states that occupational deafness is a slow, progressive nerve type deafness (as opposed to conductive type deafness). The first

stage indicated by audiometry shows a dip at 4,000 cps. Its pathologico-anatomical condition consists of degenerative changes in the organ of Corti and the spiral ganglion, beginning in the external hair cells and most pronounced in the basal turn.

Rock and roll music is often loud and, one would assume, could produce a hearing loss, especially among the musicians playing the music. Rintelmann (1968) reports that when rock and roll musicians were exposed to approximately 105 db SPL of music for an average of 11.4 hrs. a week for 2.9 years, 95% of them did not incur hearing losses. He attributes this, in part, to exposure that is intermittent with a series of short on-times followed by very brief off times.

Lipscomb (1969) noted numerous hair cells destroyed in guinea pig cochleas after the animals were exposed to a total of 88 hrs. (27 stimulation periods) of high intensity rock and roll music. He also tested school children for high frequency hearing impairment. High school seniors had a higher incidence of high frequency hearing impairment than younger groups. He concludes that excess exposure to loud music could account for the increasing incidence of high frequency hearing loss among teenagers and college freshmen.

Various guidelines have been developed for the purpose of predicting the maximum intensity levels and durations of noise exposure to which individuals can be safely exposed with out incurring permanent hearing loss. These are termed damage risk criteria (DRC). Smitley and Rintelmann (1971) exposed 40 people to rock and roll music at 110 db SPL for 60 minutes. One

group received continuous sound, the other intermittent. Their results show that at all frequencies and at all recovery times, the continuous exposure condition produced greater threshold shifts than did the intermittent exposure condition. DRC based on TTS, they conclude, would suggest that rock and roll music is potentially hazardous.

Air turbine dental handpieces when operated at ultra speeds produce an annoying high frequency whine. Aside from the objectionable aspect, the question has been raised as to the possibility of hearing loss from long term exposure to this noise.

Sound analysis studies have reported the frequencies of handpieces of all manufacturers (Ward and Holmberg, 1969; Smith and Coles, 1966). Early models recorded ranges to 8,000 cps. Improved design in ball bearing handpieces, and improved exhaust systems have reduced frequencies to the 2,000 to 6,000 cps range. Potential hazards exist at frequencies over 1,000 cps (Hopp, 1962). It must be recognized that the sound analysis studies are carried out under ideal analysis conditions with new equipment operating at optimum rotation speeds. Handpiece wear, bur concentricity, misuse, poor maintenance, and individual operatory design can influence the frequencies from individual handpieces (Schuchard and Flower, 1974).

Decibel readings can be influenced by the aforementioned conditions. New free-running roller bearing handpieces at recommended air pressures record 68 to 97 db readings; Midwest American reports the recording for air-bearing handpieces to be approximately 10 db lower. Additional conditions that vary the recording are the distance and position of the handpiece in

relation to the recording device (Delheim, 1971). A decibel level drop of 8 to 12 db has been recorded when handpieces were subjected to cutting torque (Schuchard and Flower, 1974). Accepted decibel ratings are safe range, 0 to 70 db; moderate risk range, 80 to 100 db; and high risk range, 110 to 130 db (Schuchard and Flower, 1974).

Exposure times are intermittent and have been estimated by Midwest American and by Delheim (1971) at from 12 to 45 minutes a day. Variations of exposure time depend on the mode and type of dental practice and vary within individual offices (Sockwell, 1971).

Sockwell (1963) reports that the noise from a belt driven free running handpiece at 12 inches is 76 db. The cutting action produces an increase in the noise level to 84 db and a frequency range of 7,100 to 7,500 cps. Diamond stones are about 3 db quieter in operation than carbide burs at 2,000 cps according to Penn and Kortsch (1963). According to Sockwell (1963) the sound level may drop about 2 db during cutting procedures in the mouth because of a loss of speed.

Even among handpieces from the same manufacturer there is considerable variation in noise levels. Kessler (1961) reports noise levels from 75 to 104 db at a distance of six inches when air turbine handpieces are operated at maximum speeds.

Cantwell, et al, (1965) found that turbine handpieces with air bearings have noise levels of less than 68 db free running at 40 to 60 pounds of air pressure. Without the friction and noise of ball bearings, these handpieces produce speeds above 800,000 rpm, thus producing some frequencies

that are ultrasonic. Most adults do not hear frequencies above 12,000 cps.

Weston (1962) tested air turbine drills from five countries. He found that those with the turbine in the head were slightly louder (74-88 db) than those with the turbine in the handpiece and a shaft drive to the head (70-75 db).

Robin (1960) did tests to analyze the noise of four different turbine drills. In each the speed was run at 250,000 rpm. The distance from the head was 12 inches. (This was considered to be the average distance between a dentist's ear and the patient's tooth). Analysis of the frequencies of the noise generated showed that there were, in fact, three components, at 5,500, 4,000 and 2,400 cps. The level of noise at each frequency was, respectively, 80 db, 60 db and 60 db. In one drill, however, the 4,000 cps gave an 80 db level. This shows that the 4,000-5,000 cps frequencies do tend to reach an injurious level. Robin concludes that the noise level of high speed drills is such that, without any precautions, some dentists may gradually get permanent cochlear damage, and that manufacturers should ensure that such a drill does not exceed a noise level of about 75 db at 12 inches.

Noise above ambient levels in any environmental situation poses a potential health hazard. The advent of low torque, ultrahigh-speed cutting instruments and their high frequency of audible vibration, in addition to background noise, has led investigators to study possible auditory damage to dentists resulting from extended exposure.

Brenman (1960) exposed human volunteer subjects to a controlled amount of airborne noise from an ultrasonic prophylactic instrument and an air turbine. Tooth contact exposures were not made. The effect of the ultrasonic prophylaxis instrument was more pronounced than with the air turbine. The human volunteers exhibited audiograms with a dip in the 4,000-6,000 cycle frequency range after an exposure to the ultrasonic prophylaxis instrument. This effect appeared to be reversible within three days.

Taylor (1964) measured sound pressure levels and did octave band analyses on a number of air rotor dental drills. These noise levels were then equated with exposure time and the results compared with damage risk criteria, above which hearing damage will occur. A survey of the hearing of dental practitioners was undertaken, using pure-tone air-conduction audiometry in a special quiet chamber. His results show that, after 3 to 4 years use of the air rotor drill, dental practitioners begin to show hearing defects in the 6Kc and 4Kc audiogram regions.

In tests done by Ward and Holmberg (1969) the hearing of dentists was compared to that of people who were not dentists. They concluded that intermittent high speed drill noise is only slightly hazardous to hearing, especially when compared with other sources of hearing damage in everyday life, such as gunfire.

Keller (1965) tested 89 turbine drill users. The average hearing curve showed a distinct loss of hearing above 2,048 cps and especially between 2,048 and 8,192 cps. The frequency range of the drills was about

2,000 to 6,300 cps, or about 120,000 to 378,000 rpm. No comparable losses were noted in dentists who did not use high speed turbine drills.

On the other hand, Nixon and Knox (1967) tested the hearing of 30 dentists. A control group was also examined. The results of the tests over a 4 year period showed similar threshold changes occurring in both dentists and controls.

Hopp (1962) investigated the acoustic trauma potential of exposure to high speed instruments. He analyzed noise levels and frequency spectrums of high speed drills and found they were capable of producing hearing losses. He did not find, however, significant hearing loss among dental students tested over a 23 week period.

Taylor, et al., (1965) tested a population of dentists and found statistically significant high frequency hearing losses at 4,000 and 6,000 cps.

Weatherton, et al., (1972) did a study over three years involving students and staff practitioners at a school of dentistry. They reported no change for the students. However, the staff participants exhibited some change. They did not conclude that turbine noise was involved because the changes could be attributed to age.

Another study involving dental students (Skurr and Bulteau, 1970) over a two year period found evidence of hearing damage of unknown cause in a portion of the 21 to 23 year old participants. The students who had hearing impairment at the start of the study suffered further hearing deterioration. They concluded that it was difficult to assess how much

hearing loss was due to turbine noise. Hearing deteriorated in 5 students of a group of 17 who were considered to have normal hearing initially.

Both Terranova (1967) and Grundy (1966) discuss surveys of dentists asked to list symptoms attributed to the air turbine. Terranova reports that the most important disadvantage given by the participants was the noise produced by the air turbine drill. Grundy reaches a similar conclusion.

Bernier and Knapp (1959) report that high speed dental drills producing noise levels above 75 db in frequency ranges between 1,000 and 8,000 cps may cause hearing damage.

Cooperman, Wallace and Nerlinger (1965) say that several ultra speed handpieces produced noise levels that bordered on or exceeded dangerous exposure levels. Morrant (1960) has similar findings.

BuMed Instruction 6260.6A, a document issued by the Bureau of Medicine and Surgery of the U.S. Navy (Naval National Medical Center, 1962), recommends that a hearing conservation program be instituted when the sound pressure level reaches 85 db in specified octave bands, and that this program should be mandatory where the noise level reaches 95 db. However, this criterion applies to continuous noise exposure for a lifetime. The damage risk criterion can be applied to high speed dental drills by taking into consideration the daily time exposure factor. Frequency analyses reveal that the highest noise levels are in the octave bands 2,400 to 4,800 and 4,800 to 9,600 cps. The maximum permissible daily exposure times in minutes for various decibel levels at these frequencies are as

follows: 480 min.,85 db; 240 min.,88 db; 150 min., 90 db; 50 min.,95 db; and 15 min.,100 db. Analyses of the noise measurements at 12 inches from the dentist's ear indicates that repeated daily exposure of less than 150 minutes to the high speed drill is within permissible limits and should not constitute a hazard to hearing.

Dental handpieces are normally used intermittently. Schubert and Glorig (1963) report that the high speed handpiece is used by the average dentist for only about 12 minutes each day. It is their opinion that if the 12 minutes were continuous, a noise level even as high as 101 db would not cause a hearing loss. Even though the general practitioner may have only 12 minutes exposure, others may be exposed for much longer times, such as those who specialize in operative or crown and bridge procedures. Dellheim (1971) reported longer periods of use.

Cantwell, Tunturi and Manny (1960) reported that several air turbine handpieces of the same make produced noise levels above 84 db in the range between 4,800 and 9,600 cps. They concluded that although a temporary threshold shift may occur, no permanent hearing loss could be expected from the normal use of the instruments tested. They base their conclusion on hearing loss standards set up by the Air Force. These regulations are based on statistics to cover the majority. It should be kept in mind, as stated above, that individuals will differ in their susceptibility to noise induced hearing loss.

Kessler (1961) states that although many authorities feel that the length of time the average dentist uses his air turbine handpiece is so

short that he does not have to worry, it would probably be a good idea for dentists to have audiometric checkups at regular intervals.

Glorig (1966) states that hearing loss occurs in the higher frequencies first and is not noticed ordinarily because it does not interfere with normal conversation. However, if audiograms reveal a hearing loss in the higher frequencies, this could mean a susceptibility to further hearing loss at the lower levels. In such cases he advises one to avoid continued exposure to high noise levels and to use some type of protective device.

It is impossible to protect individuals from every type and degree of hearing injury. The direction should be to prevent occurrence of severe impairment and to retard the development of established injury. The Council on Dental Materials and Devices (Schuchard and Flower, 1974) recommends that preventive measures for noise attenuation should be directed in three areas: optimum maintenance of rotary equipment, reduction of the ambient noise level in the operatory (soundproofing, acoustical ceilings, baffle drapes, resilient floors, rational location of the compressor and other noise-making equipment), and personal protection through use of ear plugs (cotton with petroleum jelly, defibered soft glass, or plastic plugs, capable of 20 to 35 db reduction). The Council recommends that practitioners concerned about the potential impairment should have an otologic examination and have an audiometric evaluation in a silent room, to assess the present condition. Noise levels in the individual offices should be studied with monitoring periods of more than a week. An audiometric evaluation should

be made after a typical workday and again at the beginning of the next day to observe temporary threshold shift and apparent recovery. Annual tests of hearing should be taken. Robin (1960) suggests similar measures.

Dry cotton earplugs afford little or no protection (Glorig, 1966). Kessler (1960) cautions that care should be exercised in selecting the correct type of plug. Contraindicated is a sound valve type of plug where a sudden impact noise will close the valve, with the valve opening again after the sound has passed. This type is suitable for use in industry; for example, in drop forge work. What is needed is an earplug for continuous attenuation.

Sloane (1966) describes how to easily construct an ear plug that will effectively reduce the sound at the 4,000 cps frequency range.

Ear muffs designed to cover the external ear can be used. At frequencies above 1,000 cps, certain correctly designed muffs provide more protection than plugs (Glorig, 1966).

Many investigators have used electrophysiological, histological or audiometric methods on laboratory animals exposed to varying degrees of noise in an endeavor to study noise-induced hearing loss. In contrast to most electrophysiological and all histological methods, the methods of making audiometric measurements do not disturb or interfere with the later performance of the audiotry mechanism. A compelling advantage of a method of behavioral audiometry is the fact that changes in the audiogram clearly define one of the most important aspects of what is meant by damage or injury to hearing (Miller, 1963).

Anderson and Wedenberg (1965) in a search for behavioral phenomena in the guinea pig that might serve as a response in hearing tests led to two interesting observations: (i) on exposure to moderate cold the animal develops series of shivers of a remarkably regular and persistent nature, and (ii) these series of shivers are partly, if not completely, inhibited if the animal is disturbed or excited. In the terms "disturbed or excited" lies the essence of these experiments. The tone, which to the conditioned guinea pig is an alarm signal, like many other external discomforting disturbances momentarily inhibits the shiver, producing an "immobility response." Miller and Murray (1966) describe this response as varying from a brief pause in ongoing activity to a full-blown response with a characteristic posture in which the back is arched, the head is up, and the front legs are extended; often there is an apparent exophthalmus.

To reinforce the tone as an alarm signal, Anderson and Wedenberg trained their guinea pig with a 2,000 Hz tone at 60 db sound pressure level presented for 3 seconds and terminated with a 0.2 second shock. This was repeated every 20 seconds during a training period of 30 minutes, of which there were two or three daily. Anderson and Wedenberg call their hearing test method "shiver audiometry."

Crifo (1973) proposed several simplifications to the original method of shiver audiometry proposed by Anderson and Wedenberg. The first simplification is the use, as a source of pure tones of an audiometer, instead of the more complex electro-physiological equipment. And, instead of complex systems of refrigeration, Crifo did his shiver audiometry experiments

in a cold room intended for biochemical use.

Using behavioral testing (shiver audiometry), Crifo (1973) compared the albino guinea pig's hearing threshold with the pigmented guinea pig's threshold. At all frequencies tested, he reported that the albino animals displayed significantly better hearing (lower thresholds) than the pigmented group.

In contrast, using cochlear microphonic potentials from the inner ear, Nuttall (1974) found that the maximum cochlear potential magnitudes obtainable at three frequencies (100, 1,000 and 4,000 Hz) from both albino and pigmented guinea pigs were statistically alike.

Crifo (1974) has shown that shiver audiometry of the guinea pig combined with morphological study of the spiral organ can be useful in the identification of possible ototoxic properties of drugs, as experimental tests with well-known ototoxic drugs have demonstrated.

MATERIALS AND METHODS

Subjects

Eighteen male albino guinea pigs (Cavia porcellus) weighing 200 to 900 grams were used. Throughout the training period ten of these died of unknown causes. Of the remaining eight, two did not respond to the training procedure. Two were designated experimental and were exposed to continuous high speed dental drill noise. Two were designated experimental and were exposed to intermittent high speed dental drill noise. The remaining two animals acted as controls, undergoing the same training and testing procedures as the experimental animals, the only difference being in that they were not exposed to high speed dental drill noise.

The auditory threshold and, if any, the temporary threshold shift, of each guinea pig was measured using the Shiver Audiometry technique developed first by Anderson and Wedenburg (1965) and later modified by Crifo (1973). Further modifications in equipment and technique were made for this experiment, however, to enable one to measure temporary threshold shift within minutes after the cessation of noise exposure.

Materials

The apparatus unique to this modified procedure was the test chamber. The purpose of the chamber was twofold. The first was to provide an environment which could be rapidly cooled without disturbing the animal. (Anderson and Wedenburg used a fan to blow cold air onto the test animal. Both

the noise of the fan and the motion of the air against the animal's fur could be disturbing). The second purpose was to provide an environment free of external stimuli, such as light, motion, or noise. Thus, the chamber was designed to be both thermally and acoustically insulated.

The chamber consisted of a wooden framework, measuring 1 1/2 feet x 1 1/2 feet x 3 feet. To the inside and outside of this framework were attached 3/8 inch plasterboard panels with a 1/2 inch layer of fiberglass insulation sandwiched in between. All inside seams were caulked with a latex caulking compound. Five eighths of an inch styrofoam panels were glued to all inside surfaces and over these were glued heavy gauge aluminum foil. The outside of the chamber was coated with a 1/8 inch layer of plaster and painted (Fig. 1).

Near the roof at one end of the chamber were two ventilation ducts. Two doors were on one wall; the larger door was for access to the inside of the main chamber. The smaller door covered the cooling compartment to which dry ice was added during the experiment. This container was made of sheet metal and measured 4 inches x 6 inches x 9 inches. All seams were caulked with a silicone rubber caulking compound. Carbon dioxide from the dry ice was vented from the container through a 3 foot long plastic hose of 1/2 inch diameter attached to the door. Both the large and small doors had foam rubber seals. The test chamber provided a 35 decibel attenuation of noise.

Inside the chamber was a plexiglass sheet mounted on foam rubber squares. The guinea pigs were secured in an adjustable restraining device

(Fig. 2). This was attached by two rubber bands to the plexiglass sheet. Shiver vibrations from the guinea pigs were picked up by a pneumatic pulse transducer (Physiograph Mk III, NARCO Instrument Co.) attached to the sheet. Electric signals from the transducer were sent to the preamplifier of an electrosphygmograph (Physiograph MkIV, Model ESG-3000, NARCO Instrument Co.) which in turn was connected to a physiograph (Physiograph Four, NARCO Instrument Co.). Attached with tape to the guinea pig's left hind paw was a wire electrode. This received electrical shocks from a stimulator (Physiograph MkV, NARCO Instrument Co.).

An audiometer (Zenith, Model ZA110TW) was used to produce the tones. This instrument generates pure tones at a frequency of 125 to 8,000 cps with an intensity range of 0 to 110 decibels in 5 decibel steps. It was connected to an amplifier (Bogen Challenger, Model C-10) which in turn was connected to a 6 inch loudspeaker (Realistic, Model 40-1285A) in the test chamber. Mounted at a 20 degree angle to the horizontal plane, the loudspeaker was fixed 30 centimeters from the guinea pig's pinna. This is the estimated distance of the dentist's ear from the operating drill.

Calibration of the audiometric instruments was necessary to insure that the tone emitted from the loudspeaker at a distance of 30 centimeters was equivalent to the tone indicated on the audiometer. This was accomplished by placing a sound level meter (Simpson, Model 886) 30 centimeters from the loudspeaker and then adjusting the volume control on the amplifier until the decibel level indicated on the sound level meter was the same as on the audiometer. Thus, when the audiometer was set at 50 db, the amplifier was adjusted until the sound level meter indicated 50 db.

A recording of a high speed dental drill (Star Futura II, Star Mfg. Co.) in operation was made on a one minute continuous loop tape cassette (TDK Endless Cassette, Model EC 1). This recording was used for continuous drill noise exposure. Thirty seconds were erased and the tape was then used for the intermittent (30 sec. on, 30 sec. off) drill noise exposure segment of the experiment. Recording and playback of the tape were done on a cassette tape recorder (Panasonic, Model RQ-309 AS). During recording the dental drill was held 30 centimeters from the microphone. The dental drill's sound level was 87 db when running freely and 90 to 96 db during cutting. The recording was played back through the loudspeaker in the test chamber and calibrated such that the sound level averaged 92 db.

Procedure

Before audiometric testing of the animals could take place the animals had to be conditioned to respond to the various tones of the audiometer. The object was to condition the animals to associate an electrical shock (a noxious stimulus) with tones produced by the audiometer so that by the end of training the animals would shift into a catatonic state upon hearing the tone alone, thus causing an interruption in their shivering cycle. Tones used in training ranged from 0 to 60 db at 6,000 cps.

Dry ice was added to the dry ice container about 45 minutes before training began, allowing the chamber to cool to about 4 degrees centigrade. The animal was restrained and placed in the test chamber with the wire electrode attached to his left hind paw. A distinct shiver pattern could

be detected within 5 to 10 minutes.

Once a shiver pattern was established, training could begin. At 20 second intervals a 60 db tone of 3 seconds duration was transmitted from the audiometer and was immediately followed by an electrical shock of 25 to 100 volts with a 2 millisecond duration 200 times per second for 1 to 3 seconds. In subsequent training sessions the sound level was lowered in 5 db increments down to 0 db. Conditioning to the sound took from 60 to 100 sessions of 20 to 30 minutes each. See figure 3.

For the experimental phase of the experiment, two animals were exposed to 2, 4, 6 and 8 hours of continuous high speed dental drill noise. Forty-five minutes before termination of the noise exposure dry ice was added to the dry ice container to cool the test chamber enough so that by the end of the exposure the animal would be engaged in a regular pattern of shivering. This allowed testing of the animals almost immediately after the termination of the noise. The hearing of each animal was tested at 2, 5, 10, 15, 20, 25, 30, 45 and 60 minutes after exposure to the high speed dental drill noise to detect a temporary threshold shift, if any, and the course of recovery from the shift. Testing was carried out by transmitting tones from the audiometer, beginning at 0 db and increasing the sound level until an interruption in the shiver cycle was observed on the physiograph. This was taken to be the hearing threshold at this point. A shock was then given to the guinea pig to prevent extinction of the conditioned response.

Two other animals were subjected to a similar procedure, however,

they were exposed to intermittent, instead of continuous, high speed dental drill noise. The control animals also underwent similar training and test procedures, the only difference being in that they were not exposed to any high speed dental drill noise.

A time period of at least 24 hours was allowed between consecutive exposures of the same animal. For example, after the 2 hour exposure and test the animal was allowed a rest period of at least 24 hours before the 4 hour exposure and test.

1a.

1b.

Figure 1a. Test chamber. Guinea pig is inside in the restraining device mounted on the plexiglass sheet. Note the dry ice container, visible to the left of the main door.

1b. Equipment as set up for the experiment. Visible from left to right are the test chamber, preamplifier, tape recorder, amplifier and audiometer. In the foreground is the physiograph.

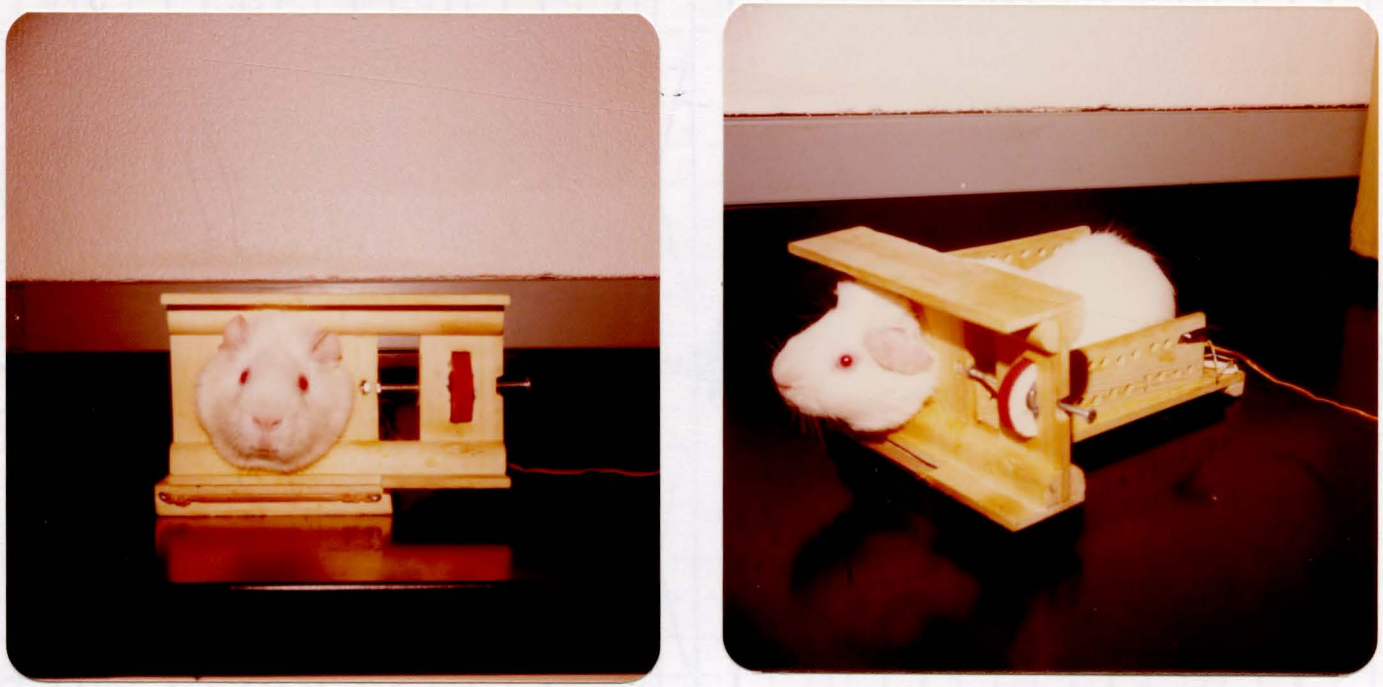

Figure 2. Two views of a guinea pig in the restraining device.

Figure 3. Interruption in the regular shiver pattern due to a 0 db sound.

RESULTS

The results are presented in Table 1. The temporary threshold shift measured from two to sixty minutes after exposure to the high speed dental drill noise for each animal can be found in the Appendix (Tables A1-A6).

Guinea pigs 1 and 2 were each exposed to continuous high speed dental drill noise for 2, 4, 6 and 8 hours. In each test the animal was engaged in a regular shiver pattern until the end of the noise exposure period. However, the moment the noise exposure ceased the animals stopped all movement and did not resume a regular shiver pattern until 2 min. after exposure. In two instances the shiver pattern did not begin again until 5 min. after exposure (guinea pig 1, 2 hr. exposure and guinea pig 2, 4 hr. exposure). Therefore, the earliest a temporary threshold shift could be tested for was 2 min. after the exposure period ended, except in the two cases mentioned above, when testing began 5 min. after the exposure period ended.

Guinea pig 1 did not show a temporary threshold shift after 2 or 4 hours of exposure to the high speed dental drill noise. After 6 hours of exposure, though, the $TTS_{2\ min.}$ was 10 db and the $TTS_{5\ min.}$ was 5 db. By 10 minutes after exposure, no TTS was observed. After 8 hours of exposure the $TTS_{2\ min.}$ was 10 db, the $TTS_{5\ min.}$ was also 10 db, the $TTS_{10\ min.}$ was 5 db, and by 15 min. after exposure no TTS was recorded.

Guinea pig 2 did not show a $TTS_{2\ min.}$ after 2, 4, 6 or 8 hours of exposure to high speed dental drill noise. However, after the 4 hour

exposure, the $TTS_{10\ min.}$ and $TTS_{15\ min.}$ were both 5 db. These were the only two instances of TTS observed for this animal.

Guinea pigs 3 and 4 were exposed to intermittent (30 sec. on/ 30 sec. off) high speed dental drill noise for 2, 4, 6 and 8 hours. The only TTS observed was in guinea pig 3. After 8 hours of exposure its $TTS_{2\ min.}$ was 10 db. This recovered to 0 db by 5 min. after exposure.

The two control animals, guinea pigs 5 and 6, were subjected to the same procedure as the experimental animals, the only difference being in that they were not exposed to any high speed dental drill noise. They did not show a threshold shift for any exposure period.

In all animals the hearing level was checked before each exposure to noise and was found to be 0 db in each case.

Table 1. Temporary threshold shift after high speed dental drill exposure.

Time After Exposure (minutes)	Noise Character-istic	Animal Number	Threshold Shift (decibels) After "X" Hours of Exposure			
			Hours of Exposure 2	4	6	8
2	continuous	1	*	0	10	10
2	continuous	2	0	*	0	0
2	intermittent	3	0	0	0	10
2	intermittent	4	0	0	0	0
2	control	5	0	0	0	0
2	control	6	0	0	0	0
5	continuous	1	0	0	5	10
5	continuous	2	0	0	0	0
5	intermittent	3	0	0	0	0
5	intermittent	4	0	0	0	0
5	control	5	0	0	0	0
5	control	6	0	0	0	0
10	continuous	1	0	0	0	5
10	continuous	2	0	5	0	0
10	intermittent	3	0	0	0	0
10	intermittent	4	0	0	0	0
10	control	5	0	0	0	0
10	control	6	0	0	0	0
15	continuous	1	0	0	0	0
15	continuous	2	0	5	0	0
15	intermittent	3	0	0	0	0
15	intermittent	4	0	0	0	0
15	control	5	0	0	0	0
15	control	6	0	0	0	0
20	continuous	1	0	0	0	0
20	continuous	2	0	0	0	0
20	intermittent	3	0	0	0	0
20	intermittent	4	0	0	0	0
20	control	5	0	0	0	0
20	control	6	0	0	0	0

* The moment the noise exposure ceased the animal stopped all movement and did not resume a regular shiver pattern until 5 minutes after exposure.

DISCUSSION

It should be noted that although a TTS was not observed in many of the experimental animals, or that the TTS was of small magnitude or duration, one cannot conclude necessarily that a TTS did not occur or that those that did occur were not of greater magnitude or duration. The limit of sound level produced by the audiometer is 0 db and, therefore, the limit of testing for a TTS was 0 db. It is important to note that the decibel is actually a dimensionless number. It is merely a ratio. If sound pressures are expressed in decibels, as they usually are, this requires that a standard reference pressure be used. A standard now widely accepted is 0.0002 dyne/cm^2. This pressure was adopted because it approximates the least pressure required for the average human listener to hear a 1,000 cps tone. Zero decibels does not mean that there was no sound. It simply means that the observed and reference values were equal because the log of one is zero. The fact that a sound of 0 db may be inaudible is irrelevant and does not erase the more compelling fact that sound energy was still present.

The reference value used to calculate decibels and, thus, to calibrate audiometers, is based on the hearing of humans. Albino guinea pigs, however, on the average have more acute hearing than humans. Crifo (1973), using shiver audiometry, measured the auditory threshold of 31 albino guinea pigs. At 6,000 cps the average auditory threshold was -7.1 db with a standard deviation of ± 1.3 db. Consequently, a threshold shift

may have taken place but was not detected due to the limits of the equipment used. In addition, the animals were only tested for a TTS at 6,000 cps although they were exposed to a noise that probably had a range of several kilocycles on either side of 6,000 cps. They may have experienced temporary threshold shifts of greater magnitude or duration at other than the test frequency.

Recovery from the TTS produced by the continuous high speed dental drill noise in animal 1 seemed to depend on the length of exposure. The TTS produced after 6 hours recovered more quickly than the TTS produced after 8 hours. Furthermore, the TTS produced after 8 hours of intermittent exposure observed in animal 3 recovered faster than the TTS in animal 1 after continuous exposure of the same duration.

After 4 hours of exposure to continuous high speed dental drill noise, guinea pig 2 had a TTS of 5 db at 10 and 15 min. after exposure. The TTS measured before and after the 10 and 15 min. tests were 0 db. Although this may have been due to an inconsistency in response of the animal at these times, it is not unlike the phenomenon observed by Hamernik and Henderson (1974), Sitler (1972) and Luz (1971). In evoked response audiometry tests a consistent finding has been the growth of a TTS to a maximum as much as 14 hours after exposure before recovery begins. They offer no explanation for this observation.

Davis, et al., (1958), found that rest periods between exposures were significant in reducing temporary threshold shift, with the amount of reduction depending on individual susceptibility. Although guinea pigs

3 and 4, exposed to intermittent noise, had, overall, less TTS than animals 1 and 2, it is interesting to note that the $TTS_{2\ min.}$ after 8 hours of intermittent exposure in guinea pig 3 is greater (10 db) than the $TTS_{2\ min.}$ after 8 hours of continuous exposure in guinea pig 2 (0 db).

In order to predict the effects of intermittent broadband sound, Ward (1959) developed the formula

$$TTS_2 = 1.06R(S-91)(\log_{10}T/1.7),$$

where R is the "on" fraction, S the overall sound pressure level of the noise, and T the exposure duration in minutes, including the off periods. Based on this formula, the expected $TTS_{2\ min.}$ for 2, 4, 6 and 8 hours of intermittent exposure to a 92 db sound would be, respectively, 0.98 db, 1.14 db, 1.27 db and 1.30 db. In all cases except guinea pig 3 after 8 hours of exposure ($TTS_{2\ min.}$ of 10 db) the experimental value (0 db) was very near the predicted value. One shortcoming of Ward's formula is that it does not take into account the frequency component of the noise.

In both the continuous and intermittent test groups, one animal suffered a TTS while the other animal did not. This is consistent with the findings of Sockwell (1971), Robin (1960) and Burns (1973), that hearing loss depends, in part, on individual susceptibility to noise exposures.

Only temporary, not permanent, threshold shifts were observed. These may have been due to exhaustion of enzymes and glycogen stores, diminished oxygen tension, decreased energy output, and reversible alterations in organelles of the sensory cells and nerve endings. More intense stimulation probably would have resulted in irreversible morphological

alterations and permanent hearing loss according to Schuknecht (1974).

Although only temporary threshold shifts were observed due to exposure to high speed dental drill noise, they may not appear so innocuous if the findings of Glorig (1962) are correct. He found that at 4,000 cps, the $TTS_{2\ min.}$ after exposure to a steady noise for 8 hours was equal to the permanent threshold shift after 10 years of working in the same industrial noise environment. Ward (1965) and Kryter (1966) reached similar conclusions.

The number of animals used in this experiment was far too small to draw general conclusions from the results. However, of the animals tested, one conclusion can be made. High speed dental drill noise of sufficient intensity and duration, either continuous or intermittent, can cause a small but perceptible temporary auditory threshold shift in certain susceptible albino guinea pigs.

This experiment was conducted using guinea pigs as subjects and it is, therefore, difficult if not unreasonable to conclude that high speed dental drill noise may cause a temporary threshold shift in dentists. However, the results of this experiment when viewed in the light of those experiments mentioned in the literature review seem to indicate that the high speed dental drill does not pose the threat to the hearing of dentists as was initially postulated when high speed dental drills were first introduced in the dental marketplace. This conclusion is based primarily on the facts that the high speed dental drills in use today are quieter than when first introduced, that the drills are used for relatively short

periods of time with long rest periods in between, and that most dentists are exposed to louder and/or more prolonged exposures to noise in other activities in their lives which would more likely be the cause of hearing loss (if any) than high speed dental drill noise.

Although it seems unlikely, future experiments may show that high speed dental drill noise can cause a temporary auditory threshold shift in dentists. As Miller (1974) concludes, "In spite of the efforts in many laboratories, the laws of temporary threshold shifts have not yet been completely determined. There are large numbers of variables that need to be explored."

SUMMARY

Six male albino guinea pigs were used to determine the magnitude, duration and course of recovery from a temporary auditory threshold shift caused by exposure to high speed dental drill noise. Shiver Audiometry, a method first developed by Anderson and Wedenburg (1965) to detect hearing thresholds in guinea pigs, was used. Certain modifications, however, were made for this experiment. Two guinea pigs were exposed to continuous sound, two to intermittent sound and two served as controls. Exposure times were two, four, six and eight hours. The results indicate that high speed dental drill noise of sufficient intensity and duration, either continuous or intermittent, can cause a small but perceptible temporary auditory threshold shift in certain susceptible albino guinea pigs.

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APPENDIX

Appendix Table 1. Change in threshold expressed as decibels at various time intervals following cessation of sound.

Guinea Pig 1, continuous high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes)								
	2	5	10	15	20	25	30	45	60
2	*	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0
6	10	5	0	0	0	0	0	0	0
8	10	10	5	0	0	0	0	0	0

Threshold Shift (decibels)

* The moment the noise exposure ceased the animal stopped all movement and did not resume a regular shiver pattern until 5 min. after exposure.

Table A2. Guinea Pig 2, continuous high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes) 2	5	10	15	20	25	30	45	60
2	0	0	0	0	0	0	0	0	0
4	*	0	5	5	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0	0

Threshold Shift (decibels)

* The moment the noise exposure ceased the animal stopped all movement and did not resume a regular shiver pattern until 5 min. after exposure

Table A3. Guinea Pig 3, intermittent high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes)								
	2	5	10	15	20	25	30	99	60
2	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0
8	10	0	0	0	0	0	0	0	0

Threshold Shift (decibels)

Table A4. Guinea Pig 4, intermittent high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes)								
	2	5	10	15	20	25	30	45	60
2	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0	0

Threshold Shift (decibels)

Table A5. Guinea Pig 5, control animal, no high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes) 2	5	10	15	20	25	30	45	60
2	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0	0

Threshold Shift (decibels)

Table A6. Guinea Pig 6, control animal, no high speed dental drill noise.

Time of Exposure (hours)	Time After Exposure (minutes) 2	5	10	20	25	30	45	60
2	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0

Threshold Shift (decibels)

APPROVAL SHEET

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The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science.

8/17/79
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