AMERICAN ACADEMY OF PEDIATRICS

Committee on Environmental Hazards

Noise Pollution: Neonatal Aspects

The deafening effect of high intensity noise is well known—from rock music, aircraft, snowmobiles, motorcycles and the shooting of guns. The effects of hospital noise and its interaction with ototoxic drugs are less well known. The subject is of particular importance to pediatricians, because infants in incubators are exposed to substantial noise from the motor, airflow, respirators, slamming of incubator doors and the baby’s own crying. Furthermore, animal experimentation shows that the ototoxic drug, kanamycin (often given to the premature infant to combat sepsis), can potentiate the effect of noise on hearing loss as much as 100-fold. Whether or not an interaction between noise and potentially ototoxic drugs occurs in man is as yet unknown.

MEASUREMENT

Noise has frequency and intensity. Frequency is measured in cycles per second, designated hertz (Hz). The young human ear is sensitive to a frequency range of 20 to 20,000 Hz. White noise, the auditory counterpart of white light, has equal energy in each frequency in the audible range.

Intensity is measured in decibels on a scale which is linear with respect to audible frequencies. This measurement is designated dB (linear). Since the human ear is more sensitive to the damaging effects of high frequency sound than to low frequency, a better correlate with noise-induced hearing loss can be obtained when low frequencies are filtered out. Filtered sound level, measured on a so-called A-weighted scale, is designated dB(A). Room conversation produces 60 to 70 dB(A), rock music 100 to 120 dB(A) and snowmobiles 105 to 135 dB(A) for the driver. The sound pressure level from a source of noise is inversely proportional to the square of the distance from the source.

NOISE-INDUCED HEARING LOSS

Excessive noise exposure produces deafness by damaging the organ of Corti, especially the outer and inner hair cells. In adults, approximately 80 dB(A) is the maximal sound intensity which does not produce sensorineural hearing loss regardless of duration. At slightly higher levels, a small percentage of people will develop impaired hearing. The percentage will increase with increased intensity of exposure. The industrial standard, according to the Walsh-Healy Act, permits 90 dB(A) exposures for an eight-hour day. This limit would be reduced to four hours if the intensity of sound energy were doubled, as occurs with an increase of 3 dB(A), say from 90 to 93 dB(A). The law allows exposure to 105 to 110 dB(A) for a few minutes. The human ear is more sensitive to the damaging effects of high frequency sound than to low frequency. The hearing loss of industrial workers, who are mainly exposed to noise of a wide frequency range, centers at 4,000 Hz. Permanent hearing loss at lower frequencies is produced by longer exposures.

OTOTOXIC DRUGS

Ototoxic drugs fall into four main groups: salicylates and quinine, potent diuretics (ethacrynic acid and furosemide), certain antineoplastic drugs, and aminoglycosidic antibiotics. The last includes streptomycin, kanamycin, neomycin, gentamicin and tobramycin. Initially these antibiotics destroy hair cells in the basal turns of the organ of Corti and produce a high-frequency permanent sensorineural hearing loss (4,000 to 8,000 Hz). As the ototoxicity develops, hair cells in the apical turns are damaged, and hearing loss involves the speech frequencies (500 to 2,000 Hz). Early signs of ototoxicity can be detected by sequential audiometric examinations in adults, but not in newborn infants. The development of ototoxicity is directly related (1) to the dosage of the drug—both total dosage and average daily dose, (2) to length of treatment, and (3) to the presence of impaired renal function.

The risk of ototoxicity is low following the parenteral administration of aminoglycosidic antibi-
otics to infants and children with normal renal function. In one study, only two of 22 infants and children treated with kanamycin and isoniazid developed hearing loss, which may have been due to tuberculous meningitis. Another study suggested that larger doses of kanamycin, especially when renal function is impaired, produced high-frequency sensorineural hearing loss similar to that observed in adults—the effect being related to the total dose administered. Eichenwald has shown in a four-year prospective study that premature and full-term infants suffered no increased hearing loss as compared with matched controls when treated with either kanamycin at 15 mg/kg/day intramuscularly for six to ten days, or streptomycin at 40 mg/kg/day intramuscularly for six to 12 days. These observations are reassuring, but do not exclude the possibility of hearing loss from greater doses, other aminoglycosidic antibiotics, or associated disorders, such as renal impairment.

POTENTIATION BETWEEN NOISE AND OTOTOXIC DRUGS

The potentiation of noise-induced hearing loss by the ototoxic aminoglycosidic antibiotics has been established in animal studies; i.e., cellular damage in the organ of Corti after the combined exposure to noise and antibiotics greatly exceeds that expected from adding the separate effects of each agent.

Guinea pigs placed in incubators and exposed to 58 dB(A) and given kanamycin for five weeks at 15 or 50 mg/kg/day sustained permanent pathologic changes in the cochlea. In another study, simultaneous exposure to 250 mg/kg/day of kanamycin for eight days and noise at 90 dB(linear) caused the loss of 47% of hair cells in the cochlea, as precisely determined by phase contrast microscopy. In both studies, each agent alone had little effect. A potentiating ototoxic effect of neomycin and noise has also been demonstrated by pathological and electrophysiological studies. These studies showed that the administration of ototoxic drugs produces an increased susceptibility to noise damage. In experimental animals, levels of noise as low as 58 dB(A), in themselves benign, potentiated the risk of hearing loss from known ototoxic drugs.

To date only two aminoglycosidic antibiotics, kanamycin and neomycin, have been shown in experimental animals to potentiate the ototoxicity of low levels of noise. There is a need to evaluate interactions between noise and other aminoglycosides such as streptomycin, gentamicin, and tobramycin as well as the ototoxic diuretics. Pathologic studies in guinea pigs exposed to sodium salicylate and noise in combination have not shown a major potentiative effect, but in man the possibility of hearing loss cannot be ruled out.

AGE-DEPENDENT SUSCEPTIBILITY TO NOISE-INDUCED HEARING LOSS

Several recent studies present evidence indicating greater susceptibility of young animals to noise-induced physiological and pathological alterations. Newborn guinea pigs are more susceptible to high intensity noise exposure (120 dB(linear) of white noise for 30 hours), as measured by pathological changes in the organ of Corti when compared with adult guinea pigs. Electrophysiologic experiments have demonstrated an increased susceptibility to noise damage in 8- to 12-week old kittens compared with adult cats after a 50-minute exposure to 5,000 Hz. The incidence of new cases of noise-induced hearing loss in two groups of workers, 18-29 years and 30-45 years of age, respectively, both exposed to industrial noise was three times higher in the younger group after three years’ exposure to the same noise parameters.

HOSPITAL NOISE

In two studies, infant incubators produced continuous noise levels of between 50 and 86 dB (linear). Oxygen inlets produced an additional 2 dB(linear). Slamming of incubator doors and infant crying produced 90 to 100 dB(A). The mean noise levels measured in the center of a surgical recovery room were 57.2 dB(A) (69.8 dB(linear)), while those measured at the patients’ heads were 65.6 dB(A) (80.0 dB(linear)).

Auditory Effects

These noise levels are not currently considered a risk to hearing because they are below 80 dB(A). However, industrial criteria are not applicable to hospitalized infants. The criteria are based on intermittent exposures of eight hours per day, not continuous noise, as in hospitals. More damage is done by continuous noise because there is no opportunity for recovery. Infants may well be more susceptible than adults to noise-induced hearing loss. Infants in incubators are often treated with potentially ototoxic drugs and may thus be at high risk of developing deafness. The incubators produce noise levels which in animal studies cause a large potentiation of cochlear damage in the presence of kanamycin. Hospital noise, including incubator noise, must be considered as a possible cause of childhood deafness.

Endocrine and Cardiovascular Responses

Noise, in common with other stresses, increases the secretion of ACTH, with measurable increases
seen at levels as low as 68 dB(linear). Changes in heart rate and peripheral vasoconstriction occur at levels as low as 70 dB(linear). Noise in incubators and recovery rooms exceeds these thresholds.

Effects on Speech and Language Development

It has been suggested that incubator noise impedes language stimulation to the infant by masking, attenuating, or distorting sounds. The hypothesis that long-term exposure impairs speech and language development needs testing because of its importance.

Effects on Sleep

One study has been made of the waking-noise thresholds of infants exposed to 50 to 80 dB(linear) in the range of 100 to 7,000 Hz. A level of 70 to 75 dB(linear) for three minutes led to obvious disturbance or awakening in two thirds of the children. All infants awakened after 75 dB(linear) for 12 minutes.

Reduction in Noise

Noise can be reduced either at the source or at the receptor, i.e., by occlusive devices. Efforts should be directed at reducing noise at the source, because use of occlusive devices may have unforeseen adverse effects, e.g., local reaction or sensory deprivation with delayed speech and language development.

SUGGESTIONS FOR THE FUTURE

1. Criteria have not yet been established to protect infants from the effects of noise. In the meanwhile, on the basis of animal experimentation which shows that noise markedly potentiates the ototoxicity of kanamycin, it would be prudent (1) for manufacturers of incubators to reduce the noise from motors as much as possible, preferably below 58 dB(A), a level which potentiates the ototoxicity of kanamycin in experimental animals; (2) for physicians to limit the use of aminoglycoside antibiotics and other potential ototoxic drugs in neonates insofar as is consistent with good medical practice; and (3) for hospital personnel to eliminate unnecessary noise, including radios played at high volume in the premature nursery.

2. To establish criteria for protecting infants from the effects of noise, additional information is needed on (1) the relation between disorders of hearing, speech or language and a past history of exposure to incubator noise and/or ototoxic drugs (i.e., retrospective studies); and (2) the relation of such exposures to subsequent hearing or speech impairment (i.e., prospective studies).

3. Physicians should be alert for unnecessary noise in nurseries (or use continuous sound level recorders when feasible), and take appropriate action for abatement.

This report was prepared by Stephen A. Falk, M.D., of the National Institute of Environmental Health Sciences in consultation with the Committee on Environmental Hazards.

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Committee on Radiology

Introduction of Newer Modalities in Pediatric Radiology

Many new and exciting diagnostic modalities are being introduced into radiology, and some of them are beginning to have an impact on pediatric practice. Most of these modalities are noninvasive techniques which can be performed with ease and safety in the pediatric patient. For example, the application of axial tomography with computer integration of the radiologic image (EMI Scanner, ACTA Scanner) may revolutionize the practice of neuroradiology as we now know it. Nuclear medicine is only beginning to blossom in its many physiologic and morphologic applications, particularly with the introduction of Gamma Cameras and short half-lived scanning agents associated with minimal radiation dose. The use of ultrasonography is becoming particularly important in the evaluation of acquired and congenital heart disease and in the evaluation of abdominal masses. Thermography and xeroradiography have yet to find a significant use in pediatric practice. Fine tuning and modification of diagnostic radiologic procedures used routinely in adult medicine have made the application of such modalities as angiography relatively safe in skilled hands when they are used for children.

These newer diagnostic modalities must be subjected to the same critical analyses regarding efficacy, cost, and safety that currently are demanded and accepted for the introduction of new drugs and other forms of therapy. They will have to be evaluated to establish whether or not they are simply duplications of more conventional methods of diagnosis and fail to improve patient care. Publication of their availability and use, and even advertisements, should be considered carefully before they are accepted for application in pediatrics. Careful evaluation by the radiologist and the practitioner will help to avoid trouble.

In subsequent articles in Pediatrics, the Committee on Radiology will review some of these new modalities and refinements in diagnostic radiology to provide a critical background for their effective introduction into pediatric practice.

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