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PRENATAL EFFECTS OF EXPOSURE TO HIGH-LEVEL NOISE

Report of Working Group 85

Committee on Hearing, Bioacoustics, and Biomechanics
Assembly of Behavioral and Social Sciences
National Research Council

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DEDICATION

This report is fittingly dedicated to the memory of

HARLOW ADES

chairman of the Working Group on Prenatal Effects of Exposure to High-Level Noise, whose untimely death in October 1977 was a great lost to us all. Dr. Ades, a professor of anatomy at the University of Illinois, provided the framework for the report and his influence is also strongly reflected in the recommendations.

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LIMITATIONS OF RESEARCH AND ITS EVALUATION

Contrary to popular belief, a specific noxious environmental agent does not usually produce specific, characteristic fetal injury. The actual expression of fetal injury caused by exposure to a harmful agent depends on six major variables:

- The genotype of the species and perhaps of the individual;
- The developmental stage (gestational age) at which the exposure occurs;
- The mechanism through which the agent reaches the fetus (direct or indirect);
- The nature of the noxious agent;
- The synergism of the agent with other factors in the environment; and
- The age of the mother.

The genotype is an unknown factor beyond our control. Investigators of the effects of noise must assume that subjects in control and experimental groups are equally susceptible to injury.

In the case of exposure to high-intensity noise, the mechanisms through which the noxious sound may act on the fetus are the direct effects of noise on the developing fetus and the direct effects of noise on the mother with indirect neuroendocrine effects on the fetus. Direct and indirect effects must be separated if their palliation is to be achieved. For example, if the effect of noise on the fetus is direct, ear protection for the mother may protect her, but not the fetus; if the effect of noise on the fetus is indirect, through alterations in the mother's neuroendocrine balance, ear protection for the mother may protect the fetus as well. The concept of a teratogen or a noxious agent as a maternal or fetal stressor raises the issue of interaction between maternal and fetal endocrine glands and their target tissues.

Evaluation of developmental disturbances in human infants is usually limited to gross inspection and analysis of the disturbance may be possible only at surgery or even at autopsy. A number of behavioral disturbances and morphological defects are not manifest until later in childhood. In small groups, genetically determined defects, spontaneous, idiopathic defects, and defects caused by unsuspected or uncontrolled environmental factors may seriously compromise the results of both prospective and retrospective studies. Pedigrees should be obtained to eliminate the genetic defects of morphology and behavior.

In research on the effects of noise exposure, both control and experimental populations are also necessarily exposed to noxious agents other than noise. The effects of medication, drugs, air pollution, industrial toxins, and maternal diet should be evaluated and either eliminated or matched in the control populations. Recent evidence linking smoking with low birth weight, prematurity, and malformation should be considered as well (Meyer and Tonascia, 1977; Naeye, 1978).

Spontaneous defects can be expected to appear in both experimental and control populations on the order of 1-3 per 100 live births, depending on the thoroughness of the examination and on the racial mixture and the socioeconomic status of the populations. If we consider only possible fetal injury to auditory function we find a natural incidence of deafness of 1 per 1000 live births in the United States (World Health Organization, 1972:Sec. 497). Of known teratogens affecting the fetal ear the best documented are maternal syphilis, maternal rubella, maternal toxoplasmosis, and maternal ingestion of thalidomide (see Bergsma, 1973, for references). Other substances suspected of causing congenital deafness are quinine, salicylates, and aminopterin. Streptomycin and related drugs may produce auditory injury at any age; some people have a familial sensitivity to these drugs (Kern, 1962).

None of these teratogens is specific for injury to the ear, but each may produce such injury. The fetal ear is, of course, no more vulnerable to injury than are other fetal organs; it appears to be rather less so than many organs that develop earlier in embryonic life. For any organ or organ system, a similar set of circumstances for fetal injury can be described.

REVIEW OF RESEARCH

Most of the research reports reviewed here were selected for their recency, adequacy of research methods and instruments, and reasonable description of experimental and control subjects, if the latter were included. Except as otherwise noted, the studies used human subjects.

Prenatal Development of the Inner Ear and the Auditory Nervous System in Humans

Bredberg (1967) describes the results of his own study of the development of the inner ear in the human fetus and gives a brief summary of the results of earlier studies. The earliest investigations date back to the 1920s; standard light microscopy was used in examining cross-sections of cochlear specimens. Bredberg made use of light/phase contrast microscopy with surface preparations in order to examine the whole length of the organ of Corti from base to apex. In agreement with the results of earlier investigations (Bast and Anson, 1949; Ormerod, 1960), Bredberg found in the fetus that "full differentiation of the organ of Corti occurs at the age of six months." We know from anatomical data, then, that at six months after conception the sensory endorgan of hearing is apparently mature. Data cited below from electrophysiological recordings indicate that it is also functional at approximately this age.

Measurement of Sound Attenuation

Several reports have been made of attempts to measure sound pressure levels near the head of the fetus when external sounds or vibrations stimulate the abdomen of the pregnant mother. The sonic environment of the fetus is dependent on the attenuation of external sounds by the mother's abdomen, the uterine wall, and the amniotic fluid in which the fetus floats and by the intrinsic sound primarily from the mother's circulatory system.

Bench (1968) made measurements of one pregnant woman at 37 weeks of gestation. External sounds -- pure tones of 200, 500, 1000, 2000, and 4000 Hz -- were delivered through an air-coupled loudspeaker held in a foam rubber annulus so that the loudspeaker diaphragm was one-half inch from the abdominal wall. The loudspeaker was positioned over the fetal head. Intrauterine sound pressure levels were measured using a crystal microphone enclosed in a thin rubber membrane and placed within the vagina against the external cervical os. With the external stimulus level at 120 dB (re 0.0002 microbar), the following attenuations of sound pressure were measured: 19 dB at 200 Hz; 25 dB at 500 Hz; 38 dB at 1000 Hz; 48 dB or greater at 2000 and 4000 Hz. Internal background noise level, probably due to the mother's cardiovascular system, was approximately 72 dB.

Grimwade et al. (1970) measured the attenuation of sounds through the abdominal walls of nine pregnant patients "by placing a source of pure tone frequency on the abdominal wall and recording the intrauterine intensity." Intrauterine sound pressure levels were measured by a one-quarter-inch condenser microphone (Bruel and Kjaer Type 4136) that was passed "through the cervical canal to lie beside the head of

the fetus in the lower uterine segment. The microphone was connected to a sound level meter and frequency analyser (Bruel and Kjaer, Type 2107)." For pure tone stimuli the following attenuations of sound pressure were measured: 39 dB at 500 Hz; 40 dB at 1000 Hz; 52 dB at 2000 Hz; 78 dB at 4000 Hz and 85 dB at 5000 Hz. Background sound levels had a mean value of 85 dB with peaks at 95 dB occurring in time with the mother's pulse. The background sound energy was in the range of 20-300 Hz. Corrections were made for measurements of attenuation in a later report by Walker et al. (1971). They pointed out that errors in the original measurements were introduced for two reasons: "(1) There is an impedance mismatch across each of the fluid/rubber/air interfaces between the amniotic fluid and the diaphragm of the microphone; (2) the microphone is designed to operate with normal barometric pressures behind the condenser diaphragm, which in this case is sealed by a rubber membrane." The corrected values for attenuation of pure tones applied to the abdomen were: 20 dB at 50 Hz; 22 dB at 100 Hz; 25 dB at 200 Hz; 30 dB at 500 Hz; 37 dB at 1000 Hz; 70 dB and greater at 4000 Hz and higher frequencies.

Making certain assumptions about the sound transmission characteristics of the human torso, Westervelt has calculated the attenuation of external sounds by the bodies of pregnant women (see appendix). His calculated results are compared with the measured results reported by Walker et al. (1971) in Figure A-1 of the appendix.

Responses of Fetus to Sounds External to the Mother

In order to show that the fetus is directly stimulated by environmental sounds external to the mother, a number of recording methods have been used, namely, fetal movements; changes in heart rate; and changes in electroencephalogram (EEG) -- in particular, the computer-averaged responses to repeated sound pulses.

Fetal Movements

Sontag and Wallace (1934; 1936), Sontag and Richards (1938), and Sontag (1944) recorded fetal movements elicited by vibratory stimuli (120 Hz) applied directly to the abdomens of pregnant women. In a study primarily concerned with changes in heart rate elicited by tonal stimuli transmitted through the air from a loudspeaker to the mother's abdomen, Bernard and Sontag (1947) mention that the stimuli elicit "sharp body movements." In their study of heart rate changes they noted distinct fetal movements in response to tonal stimuli.

Changes in Fetal Heart Rate

Sontag and Wallace (1934; 1936), Sontag and Richards (1938), and Sontag (1944) reported an increase in fetal heart rate in addition to fetal

movements when a vibrating stimulus (120 Hz) was applied directly to the abdomens of pregnant women.

Bernard and Sontag (1947) found that the fetal heart rate accelerated after exposure of the fetus to tonal stimulation. They used tonal stimuli brought to the mother's abdomen by a loudspeaker mounted on a rubber baffle so that there was an air space between the loudspeaker and the abdominal wall. The loudspeaker was placed over the region of the head of the fetus. Heart sounds were picked up by a "microphone strapped to the maternal abdomen at the point where the sound was loudest." The sounds were amplified and recorded by means of an ink writer on a moving tape.

Murphy and Smyth (1962) recorded an increase in fetal heart rate elicited by stimulus tones of 500 and 4000 Hz at a level of 100 dB (re. .0002 dynes/cm^2).

Dwornicka et al. (1963) reported acceleration of the heart rate of the fetuses of 32 pregnant women in the last month of pregnancy. The stimuli were tones of 1000 or 2000 Hz, of 5 second's duration at an intensity of 100 dB,[1] measured at the transducer placed on the mother's abdomen. Heart rate was measured by phonocardiograph. The mother's pulse rate was recorded and was found to remain constant. The latencies of the fetal heart changes were as short as one second after tone onset, ruling out the likelihood that the fetal responses resulted from some reaction of the mother. In two cases, a deceleration in the fetal heart rate was noted, followed by acceleration.

Johansson et al. (1964) recorded changes in the fetal heart rate elicited by a tone of 3000 Hz at approximately 110 dB sound pressure level (SPL). The sound pressure level was measured by an intrauterine microphone, and the sound was delivered through a vibrator placed on the mother's abdomen. The 3000 Hz tone was used to avoid tactile stimulation. The heart rate changes were recorded by phonocardiograph. In 45 tests on 10 fetuses, 35 tests showed a significant increase in heart rate. Fetal movements in response to the tonal stimuli were also observed. In a second series of experiments, the reactions of the fetuses of 15 women were examined using tonal stimuli of 500, 1000, 2000, and 3000 Hz at an intensity level of 110 dB. No reactions were recorded at 22 weeks -- the earliest age at which fetuses were tested. Definite responses were seen at 26 weeks, and the responses became stronger with increase in age of the fetuses. In both studies, postnatal hearing tests indicated that the children had normal hearing.

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[1] No reference level was given. It is assumed to be 0.0002 microbar, or 0.0002 dynes/cm^2. It is assumed to be the reference level in the remainder of this report when authors give sound pressure levels in dB SPL.

Bench and Mittler (1967) reported increases in the fetal heart rate elicited by a 500 Hz tonal stimulus, at 105 dB (re. 0.0002 micobar), delivered through a loudspeaker air-coupled to the mother's abdomen. In a later report, Bench and Vass (1970) questioned the validity of the earlier studies, both their own and those of others, in which evidence of a change in the fetal heart rate in response to sound stimuli was reported.

Grimwade et al. (1970) used both vibrators and sound stimuli to record the fetal heart rate in 32 patients in labor. Intrauterine levels of 70-90 dB SPL were measured in response to tonal stimuli from either an external loudspeaker or an intrauterine crystal earphone. A mechanical vibrator and clinical percussion were also used as stimuli. Of 330 applications of sound or vibratory stimuli, changes in fetal heart rate occurred in 41 percent. Vibratory stimuli were more effective than sound stimuli.

Despite the criticism of Bench and Vass, the overall evidence, in view of the controls used in these studies reporting changes in the fetal heart rate, favors the view that changes do occur in response to high-intensity vibratory or sound stimuli applied to the abdomen of the mother. The changes are neither large nor consistent. There is no evidence that the changes are injurious in themselves or that they represent injury elsewhere in the fetus.

Evoked Responses to Acoustic Stimuli

Electroencephalograms (EEGs) of the human fetus have been recorded by a number of investigators (e.g., Lindsley, 1942; Bernstine et al., 1955; Dreyfus-Brisac et al., 1956; Rosen and Satran, 1965). With the development of the method of averaging the EEG response evoked by sensory stimuli, it is now possible to record the EEG activtity elicited by an acoustic signal.

Barden et al. (1968) recorded the evoked responses to tonal stimuli in six fetal subjects at term gestation. The mothers, all of whom had normal pregnancies, volunteered for the inducement of labor and for the electronic monitoring procedure. Fetal heart rate and EEG "were recorded from a skin-clip electrode attached to the presenting vertex of each subject." An intrauterine transducer to deliver the acoustic stimuli, an intrauterine microphone to measure stimulus intensity, and the EEG recording electrode were placed on or near the head of the fetus after artifical rupture of the membranes surrounding the fetus. A tonal stimulus of 1000 Hz at a sound pressure of 88-105 dB and 450 milliseconds (msec.) in duration was presented 110 times, and the evoked responses were averaged by a computer (CAT-model 400B). In one subject, an average evoked response was successfully recorded at a sound pressure level of 88 dB: it consisted of a positive wave with a peak latency from signal onset of 75 msec., a negative wave with a latency of 100 msec., and a sharp peak component that appeared at

about the end of the stimulus. No clear evoked responses could be recorded from the other 5 subjects; the investigators attribute this failure to the use of an oxytocin infusion in order to effect labor progress. Oxytocin infusion was not used in the subject from whom evoked responses were successfully recorded. No changes in the heart rate of the fetuses or of the mothers were seen.

Sakabe et al. (1969) have reported recording typical evoked responses to acoustic stimuli in six fetuses ranging in age from 32 to 38 weeks. Recording electrodes were placed on the median line of the abdominal wall of the mothers. The acoustic stimuli were tone bursts of 1000 Hz, 50 msec. in duration, presented every 4 seconds through a bone conduction vibrator. The number of presentations ranged from 80 to 1000. The averaged evoked responses consisted of a negative wave with a peak latency of 100-150 msec. as measured from stimulus onset; a positive deflection with a latency of 200-300 msec.; a negative deflection with a latency of 500-600 msec.; and a positive deflection with a latency of 700-800 msec. The 200-300 msec. and 500-600 msec. waves were most clearly defined. Similar evoked responses were recorded from an infant born prematurely during the 37th week of pregnancy. Weitzman and Graziani (1968) have also described similar averaged responses to auditory stimuli in prematurely born infants.

Effects of Prenatal Noise Exposure as Evidenced at Birth or Later

Human Studies

In order to assess the hazards of prenatal noise exposure or to set acceptable limits of such exposure, the most useful data would come from studies that compare children born to mothers who have been repeatedly exposed to high-level noise during pregnancy with children born to mothers who have lived in a "normal" noise environment. There is, unfortunately, very little of such evidence, and what is available is difficult to evaluate because of the small sample populations, inadequate control populations, and limited information as to actual noise exposures.

Rehm and Jansen (1978) examined the obstetric records of 1,452 births from 4 hospitals in 3 residential areas with different levels of aircraft noise exposure. They reported some tendency for an increased percentage of premature births in areas with higher levels of aircraft noise, but actual levels and durations of noise exposure were not known and the number of cases in the different areas was too small for adequate analysis of the data.

Ando and Hattori (1970; 1973; 1977a; 1977b) compared children born to mothers living near a major Japanese airport with those born to mothers living in a quieter region. They found that children of

mothers who had moved to the airport area after the fifth month of pregnancy were more disturbed by noise than were children of mothers who had lived there throughout pregnancy. In addition they found an increase in the rate of maternal toxicosis associated with an increase in low birth-weight infants and a lowered level of placental lactogen in the mother's blood.

Jones and Tauscher (1978) examined the records of 2500 white and 2500 black births within the 90 dBA contour of the aircraft landing zone at Los Angeles International Airport and compared the observed birth defects with birth defects in the rest of the country. They found an increase in the number of infants with birth defects among both black and white births in the high noise area. After correcting their data for race and for polydactyly, which is more common in black infants, the statistical significance of the difference due to noise was low.

Edmonds, et al. (1979) examined records of about 7700 births to women living near the Hartsfield International Airport near Atlanta, Georgia, who were subjected to noise over 65 dB L_{dn} (weighted day-night average level). After correcting their data for variations in hospital reporting, socioeconomic status, and race, they found no significant differences in the incidence of 17 types of birth defects.

In each of the above studies, some variables were necessarily uncontrolled. Maternal age, parity, illegitimacy, and especially time spent in areas other than the mother's residence are some examples. Not one of the studies claimed to prove that sound was the cause of the observed effects. Retrospective studies are limited, of course, by the records already in existence. The measured variables are often not exactly those the experimenter would have chosen, and their reliability is usually outside the experimenter's control.

Animal Studies

Numerous studies (Nawrot, et al., 1981; Scibetta and Rosen, 1969) have been reported in which female animals have been exposed to high-level noise during pregnancy. Most of these studies have used small animals as subjects: mouse, rat, hamster, chicken, and guinea pig. No comprehensive review of the results of these studies is given here because it is questionable whether such animals are suitable models for humans. For example, the mother's body offers little protection, prenatal development of the auditory system differs from that of the human fetus, and many of the animals show unusual startle or fright reactions to loud sounds. Nevertheless, a review of experiments using small animals is worthwhile for any investigator undertaking studies with more appropriate animal models; physiological changes that have been noted may suggest changes to be examined in any other mammals studied. James Saunders (personal communication; see also Bock and Saunders, 1977)

has called attention to the finding that in five mammalian species (quantitative data available for the mouse, hamster and rat, less so for the guinea pig and kitten) during the development of the auditory system there is a critical period of heightened susceptibility for severe trauma from noise exposure. "Although the final story is far from complete, it would appear that the critical period occurs during and just after the receptor organ becomes functionally mature. The pathology from noise exposure during this critical period varies somewhat from massive to substantial cochlear damage, but it always appears to be restricted to the outer hair cell system." Saunders adds that the relevance of these animal studies to humans is uncertain. Unlike that of humans, the morphological and functional maturation of the species referred to above, except the guinea pig, does not occur until after birth. "Nevertheless, in these species the ontogenetic sequence of auditory development appears to be no different from that seen in species whose auditory system matures precocially, i.e. during embryonic or fetal life." Saunders concludes that, if there is a similar period of heightened susceptibility to acoustic trauma in humans, it might be expected to occur during the final stages of cochlear development, six to nine weeks before birth.

Measurements made of attenuation of sound by the bodies of pregnant ewes have been reported by Armitage et al. (1980) and by Dunn et al. (1981). Armitage et al. found that "external sounds are attenuated by about 16 to 37 dB, most attenuation occurring at frequencies around 1 kHz." Dunn et al. reported that "the attenuation of an external 130 dB SPL acoustic signal within the uteri of sheep was 15-25 dB in the frequencies below 2000 Hz and 30-40 dB in the frequencies above."

The measurements made by Dunn et al. (1981) were part of a study, Effects on the Auditory System from In Utero Noise Exposure. Of a total of 18 pregnant ewes, 9 were exposed to broadband noise at 130 dB SPL for 4 hours per day, 5 days per week; the other 9 served as controls. Exposures were made during the last five months of pregnancy.

Hearing of the lambs born of both exposed and control mothers was evaluated by recording auditory brain stem responses from scalp electrodes when the lambs were between 30 and 40 days of age. The brain stem responses of both exposed and control lambs were found to be within the range of normal thresholds.

Examination of serial sections of the cochleas of all lambs after sacrifice gave the following results. "Seven of the 18 exposed ears (38%) exhibited bulging Reissner's membrane as is seen in cochlear hydrops, 10 ears (55%) showed misshapen or indistinct supporting cells of the organ of Corti that resembled degenerating cells, and 6 (33%) had anomalies (collapsed or fused) of Reissner's and/or the tectorial membrane. The control ears exhibited the same morphological anomalies, but the frequency and severity of the conditions were less than in the exposed group. The control ears exhibited distended Reissner's membrane

similar to cochlear hydrops in 3 of the 18 ears (17%), anomalies of the supporting cells in the organ of Corti in 4 ears (22%), and anomalies (fused or collapsed) of Reissner's or the tectorial membrane in 4 4 ears (22%).

Dunn et al. concluded that "Even though the exposed ears exhibited almost twice the number of morphological anomalies found in the control group, we are reluctant to interpret these findings as being directly related to the noise exposure since they occurred in both groups. We are inclined to interpret them as being postmortem fixation artifacts. On the other hand, the higher incidence of cellular anomalies in the cochleas of the noise-exposed lambs than of the controls could not be explained."

SUMMARY AND CONCLUSIONS

The inner ear and its connections to the central nervous system have reached a state of maturity by 26 weeks gestation so that sounds of sufficient intensity external to the mother may produce responses of the fetus mediated by the fetal auditory system. Before the auditory system is developed, high-intensity external sounds may affect the embryo or fetus directly by mechanical movement or indirectly via the maternal endocrine and circulatory systems. There is no conclusive evidence of detrimental effects of high-intensity external sound in higher mammals.

Tones of 100-120 dB at the mother's abdominal surface are attenuated by the mother's body and the tissue and fluids surrounding the fetus by approximately: 20-25 dB for single frequencies from 50-200 Hz; 25-30 dB at 500 Hz; 40 dB at 1000 Hz; 50 dB at 2000 Hz; and 70 dB or more at 4000 Hz and higher frequencies. Internal background noise levels of 70-85 dB SPL have been measured in the vicinity of the fetal head; the background noise is probably generated by the mother's circulatory system.

Data on prenatal noise exposure of human subjects are limited in number, lack information on individual noise exposures, have inadequate sample populations, and do not have appropriate control populations. Until better information is available, however, it would appear prudent for pregnant women to avoid exposures of long duration (several hours per day) to sounds of 90 dB SPL and above, the maximum level currently suggested by the U.S. Department of Labor for unprotected ears.

Only limited references have been made to animal experiments because most of them have used animals that differ from humans in many of their basic anatomical and physiological characteristics. Factors that should be considered in selecting an appropriate animal model are given in the following section.

RECOMMENDATIONS FOR RESEARCH

Human Studies

There are two broad categories of approach to the study of the effects of noxious agents in pregnancy. The retrospective approach attempts to reconstruct, from records or from memory, the history of an individual's exposure to the agent during pregnancy, before the beginning of the study. The prospective approach begins before exposure to the agent, and the pertinent events are recorded at the time of their occurrence.

Retrospective studies have an advantage in that large numbers of data may already be available. It is recommended, therefore, that a search be made for sources of data on women who have been exposed to noise during pregnancy. The data should have been carefully recorded and should include such information as period of exposure during pregnancy, daily duration of exposure, noise levels measured, and other environmental pollutants involved. The most likely places in which pregnant women are exposed repeatedly to noise at high levels are probably industrial situations. Special difficulties are encountered in studies carried out in areas around airports. Exposure to high levels of noise may be infrequent, exposure of individuals is difficult to measure, and stress may occur not only because of noise but also because noise interferes with other activities and may be a reminder of the danger of aircraft accidents.

Prospective studies should also be considered. They have a great advantage in that the questions for investigation can be chosen in advance. The primary disadvantage is the length of human gestation and the infrequency with which most identifiable defects occur. In selecting places for prospective studies, factors to be considered include the need for careful record keeping, the adequacy of the noise measurements that can be made, the possibility of making noise exposure measurements for individuals, and other conditions such as many mentioned with regard to retrospective studies.

Human studies are likely to require early evaluation of hearing sensitivity in neonates; evoked response audiometry is the preferred and most practical method now available for such evaluation. In addition to changes in hearing, postnatal examinations of infants should consider such conditions as low birth weight and neonatal-infantile hyperactivity and hyperirritability, with their possible adverse effects on mother-child interaction.

One obvious topic for study would be to select appropriate groups of pregnant women, have them carry a personal noise dosimeter during the course of their pregnancies, and then monitor the hearing of the

infants beginning immediately after birth, using evoked response audiometry. Noise exposure could then be directly related to postnatal hearing characteristics.

The very questions asked in prospective studies may present a psychological hazard for subjects unless done with great tact. The questions may easily inspire fear or anger among the participating subjects, may bias or disrupt the investigation, or may even result in litigation following the birth of a defective child.

Animals Studies

Careful experiments should be undertaken using suitable animal subjects in order to obtain data that may be applicable to human subjects or at least may suggest anatomical and physiological changes to be looked for in human studies.

Animal experiments can be performed on much more uniform populations than can human experiments. Genetic variation and environmental conditions can be strictly controlled. The experimental sound can be programmed, pushed to levels not obtainable under normal human working or living conditions, and can be replicated if necessary. Most experimental animals have a shorter gestation period than that of humans, and the mothers and their offspring may be sacrificed for pathological investigation.

In designing animal studies a number of factors should be considered:

1. The nature of the independent variable--noise--should be specified with great care. Noise intensity, frequency, spectrum, duration, steady state or intermittent should all be controlled. Precise measures of total maternal exposure and good estimations of fetal exposure should be obtained.

2. The species selected should more closely resemble humans in important ways than is the case for animals used in most past experiments. Dogs, sheep, pigs, and macaque monkeys appear to be leading candidates for the next stage of experimentation in this area.

3. Based on the noise and general "stress" literature, the dependent variables could be selected from virtually all bodily organ, tissue, hormonal, enzyme, and protein systems. The most likely influences of noise will act through alterations of the maternal hypophyseal-pituitary-adrenocortical axis (Selye, 1976). Changes in this system have far-reaching implications for the mother and the developing organism.

Animal experiments should involve detailed continous study of maternal and fetal biochemical events throughout pregnancy. Less promising, because of the smaller amount of positive observations likely, is the traditional approach that has sought to observe various gross structural changes.

4. Because it is very difficult in real life to separate exposure to different potentially teratogenic agents, some effort must be made under tightly controlled conditions to investigate the interactive effects of noise with other environmental factors such as heat, crowding, and nutritional state.

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APPENDIX

PREDICTION OF ACOUSTIC TRANSMISSION LOSS INTO THE UTERUS

Peter J. Westervelt

INTRODUCTION

A theory is presented for the attenuation of external airborne noise transmitted through the torso of human mothers. When a fraction q of the effective area of the torso is insonified, the transmission loss is $20 \log q$ decibels, for any frequency below the resonant frequency of any existing gas. At frequencies above the resonant frequency the transmission loss increases at a rate of 12 dB per octave. These results are consistent with the limited amount of experimental data currently available. More experiments need to be performed both with models and human subjects, using hydrophones rather than air microphones modified for use in liquids.

THEORY

The human torso is a complex viscoelastic, liquid fibrous gelatinous mix that defies acoustic analysis unless simplifying assumptions are made. Thus we chose to model it as a spherical ball of water. This is not so drastic an assumption as might at first glance appear. Barring for the moment occluded gas, both the lower part of the torso and the water ball may be considered incompressible for frequencies below 10 kHz. Since the pressure now satisfies Laplace's equation as does the electrical potential, one can immediately transcribe solutions for the electric potential inside a conducting sphere to describe the sound pressure inside a liquid sphere. In particular, problem 3.5 from Classical Electrodynamics (Jackson, 1962:97) is replicated below with pressure p substituted wherever ϕ or V the potential appears.

A hollow sphere of radius a has the pressure specified on its surface to be $p = P(\sigma,\phi)$. The following two forms of solution for the pressure inside the sphere are equivalent:

$$p(\underline{x}) = \frac{a(a^2 - r^2)}{4\pi} \int \frac{P(\sigma', \phi')}{(r^2 + a^2 - 2ar\cos\gamma)^{3/2}} \, d\Omega' \qquad (1)$$

where

$$\cos\gamma = \cos\sigma\cos\sigma' + \sin\sigma\sin\sigma'\cos(\phi - \phi') \qquad (2)$$

$$p(\underline{x}) = \sum_{\ell=0}^{\infty} \sum_{m=-\ell}^{\ell} A_{\ell m} \left(\frac{r}{a}\right)^{\ell} Y_{\ell m}(\sigma,\phi) \qquad (3)$$

where

$$A_{\ell m} = \int d\Omega' Y^{*}_{\ell m}(\sigma', \phi') P(\sigma', \phi') \, . \qquad (4)$$

In applying these results, we take the origin of coordinates $r = 0$ to be the location of the embryo or fetus. $\sigma = 0$ and $r = a$ corresponds to the location of the mother's navel. The sound field will, for simplicity's sake, be presumed to be applied symmetrically about the navel, thus the field will have no dependence on the azimuthal angle ϕ, being symmetric about the axis $\sigma = 0$. Thus, for a small loudspeaker placed over the navel exerting a force F the sound pressure obtained from eq. (1) reduces to

$$p = \frac{PS}{4\pi a^2} \frac{1 - (r/a)^2}{[1 - 2r/a\cos\sigma + (r/a)^2]^{3/2}} \qquad (5)$$

where P is the pressure exerted by the loudspeaker, whose area is S. At the location of the fetus $r = 0$. and

$$p = \frac{PS}{A} \qquad (6)$$

where $A = 4\pi a^2$ the area of the model torso. The transmission loss in this case is given by

$$\text{T.L.} = 20 \log \frac{P}{p} = -20 \log q \qquad (7)$$

where $q = \frac{S}{A}$, the fraction of the torso's area that is insonified. At frequencies below 500 Hz when the entire body is exposed, q might be as large as 0.5 (no exitation presumably coming in the direction of the lungs) leading to a minimum transmission loss of about 6 dB.

At frequencies above 500 Hz two distinct complications set in. First, the body ceases to be small relative to the acoustic wavelength in the air, making the distribution of excitation pressure more complicated. This factor can be eliminated in experiments similar to those performed by Walker et al. (1971) in which the sound application is localized at the position of a small loudspeaker.

The effect of small gas pockets can be taken into account quite easily if the resonable assumption is made that the bubble oscillations are highly damped at their resonant frequency f_r given by (Urick, 1975)

$$f_r = \frac{326}{R} \quad . \tag{8}$$

in which f_r is in Hz and R the bubble radius is in centimeters. Below resonance the bubbles have no effect on the transmission loss. Above resonance the bubbles behave like a simple source. For simplicity we will consider the bubble to be located at $r=0$. It is easier to work with the alternative form, eqs. 3 and 4, in which the pressure is expanded into normal modes. The first three modes only are presented below. The first is the infinite frequency spherically symmetric "breathing" mode

$$p_1 = \frac{PS}{A} \; ; \tag{9}$$

the second is the zero frequency dipole mode

$$p_2 = \frac{3PS}{A} \left(\frac{r}{a}\right) \cos\sigma; \tag{10}$$

and the third is the zero frequency quadrupole mode

$$p_3 = \frac{5PS}{2A} \left(\frac{r}{a}\right)^2 (3\cos^2\sigma - 1). \tag{11}$$

The pressure in the first mode is valid for arbitrary primary pressure distribution, while that for the second and third modes presumes the pressure to be applied at the navel. The physical meaning of the first two modes is quite evident. The first mode represents a pressure independent of position that would result if the model torso was subjected to a uniform pressure PS/A over its complete surface. The resonant frequency of this mode is infinite as a consequence of assuming zero compressibility for the fluid.

The second mode is a manifestation of Newton's Law equating force to mass times acceleration. From the equation of motion

$$\rho \frac{\partial \underline{u}}{\partial t} = -\nabla p_2 \; , \tag{12}$$

and eq. (10), the acceleration $\frac{\partial u}{\partial t}$ of the model torso in the $\sigma = 0$ direction is

$$\frac{\partial u}{\partial t} = - \frac{3PS}{A\rho_w a} , \tag{13}$$

in which ρ_w is the density of water. Integrated over the volume of the torso, this equation becomes

$$m \frac{\partial u}{\partial t} = +F \tag{14}$$

in which M is the mass of the torso and $F = PS$, the force applied to the torso. (Of course, $-F$ is the force the torso exerts on the air, or loudspeaker, reflecting Newton's law of action and reaction, hence the negative sign in eq. (13). The resonant frequency of the dipole mode is zero since no spring restoring force is presumed to act in the $\sigma = 0$ direction.

The quadrupole and the higher-order multipole responses all vanish at $r = 0$ as do their contribution to the acceleration. Thus the first and second modes represent the relevant physical quantities, namely, the pressure and the acceleration, respectively, at the center of the model. The resonant frequencies of the quadrupole mode and all higher-order modes is zero since the restoring force of surface tension and the skin of the torso has been neglected, a very reasonable procedure at acoustic frequencies.

Should a pocket of gas exist at the center of the model and if the radius of the pocket R_b is small compared with the radius a of the model, it is a simple matter to show that the sound pressure in the vicinity of the gas bubble p_b is approximately given by

$$p_b = \frac{\rho_a C_a^2}{\rho_w R_b^2} \frac{3P\left(\frac{S}{A}\right)}{\left[\frac{3\rho_a C_a^2}{\rho_w R_b^2} - \omega^2\right]} \tag{15}$$

in which ρ_a is the density of the gas, ρ_w the density of water, C_a the speed of sound in the gas, R_b the bubble radius and ω the circular frequency. The infinity at resonance when $\omega = \sqrt{3\rho_a C_a^2 / \rho_w R_b^2}$

may be ignored since damping has been left out of this treatment. At low frequencies, below resonance the pressure reduces to the previously found value in the absence of gas. At high frequencies, above resonance the transmission loss may be expressed either as

$$\text{T.L.} = 40 \log \frac{f}{f_R} - 20 \log q \ , \qquad (16)$$

in which f/f_R is the ratio of the frequency and the resonant frequency, or

$$\text{T.L.} = -100 + 40 \log(f R_b) - 20 \log q \ , \qquad (17)$$

in which f is the frequency in Hertz and R_b is the bubble radius in centimeters.

AGREEMENT WITH EXPERIMENT

Intrauterine sound pressure measurements performed by Walker et al. (1971) yielded attenuation or transmission loss of external sound. Unfortunately, a hydrophone was not employed for the intrauterine measurements so that it was felt necessary to correct the results for "impedance mismatch" effects and static pressure effects believed to lower the sensitivity of the condenser microphone employed. Their results for the transmission loss appear in Figure A-1 as a solid line

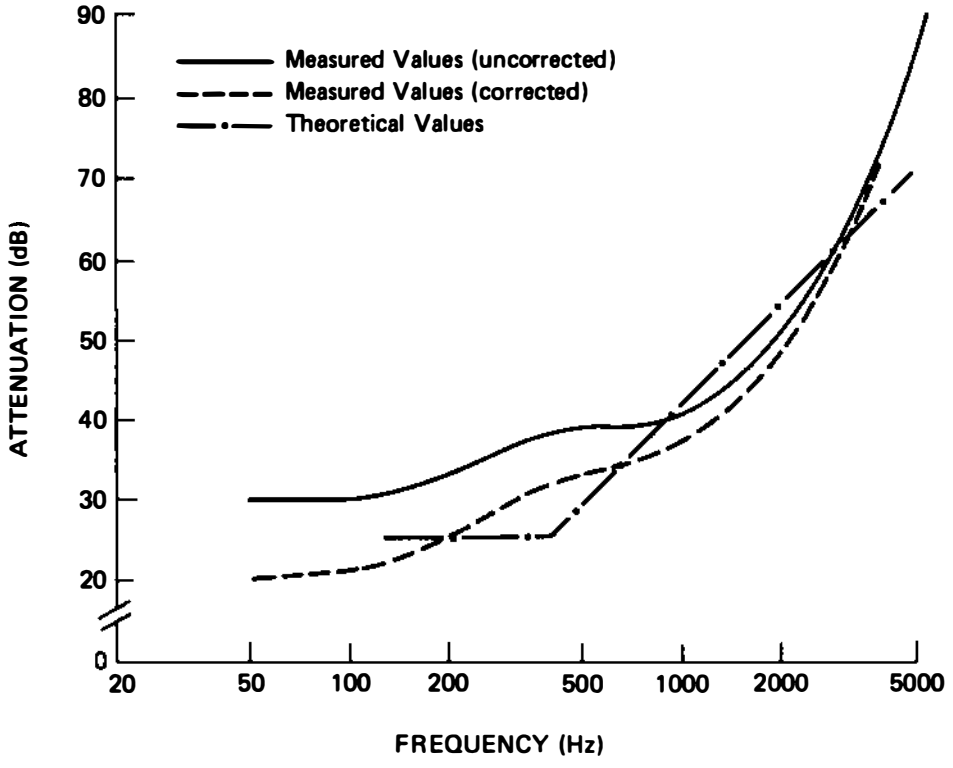

FIGURE A-1 Attenuation of external sound through maternal tissues.

for the uncorrected value and as a dashed line for the corrected values. Lacking further details of the nature of their corrections, I tend to favor their uncorrected results. The intrauterine static pressure is not that high, and at the low frequencies used, the concept of impedance matching is meaningless.

In comparing the theory with the experiment I assume the low frequency transmission loss to be 25 dB. From eq. (7) we find $q = \frac{1}{13.4}$, that is, the area of the model ball is 13.4 times the area of the loudspeaker used to insonify the abdomen. Walker et al. used a 6" loudspeaker, thus its area is $\pi(3)^2$ in^2. Hence the radius of the model ball is given by

$$\frac{\pi(3)^2}{4/3\,\pi a^2} = \frac{1}{13.4} \quad . \tag{18}$$

This leads to a radius a of 9", which is not an unreasonable value. Had 20 dB been chosen for the low-frequency attenuation, a would be 8.2".

Finally, from the figure we estimate that there exists a pocket of gas in the vicinity of the microphone with a resonant frequency of 500 Hz. This requires a bubble of radius 0.65 cm as obtained from eq. (8).

Using eq. (7) at frequencies below 500 Hz and eq. (16) for frequencies above 500 Hz, the theoretical transmission loss is plotted as a dash-dot line on the figure. Agreement between theory and experiment is not bad considering the crude model adopted to represent a complex system.

CONCLUSION

The theory presented can stand many refinements. Interfering effects of the spinal column, shear waves, and deviation of lower torso from perfect sphericity are examples. Likewise, the experimental situation sorely needs attention. Hydrophones should be used inside the body instead of modified air microphones.

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The U.S. Air Force asked the Committee on Hearing, Bioacoustics, and Biomechanics (CHABA) to respond to three specific questions regarding the effects on the embryos or fetuses of pregnant women exposed to high-intensity noise. The questions asked were: (1) What are the potential hazards of noise exposure? (2) On the basis of current knowledge, can reasonable limits be specified for conditions of noise exposure? (3) What research efforts, if any, should be encouraged in

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order to obtain better answers to the first two questions? The Air Force request was endorsed by representatives of the National Institute of Occupational Safety and Health (NIOSH) and the Environmental Protection Agency (EPA).

The Committee on Hearing, Bioacoustics, and Biomechanics appointed Working Group 85 to consider the questions raised by the Air Force. The following brief report reviews the research considered relevant by the working group, points out the problems and limitations encountered in this research and its evaluation, and concludes that on the basis of available data definite answers cannot be given to questions (1) and (2). Recommendations are made for further research.