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NOISE AND THE FOETUS:
A CRITICAL REVIEW OF THE LITERATURE

Peter G Hepper
and Sara Shahidullah

School of Psychology
The Queen's University of Belfast
Belfast BT7 1NN
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This review critically examines the effects of prenatal noise exposure on subsequent hearing and reproductive outcome. The development of the human auditory system and the intrauterine sound environment of the human foetus are described. Evidence that the foetus responds to sound is reviewed and the possible adverse effects of prenatal noise exposure discussed. Studies examining the effects of noise exposure during pregnancy on later hearing, drawing evidence from animal and human studies, are reviewed and evaluated. The effects of noise exposure during pregnancy on reproductive outcome are discussed. The implications for the health of the foetus, legislation and the protection of the foetus from noise exposure are presented.

CONTENTS

FOREWORD

*"For behold, when the voice of your greeting came to my ears,
the babe in my womb leaped for joy"*

Luke 1:44

Although there have been many anecdotal reports that the foetus hears it is only recently that investigators have demonstrated this ability beyond doubt. Indeed in recent years our picture of the intra-uterine environment, and indeed of the foetus itself, has undergone a remarkable change. No longer is the foetus viewed as a passive, unresponsive, organism, existing in an environment devoid of stimulation but rather one with a sophisticated behavioural and sensory repertoire able to respond to the diverse and changing sensory stimuli present in its environment. In recent years the realisation that the foetus is responsive to external stimuli has bought about concern over the possible damaging effects of overstimulation. Although it has long been known that environmental agents, e.g. drugs, disease, may cross the placenta and harm the foetus little attention has been given to the possible adverse effects of intense sensory stimulation. This report examines the evidence relating to possible adverse consequences of noise exposure during pregnancy on the foetus and its subsequent outcome.

We are continuing our work examining the behavioural, sensory and neural development of the foetus and would welcome any comments or questions arising from this report. These should be sent to

Peter G Hepper
School of Psychology
The Queen's University of Belfast
Belfast
BT7 1NN
N. Ireland.

Peter G Hepper
Sara Shahidullah
14-6-93

ABSTRACT

This review assesses the effects of noise during pregnancy on the subsequent hearing and health of the foetus and child. Evidence from both human and animal studies is considered. There is little experimental evidence (only 2 studies on humans) on which to base any firm conclusions regarding the effects of noise on later hearing. Theoretically the foetus is at risk from noise exposure but the intensity and duration of sounds that may cause harm are unknown. Observation of the sound environment of the foetus indicates that low frequency sounds (250Hz and below), which pass unattenuated through the maternal abdomen to stimulate the foetal ear, may be most likely to harm hearing. The use of the 'A' weighted decibel scale is thus inappropriate for assessing noise levels in relation to the foetus. Of particular concern is the application of adult models of hearing loss to the effects of noise exposure on the foetus. The auditory system of the foetus is different to that of the adult and this may result in different effects of noise exposure on hearing loss. The protection of the foetus from noise, and a reduction in the risk of hearing loss, can only be achieved by reducing the amount of noise entering the abdomen and stimulating the inner ear of the foetus. With regard to reproductive outcome noise does not appear to be a teratogen. Similarly there was no conclusive evidence that noise exposure affected other measures of reproductive outcome, e.g. low birth weight or prematurity. Any effects of noise on reproductive outcome are most probably mediated by the mother's response to noise and thus the provision of protective head gear to reduce noise exposure may reduce any adverse effects. Maternal stress in response to noise may also play a role and here it is important to reduce stress levels rather than noise *per se*. In conclusion further studies are required in order to determine definitively the effects of noise on the foetus.

INTRODUCTION

1   The aim of this review is to assess critically the effects of prenatal noise exposure on the subsequent health, in particular hearing, of both the foetus and child. The effects of noise on the hearing of exposed workers has been the subject of considerable study, and although gaps remain in our understanding there is sufficient knowledge for risk tables to be produced (British Standards Institution 1976; International Standards Organization 1990; Robinson 1991). However, where pregnant women work in a noisy environment there is some suggestion that the hearing of the foetus might also be affected, though this is much less well understood. This review examined such scientific evidence as is available and comments on whether and how improved knowledge may be obtained. When the study was commissioned by the Health and Safety Executive it was expected that the European Commission would propose a directive requiring special controls where pregnant women are employed in noisy areas, with particular concern over low frequency (500Hz and less) noise exposure. Since then the European Commission has dropped this aspect of their proposal on health and safety requirements for workers exposed to physical agents. A separate directive has however been adopted by the European Community on the improvement of the health and safety of pregnant workers (European Economic Community 1992, 1993). Assessment of exposure to noise is required where pregnant workers are exposed to specified agents, one of which is noise. The topic is therefore of continuing interest in the development of future standards for industrial noise control and the health and safety of pregnant workers and their foetuses.

2   In adults it is well documented that long-term exposure to noise in excess of 85dB(A) may result in hearing loss, and this is especially prevalent amongst those working in a noisy environment where there is a close relationship between noise immission and hearing impairment (Burns & Robinson 1970; Hamernik, Henderson & Salvi 1982; Johnson 1991; Kryter 1973; Mills & Going

1982; Passchier-Vermeer 1968; Ward 1984). Most industrial societies have established guide-lines and regulations to limit the amount of exposure to noise at work to prevent damage to hearing. In the UK, the Noise at Work Regulations (1989) provide the appropriate legislation and measures for the control and assessment of noise in an occupational setting. This requires that employers provide ear protectors to reduce the level of noise exposure when the noise exposure of employees reaches 'the first action level', a personal daily exposure of 85dB(A). Despite many years of concern over noise-induced damage in adults there has been little attention given to possible harmful effects in the foetus arising from noise exposure to the pregnant woman. Consequently there has been no consideration of, or guidance for, the levels of noise exposure appropriate for the pregnant woman and her foetus.

3   The first aim of this review is to evaluate critically the available evidence relating to the effects of noise on the foetus. In the course of surveying the literature for this review it became apparent that there were few studies which have assessed the effects of prenatal noise exposure on later health, and even fewer assessing the effects of prenatal noise exposure on the individual's subsequent hearing abilities. Although there is a large literature concerned with the effects of noise exposure after birth, especially in adults (e.g. Dancer, Henderson, Salvi & Hamernik 1992; Dejoy 1984; Hamernik, Henderson & Salvi 1982; Jones & Chapman 1984; Kryter 1973; Mills & Going 1982; Passchier-Vermeer 1968; Smith 1989; Ward 1984), there has been little examination of the effects of noise on the foetus and its subsequent development and health.

4   Some studies have been performed examining the effects of noise on pregnant animals. However interspecies differences in physiology and the auditory system mean that extrapolation from animal to humans must proceed cautiously (e.g. Dancer 1981; Decory, Dancer & Aran 1992). Animal studies may have a role in indicating some general principles of noise-induced adverse effects but cannot be used to determine the absolute sound levels, or duration of sound, which may lead to harm in the human foetus.

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5   The lack of studies examining the effects of noise on the foetus, and its subsequent outcome, mean that other sources of information must be evaluated in order to predict the effects of noise on the foetus. One obvious source is the large body of research which has examined the effects of noise exposure in adults. The results of this research may be used to predict any harm that may be caused as a result of intra-uterine noise exposure. Indeed, given the lack of empirical studies relating to prenatal noise exposure, any proposals regarding limits of noise exposure will be presumably based on studies of postnatal noise exposure. Such a direct extrapolation assumes that the effects of prenatal noise exposure on the foetus will be similar to the effects of postnatal noise exposure in adults. This, however, may not be true and is based upon the assumption that the sound environment and the auditory system of the foetus are similar to that of the adult. This is not the case and there are sufficient differences between the adult and foetal auditory system and environment to suggest there may be different effects of noise exposure in the foetus and adult. These differences are explored to determine how noise may affect the foetus.

6   This review assesses the effects of noise exposure during pregnancy on the developing foetus and its subsequent health and hearing. The available evidence drawn from empirical studies of prenatal noise exposure is discussed. Evidence from other spheres of investigation (including foetal hearing, auditory system development and noise-induced hearing loss in adults) is evaluated in an attempt, in the absence of empirical evidence, to predict the possible effects of intra-uterine noise exposure.

7   In order to evaluate the effects of noise exposure it is important to have some knowledge of noise and sound. Section II and Section III briefly consider noise in general and the physics of sound to provide background information to enable the assessment of the effects of noise exposure in the foetus.

8   Section IV discusses the development and functioning of the foetal auditory system. The perception of sound is mediated by the auditory system and the

effects of noise exposure on hearing will be determined, in part, by the functioning of the auditory system. Since the prenatal period covers the time of formation and initial functioning of the auditory system, the developmental status and maturation of hearing will play an important role in influencing the response of the foetus to sound. Section V considers the sound environment of the foetus. The foetus exists in a very different environment to that of the individual after birth and this will have important implications for the transmission of sound and consequently the foetus's experience of noise. In both of these sections particular emphasis is placed upon highlighting and evaluating how the differences between foetal auditory abilities and those of the adult may be expected to influence the response of the foetus to noise. Section VI discusses how the functional status of the auditory system before birth and the sound environment of the foetus will combine to influence sound as it reaches the inner ear of the foetus. Section VII examines the evidence that the foetus 'hears'. Recent studies have started to investigate the 'hearing' abilities of the foetus and these provide evidence of the functional status of the auditory system before birth.

9 A brief review of the current state of knowledge regarding noise-induced hearing loss is provided in Section VIII. Although there has been little direct study of the effects of noise on the foetal auditory system, research examining noise-induced hearing loss in adults may have implications for the type of damage that may be expected in the foetus. The possible effects of intra-uterine noise exposure on later hearing abilities are also discussed.

10 Section IX reviews studies which have examined the effects of prenatal noise exposure on later development and hearing. These studies are presented individually and critically evaluated. Both animal and human studies are discussed and the difficulties of extrapolating from animal to human studies are presented. The first part of this Section deals with the effects of noise exposure on the individual's subsequent hearing abilities. The second part of this Section discusses those studies which have examined the effects of noise

exposure on reproductive outcome and health. The evidence is summarised and implications for protection and legislation discussed. Directions for future research are presented.

11   Section X summarises the evidence pertaining to the effects of prenatal noise exposure and relates this to future proposals concerning health and safety aspects of noise exposure and the pregnant woman.

SECTION II

NOISE

12   Noise can be defined as "a sound, especially one that is loud or disturbing" (Collins English Dictionary). Although in terms of their physical description noise and sound are identical, noise can be differentiated from sound (defined as "a periodic disturbance in the pressure or density of a fluid or in the elastic strain of a solid produced by a vibrating object", Collins English Dictionary) by the nature of its *affective* quality. That is noise is often associated with nuisance or has an annoying quality to it.

13   Noise has, since the development of human civilisation, presented problems for its nuisance value. Examination of the writings of Roman scholars and poets reveals that noise created problems even in these early times. Laws were passed to control the noise levels in towns. For example, chariots were banned from the streets after late evening due to the noise created by their metal wheels on the cobblestone roads. Queen Elizabeth I passed a law to prevent the beating of one's wife in the evening, not because of any moral disquiet, but because the incessant sobbing of the wife after a beating disturbed the sleep of nearby citizens. Today noise is considered one of the most ubiquitous pollutants and in the UK, in common with many other countries, numerous Parliamentary Acts and local bye-laws have been enacted with the specific aim of controlling noise pollution.

14   The adverse effects of exposure to noise on the individual's hearing were also noted during early civilisations. Pliny the Elder, 1AD, noted that workers operating in noisy conditions suffered a loss in their hearing. With the Industrial Revolution the exposure of workers to intense noises became more commonplace and the effects of noise on hearing were readily identified, e.g. Fosbroke (1830). Since then there has been much concern over the effects of occupational noise on hearing.

15   The effects of noise on the individual have been divided into two separate categories of effect (Dejoy 1984; van Dijk 1986). First, noise has been studied with respect to its effects on the auditory system of the individual. Second, noise has been studied with respect to its effects on other physiological and psychological spheres of activity. This second category is rather a 'catch all' category including everything other than the effects of noise on hearing. This may include physiological (e.g. Sanders, Freilicher & Lightman 1990; van Dijk 1986), performance (e.g. Smith 1989), and health (e.g. Clark 1984), effects. Both types of effect, auditory and non-auditory, are discussed in Section IX.

16   Noise may be divided into a number of different forms (Dawson 1984):

17   Din: a sound with energy across the entire, or part of, the audible spectrum, 20Hz to 20,000Hz. It is the effects of this type of noise, i.e. audible, that shall be discussed in this paper and noise within this range has been the concern of most legislation and regulation governing noise at work.

18   Infrasound: sounds with frequencies below 20Hz. For humans sounds within this range, especially below about 16Hz, lose there tonal quality and are perceived as a "chugging" or "popping" sensation. The effects of hearing these sounds are poorly understood, however they exert a direct physiological effect on bodily function (Dawson 1982). Such low frequency sounds are often associated with vibration at similar frequencies. The effects of vibration are beyond the scope of this review and readers are referred to Seidel & Heide (1986) and Wasserman (1990) for comprehensive reviews of this area.

19   Ultrasound: sound in frequencies above the range of human hearing by air conduction, i.e. above 20,000Hz. There is some evidence that bone conduction may enable the detection of auditory stimuli above this frequency and possibly up to 60,000Hz (Corso 1963). Due to the fact that this noise is largely inaudible, and infrequent in most occupational

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settings, there has been little attention given to its effects on hearing. However concern has been expressed by the American Conference of Government Industrial Hygienists over the possible harmful effects of subharmonics of ultrasound on hearing.

20   One highly specialised form of ultrasound that may be encountered during pregnancy is that used to observe the foetus during the clinical management of pregnancy. Indeed most pregnancies in the UK will, at some time, be exposed to this form of ultrasound. Due to the widespread use of diagnostic ultrasound there have been many examinations of its effects on the foetus and possible harmful consequences of use. Although the frequencies used in obstetric ultrasound are outside of the range of human hearing (3.5-7.5MHz) some studies have assessed its effect on hearing after birth and overall have found no evidence that ultrasound affects subsequent hearing (e.g. Petitti 1984; Stark, Orleans, Haverkamp & Murphy 1984). However one study examining auditory brain stem responses (ABR) in sheep after a period of ultrasound found a decrease in the mean amplitude of the ABR and an increase in the latency of all five wave components during insonation (Siddiqi, Meyer, Woods & Plessinger 1988). These returned to normal values 30 minutes after the cessation of ultrasound exposure. Thus while ultrasound may temporarily influence the ABR by affecting neural conduction within the central nervous system a permanent effect on hearing has yet to be demonstrated.

21   Impulse noise: noise that arises from a rapid release of energy, very sudden in its onset and often of short duration, e.g. gun-fire (Hamernik & Hsueh 1991). The peak overpressures arising from these sounds may exceed one atmosphere (194dB spl). The effects of exposure to this type of noise are often very different to that resulting from continuous exposure to din.

22   Noise may be produced by an almost infinite variety of sources, loud radios, road traffic, industrial machinery and digital alarms to name but a few. This huge range of noise sources and forms which the noise may take, means that it is important to accurately quantify noise in order to evaluate the noise levels which result in damage.

Summary

23   Exposure to noise may result in damage to the auditory system as well as exerting an effect on other physiological systems, health and psychological performance. These adverse effects have been recognised for many centuries and legislation has been enacted to control both the nuisance value of noise and its adverse effects on hearing. Noise may take a variety of differing forms indicating the importance of accurate assessment of its physical properties.

THE PHYSICS OF SOUND

24   In order to understand the effects of noise on hearing it is necessary to have a very basic understanding of the physics of sound. Of particular concern are the basic terms used to quantify a sound or noise which form the basis for legislative regulations. These are discussed below. It is not intended to enter into a full discussion of the physics of sound, but rather to discuss the necessary concepts which will facilitate an understanding of the effects of noise on the developing foetus and hearing loss. Readers are referred to Gerber (1974) and Pierce (1981) for a full discussion of the physics of sound.

25   Sound is generated when an object moves back and forth in a medium (gas, liquid or solid, although in this latter case the motion may be referred to as vibration rather than sound, Oliver 1989) creating a pressure wave. The motion of the sound source initially forces the molecules in the medium closer together creating a compression (increased pressure) and then pushes them apart resulting in rarefaction (decreased pressure). The movement of the molecules is similar to the object causing the motion, and each molecule transmits the motion to adjacent molecules where the back and forth movement is repeated. In this fashion the sound wave spreads outward from its originating source.   It should be noted that although the pressure wave moves out from the source, the movement of the molecules is purely local. This pressure wave, when it impinges on the ear, is perceived as sound. Two main factors determine the perception of sound: the number of times the molecules undergo compression and rarefaction in a set time and the magnitude of the pressure change induced in the medium by the vibrating object. These factors determine the frequency and intensity of the sound and are described in more detail below.

Frequency

26   In its most simple form the motion of the vibrating object can be described as a sinusoidal waveform (Figure 1). This waveform represents the changes in pressure measured at a particular point over time. One cycle comprises a period of increased pressure and a period of decreased pressure. The number of times the cycle is repeated in one second is termed the frequency of the sound. The unit of measurement of frequency is termed the Hertz, abbreviated to Hz.
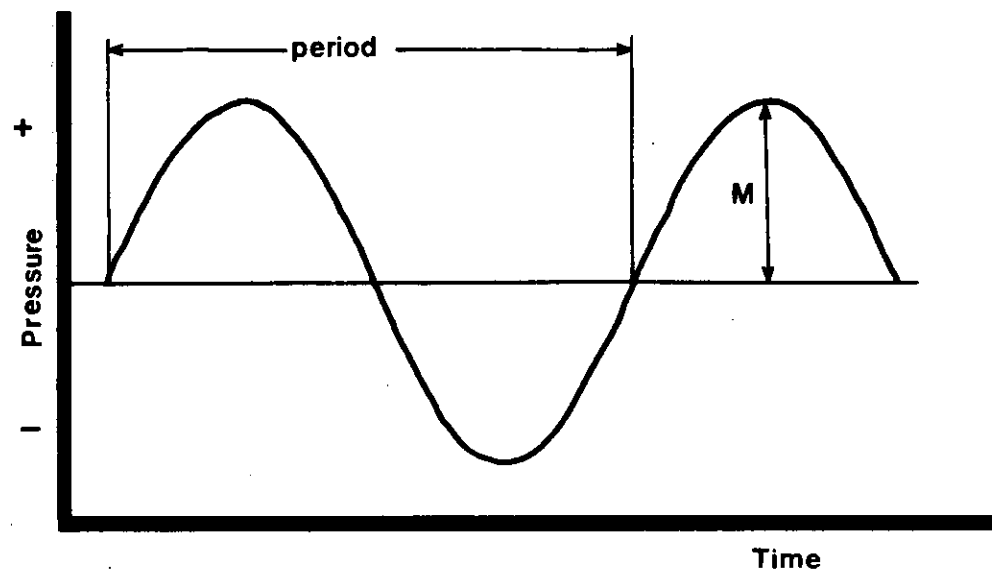
FIGURE 1. Sinusoidal waveform. The graph shows how pressure changes with time when measured at a given point. One complete cycle corresponds to a sequence of compression (+) and rarefaction (-). The number of times this is repeated in one second is the frequency (Hz). M is the point of maximum pressure and corresponds to the magnitude of the sound, measured in decibels.

27   Whilst frequency, in terms of Hertz, is the physical parameter describing the sound, the psychological parameter, i.e. what the listener perceives, is pitch. Pitch can be measured in 'mels'. Although high frequency sounds are perceived as high in pitch and low frequency sounds are perceived as low in pitch, there is not a direct 1:1 relationship between Hz and mels.

28   The range of adult hearing is approximately between 20Hz and 20,000Hz (20kHz), although hearing ability in the high frequency range decreases with age.

29   Sounds are usually made up of more than one frequency and the frequency composition of the sound may be described using a sound spectrum, an analysis which describes the amount of energy within each particular frequency (Martin 1986). Common types of sounds are:

Pure tone: a sound of a single frequency, often used in the assessment of hearing abilities.

White noise: a sound containing roughly equal sound energy at all audible frequencies 20Hz-20kHz.

Broad-band noise: a sound with energy between a certain range of frequencies or all audible frequencies and the energy at each frequency is not equal. This type of sound is most often associated with noise produced in an occupational setting.

Intensity

30   The motion of a vibrating object imparts a certain force to the air molecules. The greater this force, the further the molecules move from their resting point and the greater the compressions and rarefactions. The greater the force, the greater the particle displacement and the greater the amplitude of the sound.

31   The human ear is sensitive to a huge range of sound pressures. From the quietest to loudest sound which is able to be perceived without harm there is an increase in pressure of over 1,000,000,000,000:1. At the lowest perceivable sound pressures the ear drum may move as little as one thousand millionth of an inch (Dallos 1988).

32   In order to provide a usable means to describe the magnitude of a sound a logarithmic unit, the decibel (abbreviated to dB), was derived. The decibel is essentially the ratio between two sound intensities, I_1 and I_2 and is given by

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the equation $10 \log_{10} (I_1/I_2)$. In order for the decibel to be used as an absolute scale of magnitude, a standard reference level is used as the denominator.

33   Thus the intensity, L_I, of a particular sound, I_1, is given by

$$L_I = 10 \log_{10} (I_1/I_{ref}),$$

where the reference level, I_{ref}, is 10^{-12} watts/m^2.

Alternatively the power, L_W, of a particular sound, W_1, is given by

$$L_W = 10 \log_{10} (W_1/W_{ref}),$$

where the reference level, W_{ref}, is 10^{-12} watts.

34   For these measurements the reference levels used (I_{ref}, W_{ref}) are roughly equivalent to the intensity and power, respectively, of the quietest sound perceivable by the human ear.

35   For environmental control the intensity of a sound is assessed by determining sound pressure. Here the sound pressure (L_p) of a particular sound, P_1, is defined as

$$L_p = 20 \log_{10} (P_1/P_{ref}) \text{ dB}$$

where P_{ref} is the reference level, 2×10^{-5} N/m^2.

36   Although it is not essential to provide details of the mathematics behind these measures it is important to realise that the decibel may be calculated in different ways. Thus when reporting the magnitude of a sound using the decibel it is important to specify:

a) the quantity used to measure intensity. Note that doubling the *power* of a sound results in a 3dB increase in magnitude whereas a doubling of the *sound pressure* results in a 6dB increase in magnitude; and

b) the reference level used in the measurement. The reference level

used is essential to enable comparisons between different studies, but is often not reported. Different reference levels may be used, especially when measurements are made in different mediums (e.g. $0.1N/m^2$ is used for underwater acoustics)

37   It should also be noted that because the decibel is a logarithmic unit, whose value is a ratio between two sound intensities or pressures, the combination of decibel values is not simply one of addition or subtraction. Thus the addition of two sound sources, L_{p1} & L_{p2}, of 60dB does not result in a combined sound pressure of L_{p1+2}=120dB but rather L_{p1+2}=66dB. Thus care should be exercised when combining two, or more, separate sounds.

38   There are a number of specialised decibel based units which are commonly used in the evaluation of sound intensity and a brief description of the most relevant is given below (see Schultz 1972 for a comprehensive discussion).

Weighted sound pressure level

39   The human ear does not respond linearly across all frequencies. Assessment of the intensity threshold required to hear different frequencies indicates that different frequencies require different intensities of sound to be detected or perceived equally (Figure 2). In order to equate the measurement of sound to the response of the human ear a number of different scales have been constructed which weight the power at each frequency differentially. The most frequently used is the 'A' scale which equates to the responsiveness of the human ear and thus reduces the contribution of low frequency sounds to the assessment of sound intensity (Figure 3). Decibel measurements using this scale are reported dB(A). More generally decibel levels are reported dB(x), where x is the particular weighting scale used. A number of other weighting scales have also be used, 'B' and 'C', which give progressively more weight to the low frequencies in the assessment of sound intensity, and 'D', 'E', and 'SI', which are used in the assessment of annoyance level, perceived noise level and speech interference level, respectively.
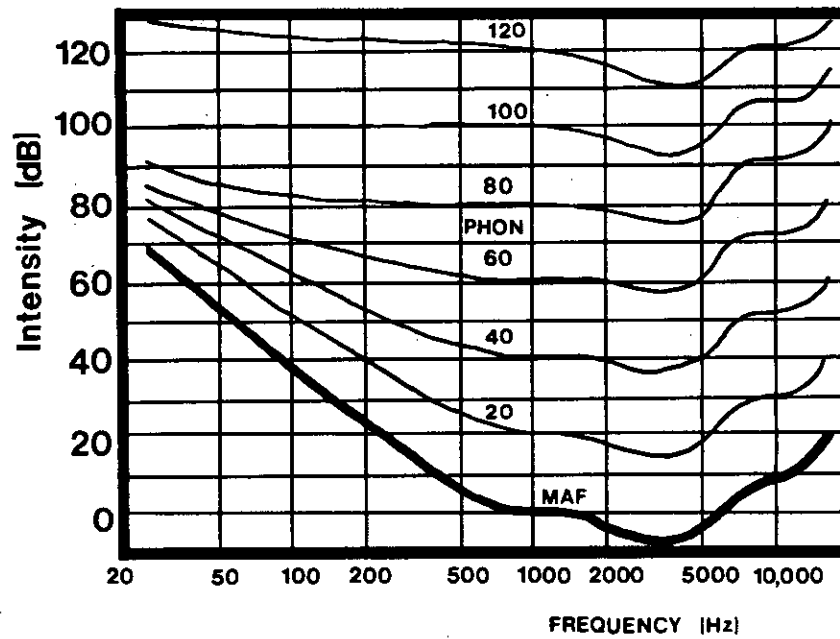
FIGURE 2. Minimum intensity level required to perceive different frequencies (MAF) and equal loudness contours (phons) showing relationship between level of loudness and intensity (dB).
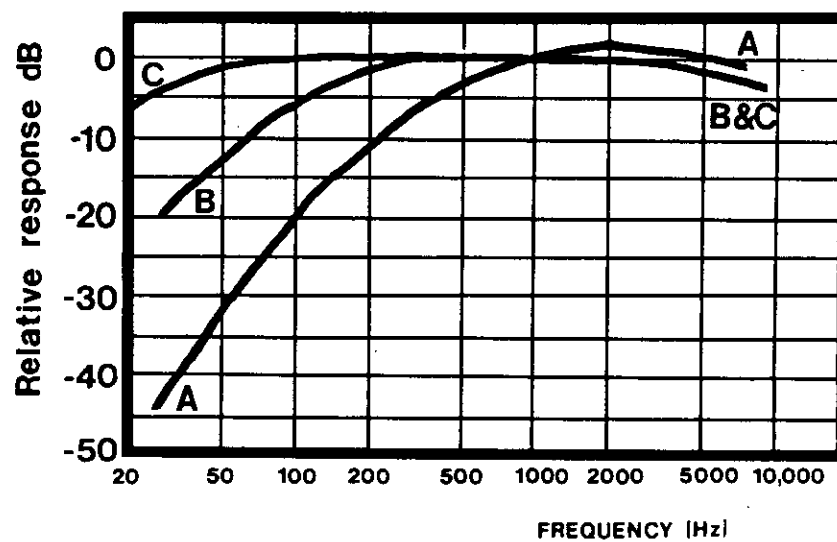

FIGURE 3. Different weighting scales used in the assessment of sound intensity. Note the reduced contribution of low frequency sounds to sound level when using the 'A' weighting scale.

40   The 'A' weighting scale has been used to assess both the response of the foetus to sound and to document the intra-uterine sound levels during pregnancy. However, the 'A' scale equates to the response of the adult ear examined using air-borne sound. The foetal ear, surrounded by amniotic fluid, having fluid present in the outer and middle ear, and an immature auditory system, may exhibit very different response characteristics. The 'A' weighting scale is thus unlikely to be representative of the response pattern of the foetal ear and is thus inappropriate for sound measurements relating to the foetus.

41   There is the suggestion that for low frequency sounds the risk of hearing impairment is increased. Again it should be noted that the 'A' weighting scale reduces the contribution of low frequency sounds to the assessment of sound pressure level. Thus if low frequency sounds are important determinants of noise-induced adverse effects in the foetus, the use of 'A' weighting scales may underestimate their contribution to noise levels.

Equivalent sound pressure level

42   The intensity of sound does not remain constant but varies over time. This is particularly true of noise in occupational settings. In these situations some measure of noise exposure is required in order to estimate the hazards of any such exposure. Based on the assumption that damage to hearing is a function of the total acoustic energy received the Equal Energy Principle, proposed by Eldred, Gannon and von Gierke (1955), has been used as a means to evaluate noise exposure over a range of varying exposure conditions. This measure assumes that total acoustic energy received is the product of noise intensity level and duration of exposure Thus if noise intensity level increases then the duration of exposure must decrease in order for the total acoustic energy received to remain constant. Thus exposures of varying intensities and durations can be equated to a level of continuous noise exposure, L_{eq}.

43   The equivalent sound pressure level, L_{eq}, has been used to assess noise
      exposure in occupational settings where noise exposure varies with time.
      Essentially this measure represents the average magnitude of the sound over
      a particular period of time (see Bies & Hansen 1990; 'Noise at Work, Guidance
      of Regulations' 1989; Schultz 1972, for formula used in its calculation). Thus
      L_{eq8h}, may be used to represent the average noise exposure over a working
      day, and L_{eq40h} to represent noise exposure over the 40 hour working week.
      From this it is possible to determine the average sound magnitude over a
      variety of different durations, e.g. in this particular review noise levels over
      the nine months of pregnancy (L_{eq9m}) would be an important indicator of
      noise exposure. One specialised form of this measure is the L_{dn} or day-night
      equivalent level which assesses noise over a 24 hour period and is often used
      to assess community noise levels.

44   The use of L_{eq} as the measure for assessing noise exposure has been criticised
      for not taking into account the temporal patterning of sound exposure and
      the effect of periods of no sound on hearing (e.g. Bies & Hansen 1990; Ward
      1984). For example, using the L_{eq} value, a continuous period of four hours
      exposure to a sound of 85dB(A) followed by four hours of no noise exposure
      is equivalent to a sound exposure of one hour at 85dB(A) followed by one
      hour of no noise, repeated four times during an eight hour period. These two
      different noise exposures however may result in different effects on the
      auditory system, but under the 'equal-energy' hypothesis are quantified
      identically. Whilst the L_{eq} may not accurately reflect the noise levels leading
      to harm, it has been argued to be a conservative measure of noise exposure,
      i.e. it over-estimates the adverse effects of noise exposure in situations where
      noise and no-noise periods are inter-mingled (Campo & Lataye 1992). As
      such it errs on the 'right side', and has been maintained in determination of
      sound intensity exposure for legislative purposes.

Acoustic impedance and resonance

45   The propagation of a sound, i.e. the transmission of acoustic energy, depends on the qualities of the particular medium through which the sound is travelling.   Whilst sound energy may pass relatively easily through one medium when the energy meets an interface with another medium the transmission of the sound may be impeded. Much of the energy in the original signal may be reflected back and not pass to the second medium. The impedance of a medium, or object, can be defined as the resistance it offers to the transmission of sound energy. Objects having similar acoustic impedances allow acoustic energy to pass easily between them. The greater the impedance the less energy that is transmitted to the new medium and the more that is reflected back.

46   This is of particular importance when considering the effects of sound on the foetus. Sounds generated externally to the mother will be initially transmitted in air, a gaseous medium. To reach the foetus they must pass from the air into the mother's body. Although the mother's body is made up of dissolved gases, liquids and solids, for the purpose of sound transmission it may be considered a relatively uniform fluid medium (Oliver 1989). Thus external sounds have to be transmitted across this, in general terms, air:fluid interface in order to be perceived by the foetus *in utero*. The transfer of acoustic energy from air to water has been estimated to result in a loss of approximately 99.9% of the sound energy, or approximately a 30dB loss in sound energy (DeWeese & Saunders 1964). Thus sounds generated externally to the mother's body may be considerably quieter within the womb than outside due to the loss of acoustic energy as sounds enter the abdomen. This is discussed in more detail in Section V.

47   Impedance depends on both the mass (the greater the mass the more likely to impede high frequencies) and stiffness (the greater the stiffness the more likely to admit high frequencies). The mother's abdomen may differentially attenuate different frequencies. That is for a broad-band sound, not all frequencies would be equally impeded. Furthermore as both the mass and

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stiffness of the abdomen change during pregnancy, especially the mass, the impedance of the maternal abdomen will change throughout gestation.

48   Some consideration should also be given to the propagation of sound within the abdomen. Acoustic energy will pass easily through a uniform medium. Sounds travelling from the external environment have to pass through tissue, gas, and liquid, to reach the fetal ear. At the beginning of pregnancy the relative composition of tissue structures may be similar to each other, and to the surrounding environment of gas and fluid. Thus their impedances may not be that different and the transmission of energy within the abdominal cavity may proceed with relatively little loss of energy. However as pregnancy progresses the relative composition of these may change, e.g. as cartilage becomes calcified and turns to bone, creating larger impedance differences between intra-uterine structures and this may affect the transmission of sound within the maternal abdomen.

49   Objects have a natural resonant frequency. That is a frequency at which it is most easily set into vibration and at which sound decays most slowly. The maternal abdomen will have a particular resonant frequency which, as the abdomen changes in mass during pregnancy, will change during gestation. The resonant frequency of the nonpregnant human abdomen to whole-body vibration is between 3-9Hz (Clark, Lange & Coermann 1962; von Gierke 1971). This is below the audible frequency range for man but whether this remains the same for the pregnant uterus is unknown. Examination of the resonant frequency of the uterus of the pregnant and nonpregnant sheep to whole-body vibration indicates there is little change in its resonant frequency due to pregnancy (Peters, Abrams, Gerhardt, Burchfield & Wasserman 1992). Thus certain frequencies, most probably very low frequencies below 20Hz, will more easily pass into the abdomen and uterus and will decay very slowly.

Summary

50   Noise, or sound, may be quantified in terms of frequency and intensity. A number of specialised units of intensity exist which are used in the

assessment of noise exposure. Sound energy may be impeded by the maternal abdomen resulting in a decrease in sound energy as the acoustic stimulus passes from the air into the abdomen. Once in the womb the acoustic energy may proceed relatively unimpeded to the inner ear of the foetus. Certain frequencies will be less impeded than others by the abdomen depending on the resonant frequency of the abdomen.

SECTION IV

THE AUDITORY SYSTEM

51   This section examines the structure, function and development of the
auditory system. Particular consideration is given to differences between the
foetal and adult auditory system and their functional significance.

52   The human auditory system may be broadly divided into two parts, the
auditory periphery, responsible for the collection and transduction of sounds
into neural impulses and the auditory nervous system, responsible for the
transmission and encoding of the neural signals. Hearing loss resulting from
noise exposure exerts its effect by directly harming the structures of the
auditory periphery (reviewed Saunders, Cohen & Szymko 1991; Saunders,
Dear & Schneider 1985). This damage to the auditory periphery may
subsequently result in alterations to the auditory nervous system (Gerken,
Simhadri-Sumithra & Bhat 1986; Saunders, Cohen & Szymko 1991), however
there is no evidence that noise exerts a direct effect on the auditory neural
pathways (Patuzzi 1992; Saunders, Cohen & Szymko 1991; Saunders, Dear &
Schneider 1985). An understanding of the function and development of the
auditory periphery is important in the determination of the effects of noise
exposure on the foetus.

53   The auditory periphery consists of three parts, the outer, middle and inner
ear (Figure 4). The outer ear, consisting of the pinna and auditory canal,
captures and transmits sound to the ear-drum causing it to vibrate. The three
ossicles of the middle ear transmit these vibrations to the inner ear. The
inner ear contains the sensory structures, within the cochlea, which convert
the sound energy to neural impulses.

54   In the following section the anatomy, development and function of the
auditory periphery is presented. The differences between the mature and
foetal auditory system are highlighted and the effect of these differences on
the functioning of the auditory system discussed.
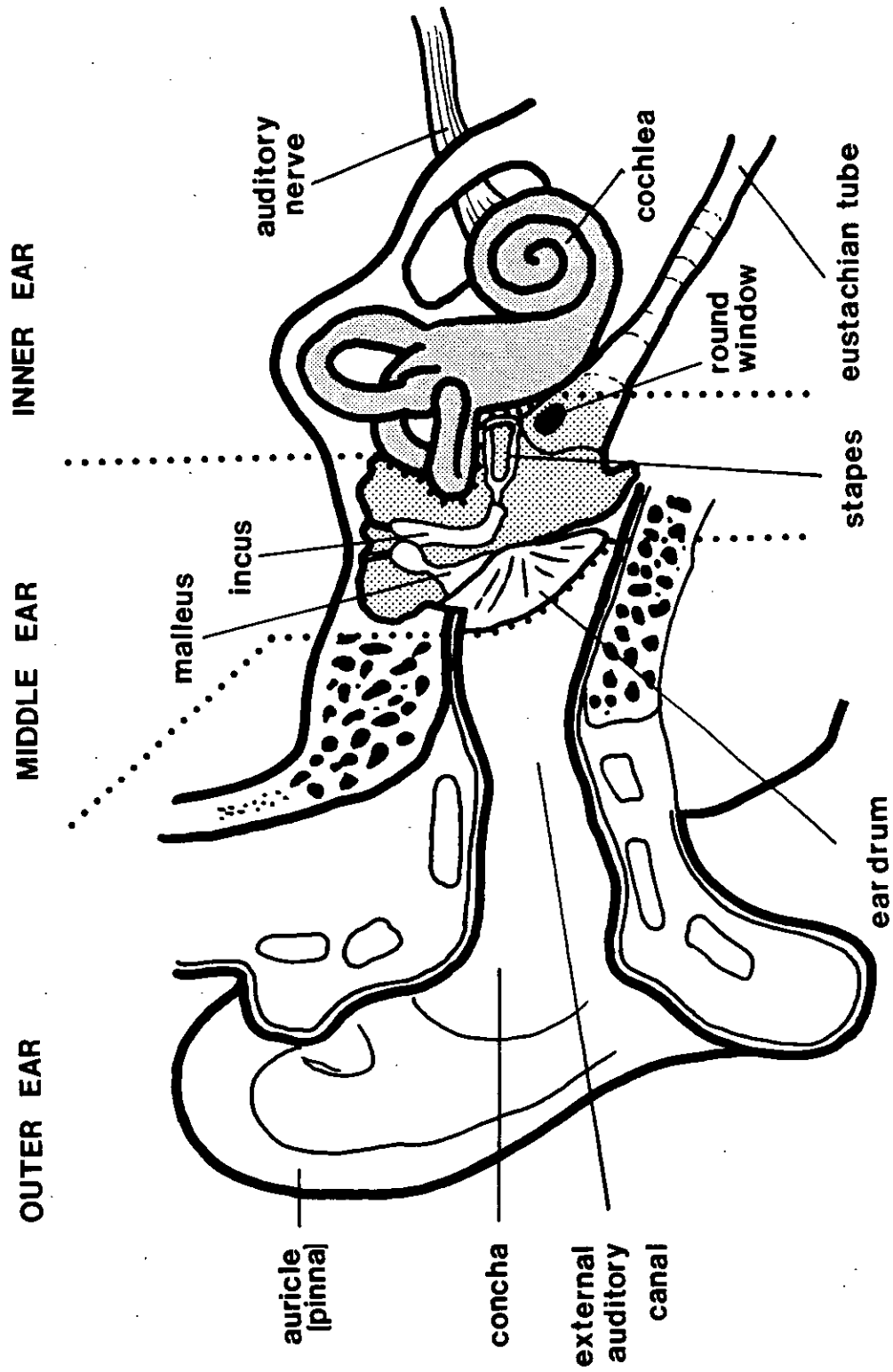
OUTER EAR MIDDLE EAR INNER EAR

auditory nerve

cochlea

eustachian tube

round window

stapes

incus

malleus

auricle [pinna]

concha

external auditory canal

ear drum

FIGURE 4. Diagrammatic representation of the auditory periphery

The outer ear

Structure

55   The outer ear comprises the auricle or pinna, the main auditory structure observable outside of the body, and the external auditory canal (Figure 4).

Development

56   The outer ear, the pinna, develops from the auricular hillocks situated around the margins of the first branchial groove (Moore 1988). Initial formation of the auricle commences around the 7th week of gestational age and reaches adult shape around the 20th week of gestation (Northern & Downs 1974). Adult size however is not reached until nine years of age (Northern & Downs 1974).

57   The external auditory canal is derived from the first branchial groove via the formation of a solid epithelial plate, the meatal plug, around the 8th gestational week (Moore 1988). During pregnancy this plug degenerates and by the 28th week of gestation forms a cavity, the external auditory meatus (Moore 1988).

Function

58   The function of the auricle is to collect sound and channel it into the ear canal. The human auricle performs this function most efficiently for frequencies above 5kHz. Sounds of lower frequencies have longer wavelengths than the auricle and may pass round it whereas higher frequencies, with shorter wavelengths, are unable to do so (Glattke 1978; Shaw 1974). This may play a role in the localisation of sound, but it is unlikely to function in the foetus.

59   The external auditory canal acts as a resonator. As the meatus is a tube, sounds within a certain frequency range, the resonant frequency, are reflected off the walls of the meatus reinforcing the acoustic energy within this bandwidth. For adults sounds between 1.5-7kHz will be enhanced, the peak increase occurring around 3kHz and resulting in a gain of 10-15dB

(Djupesland & Zwislocki 1973; Wiener & Ross 1946). Thus for broad-band sound reaching the ear canal, frequencies around 3-4kHz will be selectively amplified above other frequencies.

Functional differences between the foetal and adult outer ear

60   There are a number of differences between the outer ear of the foetus and that of the adult which may affect its function. The smaller size of the pinna means that higher frequencies than in adults will be 'captured' by the auricle (Shaw 1974). As the pinna grows in size it will capture progressively lower frequencies. Whether this role of the outer ear functions *in utero* remains to be determined.

61   The resonant qualities of the meatus are determined by its size, shape and compliance. In the foetus, as the canal is smaller in size than adults, its resonant frequency will be higher and it would thus amplify higher frequencies. The peak resonant frequency for newborns has been assessed at 3.8kHz compared to 3.0kHz for adults (Saunders, Kaltenbach & Relkin 1983). The foetus could be expected to possess an even higher peak resonant frequency. The fact that the canal is filled with fluid will also affect its resonant frequency. The gain in frequency is determined, in part, by the diameter of the canal and its compliance (in physical terms this may be related to the softness or rigidity of the ear canal). The diameter of the ear canal is smaller in the foetus than in the adult and the ear canal is much more compliant, i.e. is softer in the foetus and more rigid in the adult. Both of these factors act to reduce the gain in amplitude provided by the ear canal.

62   Taken together these factors indicate that the outer ear's influence on hearing in the foetus would be different to that observed in the adult. For a broad-band sound (20Hz-20,000Hz) it would be expected that the resonant frequency would be shifted towards a higher frequency than adults and the gain in this frequency range would be less. The extent of the frequency shift and the decrease in amplitude gain has yet to be determined.

63   Two other factors may also affect the functioning of the outer ear of the
      foetus. First, the time course of degeneration of the epithelial plug which
      results in the formation of the auditory meatus may affect the transmission
      of sound to the middle ear. The plug seems to have degenerated, and the
      meatus opened, by around the 28th week of gestation. Although there may
      be little impedance difference between the plug and surrounding amniotic
      fluid, some increase in sound transmission may occur following
      degeneration of the plug. Second, *in utero*, the outer ear will be filled with
      amniotic fluid. The effects of this will be considered in more detail in the
      next section.

The middle ear

Structure

64   The middle ear is a cavity, approximately two cubic centimetres in size,
      encased within the temporal bone (Figure 4). It lies between the tympanic
      membrane, which seals the external auditory meatus and middle ear, and the
      inner ear. The tympanic membrane is oval in shape. It is not flat but forms a
      concave cone pointing into the middle ear. The first of the three middle ear
      ossicles, the malleus, is attached to the tympanic membrane by its long
      process. The second ossicle, the incus, is joined to the malleus by an
      encapsulated joint with its long process extending at an acute angle away
      from the malleus. The long process of the incus attaches to the third ossicle,
      the stapes, by a very flexible joint. The ossicles are suspended in the middle
      ear cavity by ligaments attached to the walls and attic of middle ear cavity and
      to the ossicles. The footplate of the stapes fits into the oval window. There
      are two openings in the temporal bone which allow communication between
      the middle and inner ear; the oval window and the round window. The
      eustachian tube connects the middle ear cavity to the nasopharynx and, when
      open e.g. by swallowing or yawning, enables air to be drawn into the middle
      ear refreshing the air and equalising pressure.

65   The middle ear contains two small skeletal muscles attached to the ossicles,
      the tensor tympani and stapedius (Borg & Counter 1989). These muscles

function to dampen the vibration of the ossicles of the middle ear thus reducing the intensity of the acoustic signals reaching the inner ear. Contraction of the muscles is initiated by sound, contraction occurring approximately 100msec after the onset of a sound at threshold or 25/35msec after intense sounds or prior to speech (Borg & Counter 1989; Møller 1974). Normally only the stapedius responds to acoustic stimulation although if this stimulation is intense then the reaction involves both muscles (Møller 1974). This muscle reflex can thus protect the ear from exposure to loud sounds. On the presentation of a loud sound the stapedius contracts, moving the stapes by 50 microns, increasing the stiffness of the ossicular chain and reducing sound transmission by approximately 20dB (Borg & Counter 1989; Møller 1974). Although the muscles mature at about four months of gestational age the functional status of these muscles in the foetus is unknown.

Development

66   The middle ear cavity arises from the first pharyngeal pouch (Moore 1988). It commences development around five weeks of gestational age and continues to grow postnatally as the temporal bone enlarges (Behrman & Vaughan 1987). The stapes, the first of the ossicles to form, is derived from the second branchial arch and can be seen during the 5th week of gestational age. It is completely formed by 22 weeks of gestational age (Anson & Cauldwell 1941). The incus and malleus are derived from the first (mandibular) branchial arch and can be seen during the 6th week of gestation and reach adult form around the 22nd week of gestation (Anson, Bast & Cauldwell 1948). Ossification of the middle ear bones occurs during the 16th-25th week of gestational age (Bast & Anson 1949) and adult size is reached by the eighth month of gestation (Anson & Donaldson 1981; Moore 1988).

Function

67   The function of the middle ear is two-fold. First, it transfers sound energy to the fluid-filled inner ear where it is transduced by the auditory sensory receptors into neural impulses. This is achieved by the action of the middle ear bones. Vibrations in the tympanic membrane are transmitted via the

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malleus to the incus and on to the stapes resulting in a piston like movement of the stapes footplate at the oval window. This motion produces pressure changes in the fluid of the inner ear. The mechanical responses of the middle ear throughout its frequency range are virtually linearly related to the sound pressure at the tympanic membrane (Møller 1972).

68   The second function of the middle ear is to overcome the impedance differences between the air-filled outer ear and fluid-filled inner ear. As mentioned earlier the difference in impedance between the fluid medium of the inner ear and the air medium of the outer ear would result in a significant loss in sound energy as it passed from the outer to the inner ear. The middle ear overcomes the differences in impedance between the air and inner ear by increasing the effective pressure of sound energy striking the tympanic membrane. Two factors increase the acoustic energy striking the inner ear. First, the difference in area between the tympanic membrane and stapes footplate results in a 17:1 increase in mechanical energy (Glattke 1978). Second, the malleus-incus joint forms a lever which results in an increase in mechanical energy by a factor of 1.3:1 (Glattke 1978). In combination these actions increase the sound pressure by 22:1, or approximately 27dB, hence overcoming the impedance mismatch.

69   The middle ear cavity itself has an effect on the transmission of acoustic energy to the inner ear. The small amount of air in the middle ear cavity, when sealed behind the tympanic membrane, has a certain impedance that is determined by its 'stiffness'. Given that this small quantity of air is much less compressible than the air outside of the ear, i.e. in the environment, it will favourably admit higher frequencies compared to lower frequencies. Thus for a broad-band sound the low frequencies will be differentially attenuated compared to other, higher, frequencies (Glattke 1978).

Functional differences between the foetal and adult middle ear
70   There are a number of maturational differences between the foetal and adult middle ear which may affect its function. The middle ear bones are ossifying

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during pregnancy and becoming more rigid. Their position relative to one another changes during maturation. These may affect both the transfer ability of the middle ear and the gain in signal provided by the lever action of the ossicles.

71   The size of the tympanic membrane is smaller in the foetus thus decreasing the size difference between the tympanic membrane and oval window (Ballenger 1969). This may reduce the gain in signal provided by the middle ear.

72   The size and rigidity of the middle ear cavity will change affecting its tuning and filtering abilities. However as the middle ear cavity will be filled with fluid it is doubtful whether any effect is exerted by the middle ear cavity on sound transmission in the foetus.

73   The most important difference between the foetal middle ear and adult middle ear is that after birth the middle ear is an air-filled cavity whereas in the foetus it is fluid-filled. After birth if the middle ear fills with fluid this results in an increase in impedance and an increase in hearing thresholds. The presence of fluid in the foetal ear may have implications for both the transfer function of the middle ear and its amplification function.

74   One role of the middle ear is to overcome the impedance mismatch as sound energy passes from the air to the fluid-filled inner ear. For the foetus, however, once sound has entered the womb from the external environment, sound transmission is essentially all in fluid, i.e. from the amniotic fluid of the womb to the fluid of the inner ear. As the intra-uterine environment is essentially one of uniform impedance sound energy, once in the abdomen, will pass directly to the inner ear with little loss in energy. The impedance mismatch present after birth at the middle:inner ear interface has essentially been moved to the air:maternal abdomen interface.

75   Whether the ossicles of the middle ear function *in utero* to transfer sound energy from the tympanic membrane to the oval window has yet to be determined. Sound energy *in utero* will cause vibrations of the tympanic membrane and consequently the middle ear ossicles. Maximum motion of the tympanic membrane is achieved when there is equal pressure on either side, i.e. in the auditory meatus and the middle ear cavity (Møller 1972). After birth this pressure equalisation is maintained by the opening and closing of the eustachian tube. Although both the auditory meatus and middle ear cavity are fluid-filled in the foetus, the foetus swallows and yawns regularly from as early as 16 weeks of gestational age (Prechtl 1988) and this may ensure pressure equality between outer and middle ear. This ensures maximum motion of the tympanic membrane in response to sound energy. The middle ear ossicles thus have the potential to transmit sound energy to the inner ear and presumably to amplify the sound. The fact that the middle ear cavity is fluid-filled may affect this transmission.

76   In the air-filled middle ear when the motion of the stapes footplate on the oval window causes a movement of the oval window there is a corresponding, but opposite, displacement of the round window into the middle ear cavity. When the middle ear is filled with fluid the action of the round window may be affected which in turn may affect the transmission of sound energy within the inner ear.

77   In summary the foetal middle ear shows a number of differences from the adult middle ear which may result in different functional characteristics for the middle ear of the foetus. Exactly how the outer and middle ear function when fluid-filled is unknown. However the differences are sufficient to suggest that outer and middle ear function is different before and after birth.

The inner ear

Structure

78   The inner ear is found in the temporal bone and houses the sensory organs for both hearing, the cochlea, and balance, the labyrinth (Figure 5).
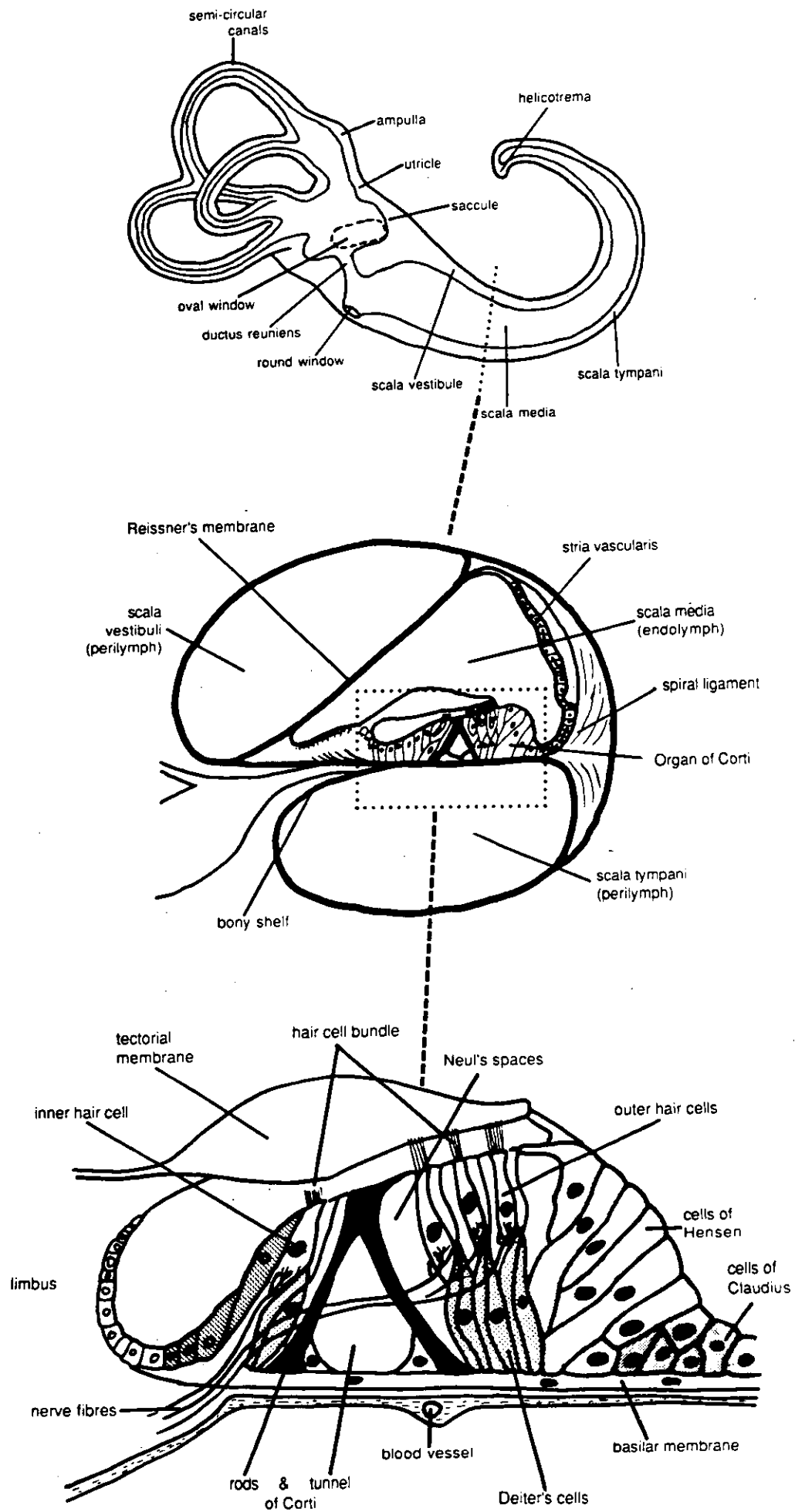
FIGURE 5. Diagrammatic representation of the inner ear; a) the cochlea, b) cross-section across the cochlea, c) Organ of Corti

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79   The inner ear is essentially constructed from five channels in the temporal
       bone (Figure 5). The bony labyrinth contains three semi-circular canals, the
       vestibule and the utricle. The fifth channel forms the cochlea, and it is here
       the sensory organ for the detection of auditory stimuli, the Organ of Corti, is
       found.

80   The cochlea is spiral in shape, like a snail's shell, and coiled approximately
       two-and-one-half times. The cochlea is divided by the membranous
       labyrinth into three ducts (Figure 5). The scala vestibuli and the scala
       tympani are both filled with perilymph (a fluid high in sodium and low in
       potassium, typical of most extracellular fluids). At the apical end of the
       cochlea the scala vestibuli and scala tympani are connected by the
       helicotrema. The round window is situated between the middle ear cavity
       and the perilymph in the scala tympani. The third duct, the scala media or
       the cochlea portion, is wedge shaped and is filled with endolymph a fluid of
       high potassium and low sodium concentration and unique amongst
       extracellular fluids. The scala media is bounded by Reissner's membrane,
       which forms its upper boundary (and the lower boundary of the scala
       vestibuli), and by the basilar membrane which forms its lower boundary (and
       the upper boundary of the scala tympani).

81   The Organ of Corti containing the sensory cells, the hair cells (about 16,000 in
       each ear) and related support structures, is situated on top of the basilar
       membrane. The tectorial membrane, a gelatinous structure, covers the Organ
       of Corti. The Organ of Corti contains two sensory-receptor cells involved in
       sensory encoding of auditory signals, a single row of inner hair cells and three
       parallel rows of outer hair cells. The outer hair cells are attached to the
       tectorial membrane and basilar membrane. The inner hair cells are attached
       to the basilar membrane only.

82   The hair cells consist, very basically, of a body and a hair cell bundle
       (stereocilia) on the top of the body. The inner hair cells are the primary
       sensory cells of the auditory nerve and are present in a single row in each

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cochlea and are responsible for relaying sensory information to the central nervous system. The outer hair cells have little afferent innervation and are responsible for actively tuning the sensitivity of the basilar membrane and hence the inner hair cells response to stimulation.

Development

83   The inner ear is the first of the three parts of the auditory periphery to form. The inner ear arises from the ectodermal otic placode which forms around the 23rd day of gestation, this invaginates to form the otocyst (Moore 1988; Streeter 1917). The differentiation of the vestibular and cochlea end organs from the otocyst occurs during the second month of gestation (Moore 1988). The cochlea exhibits a complete coil by eight/nine weeks of gestational age and the Organ of Corti emerges one week later (Pujol & Lavigne-Rebillard 1985). Development of the cochlea proceeds from base to apex (Pujol & Hilding 1973). By 20 weeks of gestational age Kollicker's organ has regressed releasing the tectorial membrane, the tunnel of Corti has opened and Neul's spaces are formed (Pujol, Lavigne-Rebillard & Uziel 1991). At this stage the morphological appearance of the cochlea is similar to other mammals where function has been established (Pujol & Hilding 1973).

84   The hair cells commence differentiation at 10-11 weeks of gestational age, innervation of the undifferentiated epithelium having occurred one week earlier (Pujol, Lavigne-Rebillard & Uziel 1991). The inner hair cells mature earlier than outer hair cells. The innervation of the inner hair cells begins at 12 weeks of gestational age and the initial pattern of innervation is similar to that seen in adults (Pujol, Lavigne-Rebillard & Uziel 1991). The later stages of synaptogenesis involve the outer hair cells which receive efferent innervation around week 20 of gestation, with mature synapses appearing between 24-28 weeks of gestational age.

85   The stereocilia of the hair cells start developing at 11-13 weeks of gestational age. Those of the inner hair cells developing before those of the outer hair cells and basal hair cells before apical hair cells. The stereocilia of the hair

cells have obtained the adult pattern by 22 weeks of gestational age. Initially the outer hair cells appear more numerous in the foetus than in the adult indicating some degenerative process may occur (Lavigne-Rebillard & Pujol 1986).

86   The normal development of the tectorial membrane, detectable by 10 weeks of gestational age, is essential for the regulation and ordering of stereocilia and in particular the attachment of the outer hair cells to the tectorial membrane (Pujol & Lavigne-Rebillard 1985).

87   Overall the developmental appearance of the cochlea at 20 weeks of gestational age has reached a stage comparable to that in animals at which a response to sound can be readily evoked (Pujol, Lavigne-Rebillard & Uziel 1990). However exactly when the cochlea has completed its development has yet to be determined.

Function

88   The inner ear essentially serves two main functions: the transduction of the acoustic energy into neural impulses by the sensory receptor cells; and second the encoding of certain auditory information present in the acoustic signal, e.g. frequency and intensity. How the inner ear achieves these functions has attracted a considerable amount of research. There now follows a very abbreviated account of our knowledge of how the inner ear works. A number of recent reviews have examined all aspects of inner ear function and readers are referred to these for further information (Abbas 1988; Ashmore 1991; Cazals, Horner & Demany 1992; Dallos 1988; Dallos & Corey 1991; Holley 1991; Hudspeth 1989; Musiek & Hoffman 1990; Pattuzzi & Robertson 1988; Pujol 1990; Teas 1989).

89   The movement of the stapes footplate on the oval window results in motion of the fluid in the scala vestibuli which leads to displacement of basilar membrane and Organ of Corti. This movement against the cochlear partition, due to a series of hydraulic phenomena, results in a 'wave-like'

motion along the basilar membrane as described by von Békésy (1960). This wave progresses from the base to apex of the basilar membrane.

90   An auditory stimulus, e.g. a pure tone, evokes a wave moving from the base of the basilar membrane to its apex. The wave, as it moves along the membrane, increases in amplitude until reaching a peak and then decreases abruptly in amplitude. Von Békésy initially described this motion as a travelling wave (1960), however the observed motion may represent either a travelling wave as proposed by von Békésy or the resonance of a series of resonators as initially proposed by von Helmholtz (1863). However the observed motion may be accounted for the fact remains that different frequencies peak in amplitude at different points along the basilar membrane. High frequencies peak in amplitude near the base of the basilar membrane, whilst low frequencies travel further along the basilar membrane and peak in amplitude nearer the apex. The peak amplitude of different frequencies is arranged monotonically along the basilar membrane, from high frequencies at the base to low frequencies at the apex.

91   The initial observations of the motion of the basilar membrane by von Békésy, performed on cadavers, found that for any particular single frequency, a wide area of the basilar membrane responded to, and was displaced by, the stimulus. This observation of basilar membrane motion was at odds with electrophysiological recordings obtained of frequency tuning curves from auditory nerve fibres. Recordings from single nerve fibres in the auditory nerve revealed each fibre responded best to a very limited range of frequencies. Plotting the sound energy required to elicit a response revealed that single nerve fibres are very highly tuned having a very narrow bandwidth and a very sharp 'cut off'. Response characteristics which were much more highly tuned than the motion of the basilar membrane would suggest. Authors were thus lead to propose the existence of a 'second filter' which further tuned the response of the basilar membrane (Evans & Wilson 1973).

92   The introduction of new techniques which enabled the observation of basilar membrane motion in cochleas of physiologically good condition in live animals, rather than the cadavers used by von Békésy, revealed that the mechanical response of the basilar membrane was in fact highly tuned and exhibited response characteristics equal to those observed in the auditory nerve fibres (e.g. Khanna & Leonard 1982; Sellick, Patuzzi & Johnstone 1982). This sensitivity and selectivity of the basilar membrane motion is the result of the action of one of the two types of hair cells present in the cochlea, the outer hair cells.

93   As a sound stimulus elicits motion in the basilar membrane the outer hair cells are excited in the same fashion as the inner hair cells (via displacement of their stereocilia) but respond in a very different way. These outer hair cells contract very rapidly in phase with the frequency of the stimulus. The outer hair cells have there longest cilia attached to the tectorial membrane and thus are anchored at their top to the tectorial membrane and at their base to the basilar membrane. The rapid contraction of these cells results in amplification and tuning of the vibration of the basilar membrane. This results in a much smaller portion of the basilar membrane vibrating and the observed motion more closely represents the electrophysiological recordings made from the auditory nerve fibres. Thus the action of the outer hair cells results in an active mechanism superimposed on the passive motion of the basilar membrane which highly tunes the response of the basilar membrane to sound stimuli.

94   Whilst the outer hair cells are important for normal hearing the inner hair cells are crucial for all hearing. This single line of sensory receptors, in each ear, transduce the acoustic energy into neural impulses. The hair cell has a bundle of stereocilia, not attached to the tectorial membrane like those of the outer hair cells, protruding from its apical end which respond to the mechanical energy. The very detailed and precise micro-structure of the hair cell and stereocilia is crucial for this response (Hudspeth 1989). The stereocilia act as a biological strain gauge, mechanical stimuli deflect the

stereocilia which open ion channels in the hair cell membrane. This alters the membrane potential which in turn affects the release of synaptic transmitter from the hair cell to stimulate the nerve. The action of the outer hair cells in tuning the basilar membrane results in a small number, perhaps even just one, of the inner hair cells being stimulated with maximum sensitivity.

95   As well as transducing the acoustic energy into neural impulses the inner ear also encodes certain aspects of the auditory signal. Two separate theories have been proposed to explain the encoding of frequency; the place principle and the volley principle.

96   The site of maximum displacement of the basilar membrane is frequency dependent. Frequencies are arranged monotonically along the basilar membrane from high frequencies at the basal end to low frequencies at the apical end. Thus this affords one possible means of encoding frequency information, i.e. by the site of maximum basilar membrane displacement. This has become known as the place principle (von Békésy 1960).

97   A second means of encoding frequency is in the firing pattern of the auditory nerve fibres. Encoding of frequency in neural firing was proposed by Rutherford (1886). One problem with this approach is that the maximum rate of firing of the auditory nerve fibres is about 1000 times per second. Thus frequencies above 1000Hz could not be encoded. One solution to this was suggested by Wever (1949) who proposed that higher frequencies could be encoded if the nerve fibres acted together as a group. Although a single fibre is limited by its refractory period a group of fibres could work together to encode higher frequencies. This has become known as the volley principle, and has been supported by the observation of phase locked firing of nerve fibres (e.g. Rose, Brugge, Anderson & Hind 1967).

98   Both mechanisms act to encode frequency, but in different parts of the frequency range. Below 1000Hz frequency is encoded in the firing pattern of

the auditory nerves. At these low frequencies the displacement of the basilar membrane is very broad, however phase locking of auditory nerve firing is easily achieved. Above 4000Hz frequency is encoded by the place principle. At these higher frequencies the ability to phase lock is lost but the displacement of the basilar membrane is highly tuned. Between 1-4kHz both mechanisms may operate together.

99   The detailed mechanics of the action of the inner ear in the neural encoding of sound stimuli is beyond the scope of this review and not necessary. What is important is an understanding that the transduction of sound energy into neural impulses by the inner ear is the result of a complex interaction between the sensory cells, both the inner and outer hair cells, their supporting structures, the basilar membrane and the tectorial membrane. The result of which is a system capable of extremely fine frequency resolution enabling the processing of complex sounds such as speech. Changes which affect the functioning of *any* of these parts may affect the ability of the ear to sense and resolve auditory stimuli.

Functional differences between the foetal and adult inner ear

100   The inner ear, unlike both the outer and middle ear, shows few differences between the foetus and the adult. Obviously there will be maturational differences in the functioning of the various parts of the inner ear but, once mature, it will function similarly to that observed in adults. The inner ear is fluid-filled before and after birth and there is no evidence to suggest that the composition of the fluid, either the perilymph or endolymph, changes. The composition of the cochlear fluids, particularly of the endolymph, is thought to be important for the functioning of the inner ear. Any changes in the composition of these fluids may affect the functioning of the hair cells. Viscosity changes may alter the shear characteristics induced in the perilymph and endolymph by mechanical energy which may in turn affect the stereocilia's response to the stimuli and affect the neural response. Maturational changes, in particular the ontogeny of outer hair cell function, may affect the efficiency of inner ear function but there has been little

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research into the performance of inner ear *in utero*, thus any effect of these changes is unknown.

The central auditory pathway

101 It is not intended to discuss the structure or function of the central auditory pathway here since this is not directly affected by noise exposure but only secondary after damage to the auditory periphery (Saunders, Cohen & Szymko 1991). Readers are referred to Paxinos (1990) and Abbas (1988) for reviews. One point, however, is worthy of note. It appears that at all levels of the auditory system efferent neural pathways are provided for feedback control and influencing afferent neural activity. This efferent influence upon the afferent neural system may have a role in improving signal detection or resolution. It may also have a role in preventing overstimulation from intense sounds (Filogamo, Candiollo & Rossi 1967; Rajan 1992). Although the role of the efferent system has yet to be fully established, knowledge of the functional development of this activity *in utero* is obviously important if it has some role in protecting the auditory system from overstimulation. If not functioning fully in the foetus its auditory system may be more at risk to the effects of noise.

<u>Summary</u>

102 The auditory system thus becomes functionally mature during the middle of gestation. The auditory system of the foetus however exhibits a number of differences to that of the individual after birth which may affect its functional characteristics. Extreme caution must therefore be exercised when extrapolating studies performed on the mature ear with air-borne sound to the foetus whose auditory system is maturing and where sound is carried in fluid. Whether the auditory system of the foetus is stimulated however depends upon it receiving appropriate stimulation. This is considered in the next section.

SECTION V

THE SOUND ENVIRONMENT OF THE FOETUS

103   The foetus, existing within its mother body, will experience a very different sound environment to that of individuals after birth. A number of attempts have been made to document the sound environment of the foetus to assess a) what sounds the foetus will be exposed to in the womb and b) what effect the maternal abdomen has on the sound stimulus as it passes from the external to the intra-uterine environment.

The intra-uterine sound environment

104   Although it was initially thought that the foetus lived in a sensory void (see Hepper 1992), the internal intra-uterine environment of the mother contains a number of potential sources of sound. The maternal heartbeat, noise from the digestive system and the maternal voice may all stimulate the ear of the foetus. Investigators have attempted to measure the sound levels produced by these noises.

105   Initial studies to assess the sound environment of the foetus reported a high level of internal noise. Bench (1968) reported a background noise level of $72dB_{spl}$, recorded from a microphone placed in the vagina of a woman at 37 weeks of gestation. Grimwade, Walker & Wood (1970) implanted a microphone in the cervix of pregnant women at term and recorded an internal noise level of $85dB_{spl}$ between 20-300Hz, but rising to $94dB_{spl}$ with the maternal heartbeat. Henschall (1972) used a phonocatheter to determine sound levels after rupture of the membranes and recorded a sound energy level of 57dB (ref $10^{-16}W$). These studies used an air-coupled microphone to record sound levels *in utero* which is not appropriate for measurements in fluid. More recent studies have used a hydrophone (more suited for use in fluid) to record sound levels and these have produced a slightly different picture of the background noise levels in the womb.

106   Using a hydrophone implanted in the uterus at the beginning of labour after amniotomy Querleu, Renard & Crèpin (1981) and Querleu, Renard, Versyp, Paris-Delrue & Crèpin (1988) recorded internal background noises from 1-1000Hz, with an average sound pressure level of 28dB(A) near the placenta and 15dB(A) further away from the placenta. The high intensity (85-90dB) sounds recorded in previous studies were observed but restricted to low frequencies below 32Hz. As such these may not be perceived by the foetus. Sounds of the mother's heartbeat or borborygmi were observed and had a sound level of 25dB above that of the background noise.

107   These studies demonstrate that the environment of the foetus is far from quiet. However these reports have been restricted to recording sound levels at the end of pregnancy. The sound levels during the early stages of pregnancy are at present unknown.

108   One point of note from these studies regards the high intensity low frequency sounds. Since these form a natural part of the foetal auditory environment it may be expected that external sounds of a similar intensity may have little harmful effect on the foetus.

The intensity of external sounds _in utero_

109   The external environment is rich in auditory stimulation and a number of researchers have attempted to determine how much of this reaches the foetus. The main aim of these studies has been to document the attenuation of sounds provided by the maternal abdomen.

110   Bench (1968), using a microphone placed in the vagina of a woman near term, recorded attenuation of 19dB for tones of 200Hz, 24dB at 500Hz, 38dB at 1000Hz and 48dB at 2000/4000Hz. Sounds in this study were produced by a speaker placed just above the surface of the abdomen. Walker, Grimwade & Wood (1971) found very similar patterns of attenuation 30dB at 50Hz and 100Hz, 33dB at 500Hz, 40dB at 1kHz, 51dB at 2kHz and 62dB at 3kHz using a microphone placed in the cervix of pregnant women near term. In this study

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a speaker was used to produce frequencies 200Hz and above and a vibrator for the 50Hz and 100Hz frequencies. The results from these latter two frequencies may not be directly comparable to the other frequencies as the device was in contact with the skin and may have also produced a vibrational component. As with studies assessing intra-uterine noise levels, the use of an air-coupled microphone may have adversely affected the accurate recording of the attenuation factors of maternal abdomen.

111   More recently studies have used a hydrophone to record maternal attenuation. Querleu, Renard & Crèpin (1981) and Querleu, Renard, Versyp, Paris-Delrue & Crèpin (1988) suggest that attenuation is low for low frequencies and higher for high frequencies, reporting attenuation of 2dB(A) at 250Hz, 14dB(A) at 500Hz, 20dB(A) at 2000Hz and 20-40dB(A) at 3.8-18kHz. These studies suggest that low frequency sounds pass relatively unattenuated through the maternal abdomen in terms of their acoustic energy whereas higher frequency sounds lose most of their energy.

112   A very different approach to the assessment of the attenuation provided by the abdomen was reported by Szmeja, Slomko, Sikorski & Sowinski (1979). Here the authors recorded the intensity of sound produced by the heartbeat. They first recorded the intensity of the heartbeat of the foetus *in utero* by placing a microphone on the maternal abdomen and repeated the measurement after the individual was born, placing the microphone over the heart of the newborn. An attenuation factor of 10.4dB at 31.5Hz, 10.7dB at 63Hz, 14.9dB at 125Hz and an average attenuation of 11.5dB over frequencies 20Hz-20kHz was reported.

Summary

113   Despite differences in recording techniques the overall pattern of results is similiar for all studies, showing much less attenuation of low frequency sounds than for high frequency sounds. It may be expected that for a broad-band sound low frequencies (250Hz and below) would pass relatively unattenuated into the abdomen whereas higher frequencies would be more

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attenuated. Two factors are worthy of note here. First, recordings were made after the amniotic sac had burst and sound levels may be different within the intact amniotic sac. Second, the observations were made at the end of pregnancy. Given the very real changes in maternal size which would affect the impedance of the abdomen, a different pattern of attenuation may be observed earlier during the pregnancy.

114   Finally it should be remembered that these results indicate the effects of the maternal abdomen on sound as it passes through the skin and are not a measure of the sound levels at the inner ear. In order to determine the sound intensity at the hair cells it is necessary to combine the evidence from studies examining the foetal sound environment and that relating to the structure and functioning of the foetal ear. This is attempted in the next section.

SECTION VI

SOUND AT THE FOETAL INNER EAR

115   Having discussed both the quality of the foetal sound environment and the functional maturation of the auditory periphery what can be said about how these effect noise from the external environment as it passes to the foetal ear?

116   First, and perhaps the greatest change that will occur, is as sound passes from the external environment through the maternal abdomen. The sound will be attenuated. The acoustic energy of the lower frequencies (less than 250Hz) of the sound will pass through relatively unattenuated but the acoustic energy contained in high frequencies will be more attenuated.

117   Once in the womb the sound energy will proceed relatively unattenuated to the inner ear. As discussed earlier, the internal environment of the abdomen will be relatively uniform and thus sound energy will pass directly to the inner ear once in the abdomen. This provides a direct route to stimulate the inner ear.

118   The action of the outer and middle ear *in utero* is unknown but if functioning they would differentially amplify frequencies present in a broad-band signal. The salience of sounds in the higher frequency range would be increased.

119   Sound energy may be passed to the inner ear via the mechanical action of the middle ear ossicles. The gain in amplification provided by this route of stimulation will not be as great as for the adult.

120   The inner ear may also be stimulated by bone conduction (Tornndorf 1972). Although the bone of the skull is only just forming, the acoustic energy in the fluid and tissue may create vibrations in the bone. This may stimulate the inner ear by similar means as occurs in adults. The decreased rigidity of the cranial bones in the foetus may mean that lower frequencies than in

adults will be conducted by the bone.

121   There may also be another route which provides stimulation for the inner
       ear. The perilymph of the inner ear is confluent with the cerebrospinal fluid
       via the cochlear duct. Acoustic energy may be transmitted from the uterine
       environment to the cerebrospinal fluid in the spinal cord or ventricles. As
       this fluid is confluent with fluid of the inner ear acoustic energy may be
       transferred to the inner ear and stimulate the hair cells. In adults no
       stimulation reaches the ear this way due to the high energy loss first from the
       air:body interface and secondly from the shielding provided by the bony
       spinal cord and head. In the foetus there will be much less of an impedance
       mismatch since sound transmission is essentially all in fluid. Further, the
       spinal cord and head have not yet ossified and this will greatly reduce the
       shielding effect of these bones enabling easier transmission of energy to the
       cerebrospinal fluid. This opens the intriguing possibility that stimulation of
       the inner ear may be achieved by this route. Indeed this would be a highly
       efficient means of transmitting acoustic energy to the inner ear.

122   The abdomen, and uterus, will have a certain resonant frequency and sound
       within this frequency range would pass relatively unattenuated into the
       abdomen and may possibly be enhanced. Examinations of the resonant
       frequency of the maternal abdomen to whole body vibration indicate this to
       be around 3-9Hz in late pregnancy (Clark, Lange and Coermann 1962).
       Although below the range of adult hearing, sounds in this frequency range
       could be expected to be enhanced increasing their intensity in the womb.
       Note also the similarity of the abdomen and womb to a Helmholtz resonator,
       a device capable of greatly increasing the intensity of sounds within its
       resonant frequency.

Summary

123   Thus sound energy may be transmitted to the inner ear of the foetus in a
       different way to that observed in adults due to the different functioning of the
       foetal outer and middle ear. Moreover sound may reach the ear by routes

unavailable to the adult, and sound within the resonant frequency range of the abdomen would be enhanced. Acting together these may possibly increase the amount of acoustic energy reaching the inner ear.

124   One fact from this review becomes clear, the functioning of the auditory system in the foetus is largely unknown. Direct comparison with the functioning of the auditory system after birth is inappropriate. Whether the foetal auditory system does function will be discussed in the next section. One other fact, however, should be considered in any attempt to model foetal auditory system functioning or its sound environment. That is, the system is not static, but developing. Over the course of pregnancy not only does the auditory system develop but the abdominal environment in which the sound travels to reach the ear changes, together these interact to affect external sounds reaching the inner ear. Thus, any attempts to describe foetal audition have to acknowledge this dynamic component.

SECTION VII

THE RESPONSE OF THE FOETUS TO SOUND

125   There is now much evidence that the foetus responds to externally presented sound or auditory stimuli. This section examines current knowledge regarding foetal hearing abilities.

126   At the turn of this Century there was much debate concerning whether the newborn infant could hear or was born deaf, little consideration was consequently given to the auditory abilities of the foetus. The earliest reports of foetal hearing appeared in the mid 1920's. Peiper (1925) reported that during late pregnancy the foetus moved in response to the noise of a car horn sounded in front of the maternal abdomen. Forbes & Forbes (1927) reported that a woman, near term, noted an increase in the movements of her foetus whilst lying in a bath of water when the side of the bath was struck with a metal funnel. Since this time there has been a plethora of studies examining the response of the foetus to sound.

127   A variety of sound sources have been used to provide auditory stimuli; loud speakers (Goodlin & Schmidt 1972; Hepper & Shahidullah 1992), electric toothbrushes (Leader, Baille, Martin & Vermeulen 1982), vibroacoustic stimulators (Crade & Lovett 1988), bone vibrators (Jensen & Flottorp 1982), car horns (Peiper 1925) and door buzzers (Prenzlau & Hoffman 1982) have all been used. This has resulted in a wide range of stimuli being presented to the foetus; pure tone sine waves (Hepper & Shahidullah 1992), saw-tooth waves (Boos, Gnirs, Auer & Schmidt 1987), pink noise (Lecanuet, Granier-Deferre, Cohen, le Houezec & Busnel 1986), phonemes (Lecanuet, Granier-Deferre, DeCasper, Maugeais, Andrieu & Busnel 1987) and music (Hepper 1991). Stimulus durations ranging from 0.5 second (Kuhlman, Burns, Depp & Sabbagha 1988) to 60 seconds (Gelman, Wood, Spellacy & Abrams 1982) have been used. Stimuli have been presented singly (Gagnon, Hunse, Carmichael, Fellows & Patrick 1986) or in a series of pulses (Luz 1985). Furthermore a wide range of intensities from 80dB to 120dB have been used. The

comparison of intensity levels between studies is difficult due to the different methods of assessing intensity that have been used. For example, sound intensity has been determined at the speaker face or up to one meter away from the sound source (Luz 1985; Druzin, Edersheim, Hutson & Bond 1989). Whilst many studies have examined the response of the foetus to sound the lack of <u>systematic</u> examination of foetal hearing has resulted in the development of foetal hearing abilities remaining largely unknown. Based on the studies which have been conducted the following attempts to summarise the current knowledge of hearing abilities of the foetus.

Development of hearing

128   Studies examining the development of foetal hearing are based upon observing a discrete response on the part of the foetus to the presentation of a sound stimulus. The foetus first appears to respond to sound around 24 weeks of gestational age. Using an eye-blink response foetuses were observed to "blink" on the presentation of a sound produced by a vibroacoustic stimulator placed on the mother's abdomen, over the foetus's head (Birnholz & Benacerraf 1983). Similarly, using an electric toothbrush as the sound stimulus, the foetus was first observed to move from 23-24 weeks of gestational age in response to the vibratory motion of the toothbrush when placed on the abdomen (Leader, Baille, Martin & Vermeulen 1982). It should be noted that in both these cases the devices used to produce the sound also produced a vibration. A recent study, using pure tone sine waves produced by a speaker, found evidence of responsiveness to sound at 20 weeks of gestational age, but the response was much more diffuse than the contingent specific response seen at 25 weeks of age reported above (Shahidullah 1993; Shahidullah & Hepper 1993).

129   One fact should be borne in mind when considering the onset of hearing. The evaluation of hearing in these experiments is made by observing a response exhibited by the foetus. For the foetus to respond it requires both a functioning motor system and a neural link between the sensory (auditory) system and motor system in order for the response to be emitted. It is entirely

possible that the auditory system is able to sense stimulation before the motor system is able to respond.

Frequency and intensity

130   The foetus has been shown to respond to frequencies ranging from 83Hz (Madison, Adubato, Madison, Nelson, Anderson, Erickson, Kuss & Goodlin 1986) to 5,000Hz (Lecanuet, Granier-Deferre & Busnel 1988) during the last trimester of gestation.

131   In a recent study (Shahidullah 1993), using pure tone stimuli, it was found that the foetus first responded to frequencies of 500Hz and 250Hz at around 24/25 weeks of gestational age compared to frequencies of 1000Hz and 3000Hz to which the foetus responded to around 29-31 weeks of gestational age. Thus it appears that the foetus responds first to tones in the low frequency portion of the adult hearing range and, as the foetus matures, it responds to increasingly higher frequencies.

132   The ability to discriminate between different stimuli has also been demonstrated in the foetus during the last trimester of pregnancy using habituation paradigms. In these studies the foetus is repeatedly exposed to the same stimulus until it no longer responds. It is then presented with a new stimulus and its response observed. If it responds to the presentation of the new stimulus it can be argued that the foetus detects a difference between the two stimuli. Using a body movement Hepper & Shahidullah (1992) habituated foetuses (35-36 weeks of gestational age) to a 250Hz pure tone sine wave and re-elicited responding by presenting a 500Hz pure tone sine wave. The study indicates that at this age the foetus can discriminate between 250Hz and 500Hz tones. Lecanuet, Granier-Deferre, DeCasper, Maugeais, Andrieu & Busnel (1987) found that foetuses (35-38 weeks of gestational age) could discriminate between two speech like sounds [babi] and [biba]. Here, after exposure to one of the stimuli, it was found that the presentation of the novel speech sound elicited a deceleration in the heart rate of the foetus. Thus the foetus is capable of quite sophisticated auditory discriminations.

133   Extending this research Shahidullah and Hepper (1993) found that foetuses at 35 weeks of gestation could discriminate between a 250Hz tone and a 500Hz tone, and between speech sounds [ba] and [bi], but fetuses aged 27 weeks of gestation were unable to make either discrimination. It was argued that the delayed maturation of the outer hair cell function may account for the lack of discrimination at 27 weeks of gestation.

134   The foetus is also capable of responding to differences in intensity during late gestation. Increasingly intense sounds have been shown to elicit a greater reaction (assessed by heart rate response, foetal movement, or maternal perception of movement) on the part of the foetus, during the last third of gestation (Kisilevsky, Muir & Low 1989; Yao, Jakobsson, Nyman, Rabaeus, Till & Westgren 1990).

135   The response of the foetus is determined by its particular behavioural state at the time of stimulation, different intensities and durations of reaction are observed in different behavioural states (Lecanuet, Granier-Deferre, Cohen, le Houezec & Busnel 1986). The effect of state however probably does not reflect a change in the responsiveness of the foetal auditory periphery but rather a more central change in the reactivity of the CNS.

Summary

136   There is no doubt that the foetus responds to external auditory stimuli from as early as 20 weeks of gestational age and possibly even earlier. Later during pregnancy the auditory system is well enough developed to enable the discrimination between different sounds. Since the auditory system functions prenatally it may also be susceptible to damage by overstimulation and this is considered in the next section.

137   One question which should be addressed is whether the foetus is 'hearing' in the womb?, i.e. is the response observed mediated by the auditory system? The indications of foetal hearing are derived from the external presentation

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of an auditory stimulus and the exhibition of some response on the part of the foetus. The assumption being that as sound was 'put into the system' then it must be this that the foetus is responding to (Hepper 1992). However it is possible that the response could be mediated by other sensory systems, e.g. the cutaneous sense which is known to develop extremely early in the human foetus at around the 7th week of gestational age (Hepper 1992; Hooker 1952). Studies of the prenatal learning of auditory stimuli would suggest that the response, at least during the last trimester of pregnancy, is mediated by the auditory system. Here particular sounds (music, mother's voice) are repeatedly presented to the foetus and its response to the stimuli examined after birth. These studies show that the newborn responds preferentially to auditory stimuli it had only previously experienced *in utero* (see Hepper 1991; and review Hepper 1989). Whilst these studies suggest that the auditory system is stimulated by external auditory stimuli *in utero* they do not rule out the possibility that other sensory systems may also be involved in mediating the intra-uterine response to sound.

SECTION VIII

NOISE-INDUCED HEARING LOSS

138   A loss in hearing may result from a number of different factors, genetic, ototoxic or environmental, including noise exposure (Martin 1986; Uziel 1985). The discussion here will be limited to hearing loss resulting from exposure to noise. The loss in hearing resulting from noise exposure can be divided into two broad categories, acoustic trauma and noise-induced hearing loss.

139   Acoustic trauma arises from exposure to impulse or impact noise, i.e. an extremely loud noise with a sharply rising wavefront, which causes immediate damage to the auditory system at the time of exposure. Acoustic trauma may cause damage to both the middle ear, e.g. perforation of the tympanic membrane or damage to middle ear ossicles, and to the inner ear, the Organ of Corti may be ripped apart (Henderson & Hamernik 1986; Roberto, Hamernik & Turrentine 1989).

140   The second category, noise-induced hearing loss, is by far the most common type of hearing loss observed in occupational settings (Phaneuf & Hétu 1990). In this case hearing loss results from daily exposure to loud noise, exposure usually continuing for a number of years. The noise levels do not produce acoustic trauma, or immediate permanent effects on the auditory system, but rather injury to the inner ear arises from the continued exposure to the sound. Exposure may first result in a temporary loss of hearing which is reversible, and also tinnitus. With continued exposure these hearing losses become permanent and at present there is no available treatment to restore hearing.

141   Hearing loss can be assessed by determining auditory thresholds, i.e. by evaluating the minimum sound intensity required for a sound to be perceived, at various frequencies. The first sign of hearing loss induced by continuous exposure to a broad-band noise, measured by pure tone

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audiometry, is a dip in sensitivity at the higher frequencies, usually between 3000-6000Hz, often peaking at 4,000Hz (National Institutes of Health 1990). It is perhaps worth pointing out that deafness itself is no protection from further damage, continued exposure to noise will increase the hearing loss. If the exposure to noise continues then further hearing losses are found. These occur first amongst the higher frequencies, e.g., 8,000Hz. Hearing loss is quite rapid during the first years or months of exposure but continuing exposure results in a slower rate of loss. The characteristic dip at 4kHz may disappear as the loss in frequencies either side of this catches up the initial loss observed at 4kHz.

142   The auditory system plays a number of roles in the analysis of auditory signals. One is to sense the presence of sound and for this the sensitivity of the ear, i.e. the minimum sound level required to perceive the sound, is important. A second and perhaps more important function, especially for communication and speech, is the ear's role in analysing sounds and breaking them down into their component frequencies. This frequency resolution role of the ear is crucial for discriminating between different speech sounds and thus communication. Whilst the former function, frequency sensitivity, is readily acknowledged and examined in cases of hearing loss similar attention has not been given to frequency resolution even though this may be a more sensitive indicator of hearing loss (Lutman 1983). Note in the recent National Institutes of Health, USA, Consensus statement on Noise and Hearing Loss (National Institutes of Health 1990), little mention is given to assessing frequency resolution.

143   Hearing loss will follow if the ear is exposed to sounds of sufficient intensity and/or duration. Predicting the levels of sound which can produce a loss, either temporary or permanent, poses significant problems. Perhaps the greatest of these is the large individual variability in response to sound exposures (Mills 1976). Even animal studies, where exposure to sound has been carefully controlled and repeated across subjects, find large individual differences in the effects of identical noise exposure on hearing (e.g. Daniel &

Laciak 1982; Borg, Canlon & Engström 1992). The reasons for this are unclear, but genetic, biological and environmental factors may all contribute to these individual differences (Saunders, Dear & Schneider 1985).

144 Despite these problems attempts have been made to determine hazardous sound levels. It is widely agreed that sound levels below $75dB_{spl}$ do not exert a permanent effect on hearing. Sound levels at $85dB_{spl}$ for eight hours a day for many years may exert a permanent effect on hearing (National Institutes of Health 1990). There have been attempts to extrapolate from this to predict the duration of exposure at which other sound levels may cause harm. This is achieved by time-intensity trading, i.e. the higher the level of sound the shorter the time of exposure per day before damage to hearing will result. A number of different trade-offs have been proposed. Based on the 'total-energy hypothesis' or 'equal-energy hypothesis', which postulates that it is the total amount of energy falling on the ear which determines hearing loss (Ward 1984), a trade-off of a 3dB increase in sound pressure for a halving of exposure time has been adopted by the European Community (European Economic Community 1986). Thus if hazardous noise exposure is assessed at $L_{spl}=85dB(A)$ for eight hours then it's equivalent noise exposure is $L_{spl}=88dB(A)$ for four hours. Others have argued for a different intensity trade-off for a halving of exposure time, e.g., 4dB(A) (U.S. Department of Defense 1987), 5dB(A) (Occupational Safety and Health Act, USA, 1970), and 6dB (Kraak, Kracht & Fuder 1977).

145 What is important for the assessment of noise-induced hearing loss is the amount of energy that reaches the inner ear. As yet there is no successful means of measuring the sound energy in the inner ear and thus sound levels are assessed externally to the ear in the environment. These are the best approximations to sound levels at the inner ear presently available. Note, that given the alteration in intensity as sound passes through the abdominal wall and on to the inner ear of the foetus, external assessment of noise levels are unlikely to be representative of the sound levels at the foetal inner ear.

Animal studies

146   The underlying physiological and anatomical mediation of hearing loss has been a subject of considerable interest. An understanding of these mechanisms has relied on studies of animals, due to the irreversible nature of the injury. One problem with animal studies is that there are large interspecies differences in the nature of sound required to induce the effects. Sound levels inducing changes in one species may not induce changes in other species or man (Decory, Dancer & Aran 1992). Thus whilst the animal studies do not enable conclusions to be drawn about the absolute intensity, or duration, of sound causing particular types of damage in man they do, in very general terms, illustrate the types of injury that may result from exposure to sound.

147   The modelling of prenatal noise exposure in humans using animals poses significant problems. The stage of development of the auditory system may differ between man and the animal species in question. For example, the onset of cochlear function in the rat occurs 10 days after birth whereas for man it is approximately 20 weeks of gestational age (Lenoir, Pujol & Bock 1986). Thus the effects of prenatal noise exposure in both species are not comparable due to the different developmental and functional status of their respective auditory systems.

148   Controlling for the maturational status of the ear poses further problems. In the above example it would require the comparison of a human foetus at seven months of gestational age with a rat, after birth, aged 10 days old. This means that the individuals will now experience sound, or noise, in very different environments. The rat will experience air-borne sound with air in its middle ear whereas the human foetus will experience sound in fluid and fluid in its middle ear. Thus equating the physiological and functional status of the auditory system overlooks the role of environmental factors, e.g. the maternal abdomen and womb, which affect sounds as they pass to the foetal ear.

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149   Problems may still arise if it were possible to equate auditory system development before birth. Damage to the auditory system is caused by the nature of the sound that reaches the inner ear. For the foetus this is, in part, determined by the characteristics of the maternal abdomen and womb. Differences in the size and mass of the abdomen will affect its attenuation and resonant properties and may consequently affect the type of damage to hearing that results. Thus the use of animals to model the effects of prenatal noise exposure poses significant problems.

Physiological and anatomical correlates of noise-induced damage

150   As discussed above the reception of sound is dependent upon the highly complex structure of the sensory cells and the intricate interactions between the various components of the Organ of Corti. Alterations in any aspect of these may affect hearing thresholds either permanently or temporarily. A number of physiological and anatomical changes have been observed in the cochlea as a result of noise exposure. These shall be briefly discussed below (readers are referred to reviews by Dancer, Henderson, Salvi & Hamernik 1992; Salvi, Henderson, Hamernik & Colletti 1986; Saunders, Cohen & Szymko 1991; Saunders, Dear & Schneider 1985; Schmeidt 1984, for further information).

151   Damage to the inner ear may result from either mechanical or metabolic changes. High intensity sounds may induce mechanical damage, whereas more long-term continuous exposure may result in damage due to metabolic exhaustion.

152   Of particular concern has been damage to the hair cells as these are the sensory organs responsible for the detection of sound. Although regeneration of hair cells has been observed in chickens (Corwin & Cotanche 1988) there is no evidence to suggest that the mammalian hair cells are capable of regeneration. Thus the loss of hair cells is, at present, regarded as permanent and irreversible.

153   Changes in both the metabolism and structure of the hair cells have been
      reported, including alterations in the endoplasmic reticulum, mitochondria
      and swelling of the nucleus. The sensory hair cells may, after initial damage,
      degenerate and be replaced with scar tissue. Noise exposure may also damage
      the stereocilia and a number of alterations in the stereocilia after exposure to
      noise have been noted, including damage to the rootlet structures and the tips
      of the stereocilia. The loss of hair cells may in turn affect the auditory
      nervous system. When a sufficient number of hair cells are lost degeneration
      of the cochlear nerve fibres may be seen.

154   The outer hair cells are more vulnerable to damage than the inner hair cells
      and this may be due to metabolic and/or morphological differences between
      the two types of cell. Furthermore different types of stimuli and exposure
      affect the inner and outer hair cells differentially.

155   Noise-induced changes have also been observed in the flow of blood in the
      cochlea. Changes in blood flow which nourishes and supports the function of
      each section of the cochlea may lead to damage to this particular structure and
      thus to the hearing potential (Nakai & Masutani 1988).

156   Acoustic overstimulation may damage the tectorial membrane which in turn
      may alter the way in which energy is coupled to hair bundles and thus affect
      hearing performance.

157   A central issue in noise-induced hearing loss is the relationship between
      anatomical or physiological damage and permanent hearing deficit. Given
      the complex interactions which determine the hearing response a loss of
      function in *any part* of the system, not only the hair cells, may result in a
      hearing loss.

The possible effects of prenatal noise exposure on later hearing

158   Despite many years of study, the underlying mediation of hearing loss is still
      poorly understood (Saunders, Cohen & Szymko 1991). The levels of sound

which produce a loss are known only in general terms and the examination of the effects of prenatal noise exposure upon later hearing have been conspicuous by their absence. Thus one is forced to extrapolate from studies of adults to assess how prenatal noise exposure may affect subsequent hearing. However given the differences between foetal and adult hearing, as outlined above, this must proceed cautiously.

159   Noise-induced hearing loss in the adult is some function of the intensity, duration and pattern of sound exposure. With regard to the foetus exposure to noise can only occur for at most nine months, i.e. the length of gestation. It is thought that for adults an exposure to sounds for nine months would require a sound intensity well in excess of 85dB(A) L_{eq9mo} to produce a permanent shift in hearing thresholds. Furthermore sound as it passes through the mother's abdomen is attenuated thus reducing the overall sound pressure level for a broad-band sound. It may be expected therefore that L_{spl}=85dB per day for nine months would have little effect on subsequent hearing.

160   Two factors temper this conclusion. First, the intensity of sound at the inner ear of the foetus is unknown. Although the abdomen does provide some attenuation, low frequencies around 250Hz may be relatively unattenuated. Further, additional routes of transferring sound energy to the inner ear may act to increase the intensity of sounds experienced.

161   Second, the immaturity of the inner ear may mean that it is more sensitive to the damaging effects of noise than that of the adult. There is much evidence from animal studies that identical noise exposures in newborn and adult animals result in a much greater deficit in hearing in the newborn animal (Bock & Saunders 1977; Bock & Seifter 1978; Lenoir, Bock & Pujol 1979; Lenoir, Pujol & Bock 1986; Saunders & Chen 1982).

162   There may also be a critical period for damage to hearing. A number of
       studies have shown that various drugs exert a greater ototoxic effect during
       the neonatal period than in adults (e.g. Uziel 1985). This is suggestive of a
       period of increased susceptibility of the developing inner ear to adverse
       effects.

163   Studies examining the role of noise exposure have found a similar period
       during the development of the cochlea where greater damage is caused by
       exposure to noise at this stage of development than at other stages (Bock &
       Saunders 1977). Although the chronological timing of the sensitive period
       differs between different animal species, the results are consistent in the fact
       that the sensitive period corresponds to the time at which the cochlea first
       exhibits evidence of adult-like functioning (Pujol, Lavigne-Rebillard & Uziel
       1990).

164   A similar stage of development in the human foetus would correspond to the
       time from six months of gestational age to some time after birth. This
       suggests that there may be a period during the last three months of pregnancy
       at which time the human cochlea and inner ear is more susceptible to
       damage from noise exposure than at other times. Thus noise exposure
       during late gestation could be relatively more harmful to the inner ear than
       at other times during pregnancy or after birth.

165   What effect may noise exposure have on later hearing abilities? In the adult
       noise-induced hearing loss is observed by a drop in threshold around 4kHz.
       This characteristic notch is partly a result of the features of the ear. The outer
       and middle ear differentially amplify certain frequencies. Thus frequencies
       around the 3-4kHz range are enhanced relative to other frequencies. This is
       not the case for the foetus.

166   It is likely that the most 'intense' sounds experienced by the foetus will be
       those of low frequency due to the attenuation characteristics of the abdomen.
       The characteristics of the ear if they exert any effect will enhance higher

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frequencies than in adults. Thus the foetus may not be exposed to enhanced stimulation by frequencies around 3-4kHz as is prevalent in adults. Furthermore as the ear matures and the abdomen changes in size with advancing gestation, this frequency selectivity may change. Thus noise exposure during the prenatal period may result in a different pattern of hearing loss as observed by audiometry than that resulting from noise exposure in adults.

167   The characteristic notch is partly due to the same place on the basilar membrane being continually stimulated and hence damage results through continued overstimulation. In the foetus however there is evidence that the site of maximum responsiveness on the basilar membrane to a particular frequency point changes during cochlear maturation.

168   Examination of newborn hearing reveals that compared to adults whose hearing range extends from 20-20,000Hz, newborn hearing is concentrated at the lower end of this range. Some have suggested that the newborn responds to frequencies in a very restricted range between 500-1000Hz (Rubel, Born, Deitch & Durham 1984), although this seems unlikely given responses in the foetus have been obtained from 83Hz to 5000Hz (Madison, Adubato, Madison, Nelson, Anderson, Erickson, Kuss & Goodlin 1986; Lecanuet, Granier-Deferre & Busnel 1988). However, whatever the range of newborn hearing it appears that hearing begins in the lower frequency range (below 1000Hz) of the adult hearing range (Maeda & Tatsumura 1992; Shahidullah 1993). Relating this to the physiological development of the cochlea suggests that the apical end of the basilar membrane, responsible for the encoding of low frequencies, should develop first and then development proceeds to the basal end responsible for high frequencies. The examination of cochlea development however suggests the opposite. The first regions of the basilar membrane to develop are the basal or mid-basal areas and development then proceeds to the apical end (Rubel, Born, Deitch & Durham 1984).

169   The resolution of these apparently conflicting results is brought about by suggesting that the place code shifts during maturation. The mid-basal area, which develops first on the cochlea, is initially responsive to low frequencies, as the cochlea matures this low frequency encoding shifts towards the apical end of the basilar membrane. Studies which have exposed animals to the same high intensity pure tones at different ages find that the site of damage moves to the apical end of the basilar membrane as the individual ages, supporting the notion of a maturational change in the place code (e.g. Lippe & Rubel 1983; Rubel & Ryals 1983). An alternative explanation proposed by Romand (1987) suggests that the shift in place code results not from passive mechanical changes in the cochlea but rather to the maturation of outer hair cells and thus the change in place code is the result of a change in the active cochlear tuning mechanism. Both would result in a shift in the place code of the cochlea, lower frequencies being encoded nearer the apical end of the basilar membrane.

170   The effect of this for possible prenatal noise-induced damage on hearing is difficult to predict. It may be that, despite the frequency selectivity of different points of the basilar membrane changing, a broad-band noise will stimulate all the developed area of the basilar membrane. Thus the functional area of the inner ear will be continually stimulated. Hearing loss after birth would then be expected to occur at those frequencies corresponding to the place on the basilar membrane most stimulated prenatally, irrespective of the frequencies encoded by that site at the time of stimulation. Whatever the effect of changing frequency responsiveness it is another reason to suggest that hearing loss resulting from prenatal noise exposure may not produce effects on the audiogram as observed in adults.

171   It should also be borne in mind that much of the auditory response is mechanical, i.e. deflection of stereocilia on the hair cell, wave propogation in the basilar membrane. These properties are passive and appropriate hydromechanical stimuli will induce them. Given that the internal environment of the foetus and womb will be relatively uniform, sound

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waves present in the womb would be expected to reach the developing hair cells and basilar membrane, from the moment of their formation. Obviously there will be some attenuation of the sound energy but it does not require a response on the part of the foetus for stimulation to move the mechanical components of the ear and thus cause damage.

172   A final consideration is that prenatal exposure may not result in a hearing deficit at, or soon after birth, but may have a more long-term effect. It is well known that aging reduces hearing sensitivity, especially to high frequencies. Prenatal noise exposure may result in 'premature aging' of the sensory receptors and as such would not be observable until much later in life. Intra-uterine noise exposure may reduce hearing thresholds in the adult population, such that at 40-50 years of age individuals exposed to noise *in utero* have reduced sensitivity compared to individuals of comparable age but not exposed to noise *in utero*. Furthermore, prenatal exposure to noise may increase the susceptibility of the auditory system to damage arising from other risk factors after birth.

173   One factor associated with hearing impairment in adults is tinnitus. The question of whether this occurs in the foetus, as it is exposed to noise, is so far unanswered. However prenatal exposure to noise may increase the likelihood of these individuals experiencing tinnitus after birth compared to those individuals not exposed to noise prenatally.

174   The effects of acoustic trauma are also unstudied in the foetus. Any increased sensitivity of the foetal ear may make the effects of impulse noise more harmful with damage occurring at lower intensities than in adults. However the attenuation provided by the maternal abdomen may act to reduce the overall energy levels of such sounds.

What is a significant hearing loss?

175   One question which should be addressed is 'what is a significant hearing loss?' There has been no internationally accepted level of hearing loss. Loss

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of hearing is particularly important for the understanding of speech. Although most of the energy contained within speech is found in the lower frequencies below 1500Hz, the energy contained above this is important for the discrimination of different speech sounds. Individuals with noise-induced hearing loss because of a loss of resolution, and sensitivity at these high frequencies, experience difficulty in the understanding of speech. British Standard 5330 (British Standards Institution 1976) defines a handicap in hearing as one where the loss is 30dB or greater. This definition was primarily directed at adults who suffered a loss in hearing from excess noise exposure and may not be applicable to children and especially not for newborns if a deficit is present at birth, where a smaller loss in hearing may be more handicapping. Some, in particular Downs, have argued that a lower level of hearing loss in children leads to a severe disability (Northern & Downs 1974). Many speech sounds are inaudible during conversation, particularly sounds of high-frequency and low intensity, e.g. 's', 'sh'. The greater cognitive processing abilities of adults and their years of experience with language enable them to cope with the losses in sound and fill in the gaps in a meaningful fashion. In young children who have yet to acquire these abilities, a similar deficit in hearing thresholds may produce a much larger effect since they lack the cognitive or language skills to cope with these losses. For newborns, who rely on auditory stimulation for the acquisition of language, a much smaller loss in hearing may be severely handicapping as they acquire vocal communication skills. Thus any deficits in the newborn infant may be significant even if not considered significant in adults.

Summary

176   Long-term noise exposure in adults results in a characteristic hearing loss, initially observed as a loss in threshold at 4kHz. Loss is some function of the intensity and duration of noise. Due to the different auditory system of the foetus, prenatal exposure to noise may result in a different pattern of hearing loss to that observed in adults after exposure to noise. There may be a critical period during the last third of gestation in which the individual is more sensitive to damage resulting from noise exposure. As any loss in hearing

will be present immediately after birth a mild loss in hearing sensitivity or resolution may be much more significant and handicapping for the developing child than the adult.

SECTION IX

NOISE AND THE FOETUS

177   Those studies which have examined the effects of prenatal noise exposure on subsequent hearing, and development in general, are considered in this section. The effects of prenatal noise exposure on hearing are considered first. More general effects of prenatal noise exposure on the individual and reproductive outcome are discussed later. In each case studies are presented individually and critically evaluated. The results of these studies are summarised and the implications for legislation discussed. Suggestions for future research are also presented.

The effects of noise exposure during pregnancy on later hearing

178   Adverse effects of noise exposure during the foetal period may be observed in the hearing performance of individuals after birth. Two distinct, but very sparse, lines of research have investigated this possibility. Both have examined the auditory responsiveness of individuals after birth during childhood. One has assessed the effects of occupational noise in the mother's working environment whilst the other has assessed the effects of exposure to the vibroacoustic stimulator during pregnancy. This latter case is considered separately at the end of this section.

179   Despite concern over the possible harmful effects of noise exposure during pregnancy on the hearing of the developing foetus, there has been little direct study of the effects of noise on the foetus and its subsequent hearing.

Human studies

180   Daniel & Laciak (1982) examined the hearing of 75 children whose mothers were employed in a weaving workshop with noise levels in excess of 100dB (unit unspecified). Children were excluded from the study if there were other factors in their history associated with hearing loss. All children were examined between 10 and 14 years of age.

181   Forty-eight males were studied. Of these 23 had normal hearing whilst 25 showed some evidence of damage to their hearing. Bilateral damage to hearing was reported in 17 subjects whilst eight were reported to have unilateral damage. Furthermore the type of damage reported was different between individuals: 10 individuals showed a loss of 20-40dB at frequencies C_1 and C_5, eight individuals exhibited a loss of 20-55dB at C_4 and six a loss of 20-30dB at C_5, one showed evidence of neural degeneration. Twenty-seven females were also examined and of these normal hearing was reported in 17 and damaged hearing reported in 10. Three individuals exhibited bilateral damage to hearing and seven were found to have unilateral damage. As with the male children a variety of different deficits in hearing were reported: five had an auditory loss of 20-25dB at C_4 and C_5, three an auditory loss of 20-25dB at C_5 and two an auditory loss of 20-30dB at C_4. The study also reports that all but two of the mothers in the total sample had a hearing loss at frequency C_5. Maternal hearing loss, however, was uncorrelated with hearing loss in their child.

182   Whilst this paper is suggestive of a hearing loss resulting from prenatal exposure to noise a number of details relating to the experimental procedures are lacking which make evaluation of the paper difficult. Not least of which is the somewhat obscure terminology, C_1 etc., which is more commonly used in music to represent successive octaves. Details of auditory testing are not given which makes evaluation of the testing procedure impossible. This is particularly important given the intra-individual variability of audiometric test results of individuals at this, and younger ages (Gerhardt 1990). Furthermore an appropriate control group has not been included. One interesting observation is the diversity of hearing loss reported. No consistent deficit, or loss of specific frequencies, was observed in all affected individuals.

183   Lalande, Hétu and Lambert (1986a,b) conducted a cross-sectional study examining the hearing of 4-10 year old children of mothers who worked

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outside of home for a minimum of one month during pregnancy. The results of this single study are reported in two separate papers. The children were included in the study if they had no middle ear problems nor any history of diseases associated with hearing loss. In the final sample, 63 female and 68 males were examined. Hearing thresholds were assessed by pure tone audiometry at frequencies of 500, 1000, 2000, 3000, 4000, 6000, 8000 Hertz. For children below seven-and-one-half years of age manual audiometry was employed to assess thresholds, whereas for older children a sweep frequency test was used. For results obtained by manual audiometry a correction factor of -3dB was added to all results. The noise levels at the sites of work (n=45) for all mothers were obtained by direct measurement (n=34), or from records of the employers (n=11). For each mother the daily dosage of noise, $L_{Aeq8hr'}$ was obtained and then values of noise exposure per week, $L_{Aeq40h'}$ trimester, $L_{Aeq480h,}$ and pregnancy, $L_{Aeq1440h}$:$L_{Aeq9mo,}$ were determined. Days not worked were taken into consideration in these calculations and for a complete trimester not worked a value of $L_{Aeq480h}$=70dB was given. Three groups of children based on their prenatal occupational noise exposure over the course of pregnancy were obtained. These groups were composed of individuals exposed to L_{Aeq9mo}=65-74dB, 75-84dB or 85-94dB.

184   Two general points should be made concerning the methodology of this study. The first concerns the exclusion of individuals from the study. Individuals were reportedly excluded if they had a bilateral middle ear problem at the time of testing (Lalande, Hétu and Lambert 1986a) or any middle ear problem at the time of testing assessed by tympanometry (Lalande, Hétu and Lambert 1986b). Furthermore a negative history of diseases associated with hearing loss was a reason for exclusion. No attempt was made to determine if there were a relationship between noise exposure and middle ear problems. This is important since prenatal noise exposure may result in an increased incidence of such problems. The relationship between the age of the children and exclusion should also have been assessed to determine any age effect.

185   The second point relates to the reporting of hearing thresholds. Hearing thresholds used in the results of this study reflect thresholds of the worse ear. Although individuals with bilateral middle ear problems were excluded, individuals with unilateral ear problems were apparently not. Furthermore the observed shifts in threshold may be due to small middle ear problems, often not detected by tympanometry, rather than some sensorineural pathology. A better strategy would have been to report data from the better ear.

186   The mean hearing loss reported for each noise exposure group from the first of the two papers (Lalande, Hétu and Lambert 1986a) is presented in Table 1. For all frequencies tested there appears to be little effect of increasing noise exposure on hearing loss of the worse ear. The authors report that, statistically, there was significant effect of noise exposure on hearing loss at 3000Hz. Hearing loss was significantly greater after noise exposure of L_{Aeq9mo}=85-94dB than both L_{Aeq9mo}=65-74dB or 75-84dB at this frequency. However, the non-monotonicity of the results with prenatal noise exposure, coupled with the fact that the data are from the worse ear, suggest that the effect is a weak one.

TABLE 1

L_{Aeq9m} (dB)	FREQUENCY (Hz)						
	500	1000	2000	3000	4000	6000	8000
65-74	5.9	2.2	1.7	2.1	3.0	5.7	9.2
	3.7	*4.8*	*5.9*	*5.2*	*6.2*	*7.9*	*9.2*
75-84	4.7	2.8	-0.2	0.6	2.9	6.1	10.9
	3.6	*4.6*	*5.5*	*5.3*	*6.3*	*7.7*	*8.8*
85-94	5.3	2.1	1.4	3.4	5.6	7.9	12.3
	3.2	*4.4*	*4.9*	*5.8*	*7.1*	*6.6*	*9.9*

TABLE 1. The mean values (reported in dB hearing loss re : ANSI 1969) and their standard deviations (in italics) of the worst hearing threshold for each frequency tested and for three classes of noise exposure (L_{Aeq9m}) before birth (n=131). From Lalande, Hétu & Lambert 1986a.

187   The paper also reports data concerning the number of individuals exhibiting a significant hearing loss. A significant hearing loss was defined as a loss of 10dB or greater between 1-4kHz, 15dB at 500Hz and 6kHz and 20dB at 8kHz. Data are reported for only 102 of the original sample as these represent the number which had both ears tested. The proportion of individuals showing a deficit in one or both ears, and at one or more frequencies, is reported in Table 2. As can be seen 31.4% of the sample showed a significant hearing loss, based on these criteria for hearing loss. For 9.8% of the sample this involved only one ear and one frequency and whilst for 21.6% this involved more than one frequency or both ears. Although there was a tendency for greater impairment (i.e. more than one frequency) with increasing noise exposure, this is not a statistically significant trend.

TABLE 2

	L_{Aeq9m} (dB)			
Hearing status	65-74	75-84	85-94	Total
Normal hearing	79.2	67.7	63.8	68.6
Hearing loss/1 ear	12.5	22.6	14.9	16.7
1 frequency	12.5	9.7	8.5	9.8
2 frequencies	0	12.9	4.3	5.9
3 frequencies	0	0	2.1	1.0
Hearing loss/2 ears	8.3	9.7	21.3	14.7
1 frequency	4.2	0	6.4	3.9
2 frequencies	0	6.5	4.3	3.9
3 frequencies	4.1	3.2	8.5	5.9
4 frequencies	0	0	2.1	1.0
No. of children	24	31	47	102

TABLE 2. The proportion (expressed as a percentage) of children with normal hearing and with a hearing loss for the three classes of prenatal exposure to noise. After Lalande, Hétu & Lambert 1986a.

188   A second paper (Lalande, Hétu and Lambert 1986b), based on the same sample, reports the effects of prenatal noise exposure on hearing at 4000Hz in more detail. There was a significant association between noise exposure and

hearing threshold for the worse ear at 4000Hz. The proportion of children having a significant hearing loss of greater than 10dB at 4000Hz was 3/4 times greater when their mothers were exposed to L_{Aeq9m} of 85-94dB compared to lesser exposures (no. of children with hearing loss at 65-74dB prenatal exposure, 2/34 [5.9%], 75-84dB 3/43 [7.0%] and 85-95dB 13/54 [24.1%]).

189   Low frequency noise, although it was not specified what the low frequencies were, was significantly associated with hearing loss. For noises with little low frequency components hearing loss was similar across all three groups of prenatal noise exposure. However for noise exposure having a large low frequency component hearing loss increased with intensity (65-74dB - 2.3dB hearing loss, 75-84dB - 3.6dB hearing loss, 85-95dB - 17.5dB hearing loss).

190   A number of other concomitant variables, vibration, exposure to chemical products, and number of hours worked, showed no significant association with hearing loss. There was however a significant interaction between noise exposure and risk factors for hearing loss. Risk factors, as defined in this study, were respiratory difficulties as a newborn, use of a ventilator, use of an incubator, jaundice and minor body malformations. If no risk factors were present then there was no difference in the hearing loss between different exposure groups. However in the presence of risk factors there was a significant effect on hearing loss of different exposure levels. Hearing loss was reported as 6.1dB, 2.4dB and 10.2dB for prenatal exposures of L_{Aeq9mo}=65-74dB, 75-84dB and 85-94dB respectively. Note that the least effect on hearing is associated, not with the group least exposed to noise, but with the group exposed to the medium amount of noise.

191   The study indicates a possible detrimental effect of noise exposure before birth on hearing after birth. However there are a number of factors regarding this study which mean this conclusion should be treated with caution. The size of the differences in auditory thresholds resulting from prenatal exposure are quite small in comparison to the reported standard deviations. Thus there is

a basic lack of power in the design to detect differences. No control group was used. The variability of hearing tests in children of this age may influence the results, those exhibiting a loss on one day may very well not exhibit a loss on the next (Gerhardt 1990). Information regarding possible confounding factors was obtained by interview with the child's mother. Little information was given as to whether the responses of the mother, especially with regard to medical factors, was checked or confirmed with independent sources, e.g. medical records. Questions may also be raised regarding the amount of noise exposure during pregnancy. Although an equivalent noise exposure for the nine months of pregnancy was used, inclusion in the study was based on mothers working a minimum of one month during pregnancy, thus individuals may have been exposed to noise from one to, in theory, nine months of their pregnancy. Little account was given as to when noise exposure occurred if not for the full nine months.

192   Low frequency sounds were found to be implicated in hearing loss, especially when related to the higher levels of noise exposure, however no information is given as to what these low frequencies were. The presence of low frequency sounds appears to be based on subtracting noise levels assessed using the 'A' weighting scale from an unweighted measure of dB. The presence of low frequency noises based on whether this difference was greater than 5dB. Further information is required regarding the frequency and energy composition of these low frequency sounds.

193   As with the previous study (Daniel & Laciak 1982) the pattern of hearing loss observed was different to that classically seen in noise-induced hearing loss in adults. This may suggest that the mediation of hearing loss in the foetus is different to that observed in adults.

194   A final note of interest in this study was that mothers were asked if the child had any learning problems at school. It revealed that children suffering from a high frequency hearing loss had a much greater chance of being reported as having a learning problem at school than those individuals with normal

hearing (3/14, 21.4% vs 4/83, 4.8%, respectively). The authors report this

difference to be significant using χ^2 test, however a more appropriate

statistical analysis given the small cell size, the Fisher Test, reveals the

difference to be nonsignificant (p>0.3). Although the results were

nonsignificant, small sample sizes were used, and the assessment of learning

difficulties was based only on maternal report and not verified by

examination of school records. It is important to acknowledge the authors'

attempt to examine aspects, other than hearing, of the individual's

performance. As was suggested earlier hearing loss present from birth may

have implications in a wide range of educational and social contexts.

195   The evidence presented by these studies, whilst suggestive of a hearing loss

resulting from prenatal exposure to noise, cannot be regarded as conclusive.

The lack of study and methodological drawbacks mean that the effects of

prenatal noise exposure on later hearing abilities require further study before

definite conclusions can be drawn.

Animal studies

196   As with study of noise exposure during pregnancy on the human foetus and

its effect of later hearing, examination of the auditory effects of prenatal

sound exposure in animals are conspicuous by their absence.

197   Cook, Konishi, Salt, Hamm, Lebetkin & Koo (1982) exposed guinea-pigs

during the last trimester of their pregnancy to the sound of a 'loom room' at

115dB(A). The duration of exposure ranged from 6-22 days prior to delivery.

A control group who were exposed to the noisy room environment, but not

to the noise, were also tested. The hearing of their offspring was measured on

three separate occasions after birth (mean time of testing 38, 45 and 52 days

after birth). Hearing was assessed by auditory brain stem evoked response to

broad-band clicks. Evidence was found of a latency shift in peak IV, the

average latency of the control group being less that the noise exposed group.

No significant effects were observed in whole wave-form RMS, peak IV

amplitude nor in the cochlear microphonic input/output functions. The difference in peak latency corresponds to a 5dB increase in hearing threshold for the noise-exposed animals. Given the mix of significant and nonsignificant results the study is suggestive of a role for noise exposure in hearing loss, but as the authors themselves suggest, other factors, e.g. stress, may also play a contributory role.

198   An examination of the effect of noise exposure during the 'critical period' of the cochlea, i.e. that is when the cochlea is purportedly most susceptible to damage arising from noise exposure, was undertaken by Leroux (1988). Here deafened pregnant female guinea-pigs were exposed to noise, 420Hz tone at $110dB_{spl}$ for eight hours day, five days a week, either before the onset of the 'critical period' 15 days before birth, or before and during the 'critical period'. A control group of pregnant guinea-pigs not exposed to noise was also assessed. Individuals were assessed 60 days after birth; hearing thresholds at 2, 4, 8, 16 and 20kHz were examined by electrophysiological recording at the level of the cochlear nucleus, and the structure of the cochlea, in particular of the outer hair cells, was examined by electronmicroscopy.

199   The final sample sizes were quite small: six ears from three animals born to control females; eight ears from five animals belonging to the group stimulated before and during the prenatal 'critical period', three animals having both ears examined, two having only one ear examined; and six ears from three animals exposed to sound before the onset of the 'critical period'. Animals were withdrawn from the final sample to be assessed if they were born during noise exposure, they had a pathology of the middle, or inner, ear or underwent an accidental manipulation (n=1 for this latter category). Interestingly only the group exposed to sound before and during the 'critical period' had individuals withdrawn for pathologies of middle and inner ear. No pathologies were reported for individuals in the group exposed to sound before, but not during, the 'critical period'.

200   Measurement of hearing thresholds revealed that foetuses exposed to sound before and during the 'critical period' had elevated thresholds, approximately 6dB, at all frequencies (2, 4, 8, 16, and 20kHz) compared to the control group and those exposed to sound only before the 'critical period'. Hearing thresholds of the control group and those exposed to sound only before the 'critical period' were identical. Four ears were subject to electronmicrographic examination of the cochleas, one from a control animal and three from the group exposed to sound during the 'critical period'. One of these showed a statistically greater number of abnormalities in the outer hair cells than the control, a greater number of abnormalities were also observed in one of the other cochleas from an animal exposed to sound during the 'critical period' but this did not reach statistical significance. However it should be noted that the relationship between a statistically significant number of abnormalities and the number required to affect functioning is unknown.

201   The results suggest that exposure to sound during pregnancy affects both function and structure of the inner ear. Furthermore the evidence is suggestive of the importance of sound exposure during the 'critical period' when the inner ear is acutely susceptible to trauma from overstimulation. However this conclusion should perhaps be treated cautiously since animals were exposed to sound before <u>and</u> during the 'critical period' thus the noise exposure of animals in this group was much greater than in the group exposed to sound only before the 'critical period'. Examination of the effects of noise exposure only during the 'critical period' would demonstrate the importance of sound exposure during this period.

202   Daniel & Laciak (1982) examined the effects of noise during pregnancy on the hearing of guinea-pigs after birth. Guinea-pigs were exposed to the sound of a 'weaving room' for eight hours per day for five days per week, six hours on Saturday and a rest day on Sunday. The sound intensity of this noise was unspecified. A control group of guinea-pigs were not exposed to sound, and after birth both groups were housed in a room with sound level of 34-

36dB(C). Hearing was assessed at 3, 7, 14, 30, 60 days after birth using the Preyer reflex. This reflex, initially described by Preyer (1908), involves the twitching of the pinna to quiet sounds and both the pinna and body to loud sounds. No differences were observed between tests on days 3, 7, and 14 and the results from these days were combined into a single examination, examination 1. Examination 2 was performed at day 30 and examination 3 at day 60.

203   Of the exposed group of 156 newborns, 110 (70.5%) were born alive and a further 10 died within 3-10 days of birth. For the control group of 30 newborns, 27 were born alive and a further two died within three days of birth.

204   With respect to hearing the control group responded to the frequencies tested (1, 1.5, 2, 3, 4, 6, 8, 10kHz) at lower intensities than did the group prenatally exposed to noise. Perhaps the most significant finding of this study was that the hearing in the exposed group progressively deteriorated after birth even though they had only been exposed to the sound before birth. Between 3-14 days approximately 50% of the exposed guinea-pigs had normal hearing, by the second examination at 30 days of postnatal age 35% had normal hearing whereas by 60 days all the guinea-pigs had a hearing disorder, and some showed no response to sound at all.

205   Interestingly, this study also examined the vestibular system of the guinea-pig and found that the group exposed to noise also exhibited evidence of disorders of the vestibular system.

206   Szmeja, Slomko, Sikorski & Sowinski (1979) exposed pregnant guinea-pigs to noise of 95-100dB (unit unspecified) in a factory hall. Noise was continuous with a 12 hour break once a week. At birth the newborn young were moved into a normal room and from 2-20 days of age the Preyer reflex was examined with a Peters audiometer. After testing histological examination of the cochlea was performed. There was no difference between the hearing

response of control and noise-exposed guinea-pigs nor were there any histological differences between the cochleas of the noise-exposed or the control animals.

207   The results from these animal studies are similar to those which have examined hearing loss in humans in that they are equivocal. Little evidence is available to determine whether damage results from hearing loss during pregnancy, and that which is available is suggestive of the fact that noise exposure may influence both auditory thresholds and structure but different studies have produced conflicting results (cf. Daniel & Laciak 1982 and Szmeja, Slomko, Sikorski & Sowinski 1979). One result which may have important implications is that of the continuing deterioration of hearing after birth even though noise exposure ceased at birth. Replication of this finding is obviously important.

Vibroacoustic stimulation

208   The vibroacoustic stimulator is one particular sound source that has gained in popularity over recent years as a clinical tool used in the assessment of foetal health (Gagnon 1989). The device provides both acoustic and vibratory stimulation. A common example in wide-spread use is the electronic artificial larynx 5C made by Western Electronic. This produces a broad-band audible noise at 110dB$_{spl}$ from 87-15,000Hz. Its surface also vibrates between 10-15,000Hz, with maximum vibration occurring at 450Hz.

209   The intra-uterine sound levels produced by this device have given cause for concern. Whilst measurements in air demonstrated a stimulus intensity of 110dB two inches from the sound source, and 82dB one meter away, these were assumed to be safe as the attenuating characteristics of the maternal womb would act to lower these levels (Gagnon, Patrick, Foreman & West 1986). However the stimulator was specifically designed for the efficient transmission of mechanical energy through tissues. A study of the intra-uterine intensity levels produced in pregnant sheep revealed an overall

intensity level of 135dB$_{spl}$ (Gerhardt, Abrams, Kovaz, Gomez & Conlon 1988). Thus the sound intensity produced by this machine may be great and possibly dangerous to the hearing of the foetus.

210   Stimulation using the device has also been questioned because of the abnormal changes in behaviour induced after presentation of the stimulus. A brief, five second or less, period of stimulation may induce changes in heart rate (Ohel, Birkenfeld, Rabinowitz & Sadovsky 1986) and body movements (Gelman, Wood, Spellacy & Abrams 1982) of the foetus lasting for up to 60 minutes (Gagnon, Hunse, Carmichael, Fellows & Patrick 1987a,b). Of most concern have been the prolonged alterations in the behavioural state of the foetus (Visser, Mulder, Wit, Mulder & Prechtl 1989), especially the prolonged duration of state 4F, the most active foetal state (Nijhuis, Prechtl, Martin & Bots 1982). These have been argued to be unnatural and may place the foetus at unnecessary risk (Prechtl 1988). Similar changes in the neonate may only arise by the presence of painful stimuli and the question of whether the stimulation provided by the vibroacoustic stimulator, or indeed other intense sounds, produce pain in the foetus has yet to be assessed. Exactly what the long-term developmental consequences of such exposure are have yet to be determined. However if pain is being induced by this stimulus, this or other similar sounds or noise which induce prolonged changes in behaviour, may be painful and should be avoided.

211   Given its extremely prevalent use in obstetric practice its effect on the auditory system needs to be considered. Recently studies have appeared which have assessed this.

212   Ohel, Horowitz, Linder & Sohmer (1987) examined the auditory brain stem evoked responses in two groups of neonates one to two days after birth. One group (n=20) had been exposed to three, five second stimulations by an electrolarynx during active labour. The second group (n=20) served as the controls and received no stimulation. There was no difference between the control and noise-exposed group on either the auditory brain stem first wave

latency or brain stem transmission times.

213   In a much larger study, Arulkumaran, Skurr, Tong, Kek, Yeoh & Ratnam (1991) examined the hearing of 465 children at four years of age after variable exposure to an electrolarynx during pregnancy between 28-43 weeks of gestational age. Hearing was assessed using a hand held audiometer with test tones of 1000 and 4000Hz at 25dB. Four hundred and thirty four children responded to the screening test and only 31 failed. Of these 31, local transient causes (impacted wax, respiratory infection) explained the hearing loss in 29 of these and retesting after resolving the initial problem resulted in normal test results. Thus only two of the 465 had any hearing abnormality. The authors conclude that there is little evidence of hearing loss due to prenatal vibroacoustic stimulation.

214   However this conclusion should be treated cautiously. The hearing test used only two frequencies, 1000Hz and 4000Hz, the frequencies chosen based on patterns of adult hearing loss. As has been previously discussed this assumption may be erroneous and a full audiometric examination is required. No control group was used. Also, as the authors acknowledge, there may have been a mild deficit too small to be detected by this test, since the test was only intended to detect a deficit of 25dB or greater. This is particularly pertinent given the results from the study by Lalande, Hétu & Lambert (1986a,b) who report deficits of the order of 2-12dB.

215   Much more work is required to determine whether vibroacoustic stimulation adversely effects later hearing abilities.

Summary

216   The sheer lack of studies examining hearing after prenatal noise exposure makes the drawing of any conclusions difficult. The studies are at best suggestive of the fact that prenatal noise exposure may affect hearing.

217   There is also the suggestion that low frequency noises may contribute to hearing loss. Given the previous discussion on sound transmission *in utero* it was seen that low frequency sounds will pass relatively unattenuated to the foetus. It may be that low frequency sounds affect hearing because these are the only sounds to reach the foetal inner ear with sufficient intensity, after attenuation at the maternal abdomen, to cause damage to the inner ear. Again this stresses the importance of abandoning the 'A' weighted decibel scale, which reduces the contribution of low frequency sounds to the assessment of noise levels, for noise measurement relating to the foetus.

218   One interesting result from the studies of children is of the different pattern of hearing loss observed after exposure. The characteristic dip at 4kHz was not observed. This provides some support for the argument advanced earlier that if prenatal noise exposure does influence hearing its effects would not be similar to the effects of noise exposure after birth. The studies also confirm another result from studies of adults and that is of the large inter-subject variability in outcome.

219   The vibroacoustic stimulator appears not to cause any damage to the hearing of the foetus, but further studies are required to assess this. Whether the stimulation induces pain in the foetus, or whether the abnormal behavioural states observed, have a damaging effect on future health and development are unknown.

Protection and legislation

220   Theoretically, sound present in the womb, if of sufficient intensity and duration, may adversely affect subsequent hearing. The lack of experimentation in this area however means that the levels at which harm is caused are unknown. Low frequency sounds may be important as these will pass unattenuated through the abdomen and thus retain the intensity of sound present in the external environment. Future studies have to address this lack of knowledge.

221 If prenatal exposure to noise does cause adverse effects on subsequent hearing these effects will be mediated directly by sound impinging upon the foetal ear. Thus the provision of ear protection to the mother to reduce her level of noise exposure will have no effect on the levels of noise exposure, and hence risk of damage, experienced by the foetus. Protection of the foetus requires the reduction of sound entering the maternal abdomen. Whilst it may be possible to use some form of protective coat which attenuates noise, and so reduces sound levels at the abdomen, this may cause additional problems for the mother in performing her normal occupational activities, for example, increased bulk, increased temperature, increased stress.

Future studies

222 Clearly there is a need for further study of the effects of prenatal noise exposure on later hearing. Any studies which assess the effects of prenatal noise exposure should not only assess hearing, both the sensitivity of hearing and frequency resolution, but also given the importance of hearing for communication, language and social skills should also be examined. Techniques are available to assess hearing in newborn infants (Davis, Wharrad, Sancho & Marshall 1991) and these may be used to assess the effects of prenatal noise exposure soon after birth.

223 There is a need for prospective epidemiological studies to assess the effects of prenatal noise exposure. These should be long-lasting especially given the fact that noise exposure before birth may not produce an immediate effect on hearing but may result in a premature loss of hearing ability in later life. As with all such studies careful control of possible confounding variables is essential. There is also a role for retrospective studies. Indeed given the suggestions of possible premature aging in the auditory system (para. 172), or increased incidence of tinnitus (para. 173), resulting from prenatal noise exposure a retrospective study may be used to, partly, address this.

224 Some authors (Gerhardt 1990) have argued for animal studies to evaluate the effects of noise exposure in the womb. The validity of these may be

questionable for legislation purposes. As discussed before (paras. 146-149) the suitability of animals for modelling human foetal hearing poses great problems. Considering factors of the development of the auditory system and the uterine sound environment the sheep is probably the best model. However animal studies have a role to play in investigating the structural consequences of prenatal noise exposure. There is probably little need for animal studies to demonstrate that sufficient noise intensity causes damage to hearing as this is well established. However a number of research questions suggested by this review may be most easily addressed using animal models. The role of exposure during the critical period and the possibility of continued progressive deterioration of hearing after exposure can be addressed using animal models.

225   One area of extreme importance is the determination of sound levels within the womb and the foetal ear. The attenuation factors provided by the abdomen and tissue, as well as the routes by which the inner ear receives stimulation, need careful consideration and investigation. In order for any legislation to provide protection the sound levels and frequencies at which harm is caused need to be known.

226   A final area in need of study is the examination of when during pregnancy the effects of noise exposure are mediated. Studies to date have simply assessed the effects of noise exposure throughout pregnancy. However the effects of noise exposure may be the result of experiencing noise at a particular point during pregnancy, for example during the last three months. Prospective studies, enabling actual measurement of noise levels at various times during pregnancy, can be used to evaluate the effects of noise exposure during different periods of pregnancy. Again this is crucial for protective legislation and regulation.

The effects of noise exposure during pregnancy on reproductive outcome

227   Whilst noise exposure may affect the individual's subsequent hearing abilities it may also exert effects on health, development and behaviour (e.g.

see Smith & Broadbent 1991). These have been variously described as 'indirect effects of noise' or 'non-auditory effects of noise' (van Dijk 1986). Essentially this refers to the effects of noise excluding those exerted on the auditory system.

228   Auditory stimuli, after processing by the inner ear, are carried to the auditory cortex by means of the auditory ascending pathway. Neural impulses from the auditory nerve also stimulate other areas of the nervous system via its input in the reticular activating system (Paxinos 1990). This may result in stimulation of the limbic system and thus affect emotional responses. The reticular activating system also innervates the hypothalamus, the highest centre of the autonomic nervous system, and through this may enhance secretion of catecholamines via the adrenal medulla, or cortisol via the hypophysis and adrenal cortex.

229   Noise not only stimulates the auditory pathway but also this second neural pathway which may result in a variety of physiological (including vascular, gastrointestinal, endocrinological, pharmacological and immunological functions) and psychological (annoyance, fatigue, performance and behavioural deficits, stress) effects.

230   The following review discusses the effects of noise during pregnancy on reproductive outcome, specifically on the health and well-being of the foetus and newborn. The specific instance of aircraft noise is discussed first and occupational noise later.

Aircraft noise

231   The effects of aircraft noise have been examined with respect to a number of different health hazards. After birth aircraft noise, i.e. living within a certain area of an airport, has been linked with increased mortality and morbidity, adverse physiological and psychological effects in children and adults (Rosenberg 1991).

232 The effects of aircraft noise on the developing foetus have received some attention. Initial studies conducted in Japan (Ando & Hattori, 1970, 1973, 1977a) examined the effects of living near an airport (Osaka) on the sleeping behaviour of infants. The infants of mothers who had lived near the airport for all of their pregnancy, or who moved there during the first five months of pregnancy, showed little reaction to aircraft noise whilst sleeping, i.e. they kept sleeping and did not wake. The infants of mothers who moved to the high noise area within the last four months of pregnancy, or soon after birth, reacted more intensely to the sound of the aircraft, i.e. they woke up or moved around. The effects of aircraft noise were evaluated by the mother via a questionnaire. Recording of EEG and plethysmogram during aircraft noise revealed a similar pattern of responding. Those infants of mothers who had moved to the airport area within the last four months of pregnancy showed a greater reaction to aircraft noise than individuals whose mothers had moved to the area before five months of gestational age.

233 Studies have examined the effect of aircraft noise on birth weight and/or gestation. Ando & Hattori (1973) reported an increase in the incidence of low birth weight (<2500gms) from 4.8% in an area of 74dB (ECPNL, Equivalent Continuous Percieved Noise Level, a special unit of noise measurement often used in the assessment of noise around airports, see Schultz 1972 for full definition and calculation) to 8.2% in an area of 90dB (ECPNL). This study also reported an increase in the incidence of toxicosis amongst mothers living in the noisy area. This effect was apparently confirmed by the examination of the incidence of low birth weight (<3000gms) before and after jet planes started to fly from the airport. An increase in the incidence of low birthweight infants was found after jet planes flew into the airport (Ando 1988).

234 Rehm & Janssen (1978) examined the incidence of prematurity (although this was defined as a birthweight of <2500gms so should more properly be called low birth weight) around Dusseldorf airport for mothers living in three areas near the airport, a low, a medium and a high noise level area (the actual

82

noise levels were not reported). Although there was a trend in the incidence of 'prematurity', more 'premature' babies born to mothers in the high noise area, this did not reach statistical significance.

235   Other studies have produced some conflicting results which partly support an effect of airport noise on the developing foetus.

236   Schell (1981) found that there was an effect of noise, assessed as peak noise level for 1% of the 24hr period, on the gestation of females. The greater the noise exposure during pregnancy the shorter the length of gestation. Gestation in this study was obtained by asking the mother whether the baby was born too soon or delayed compared to initial estimates from the hospital. This effect was only observed for female children and not for males. No effect of noise on the birth weight of male or female infants was observed.

237   Knipschild, Meijer & Sallé (1981) examined the incidence of low birth weight (<3000gms) in two groups of children born to mothers who lived around Amsterdam airport. Low noise exposure was defined as living in an environment of L_{dn} 60-65dB or less and high noise exposure if noise levels were above this. For all infants in the study (n=1840) there was no effect of noise exposure. However considering only those infants born in hospital (n=902), those from a high noise area were born significantly lighter in weight than infants from the low noise area (3354gms vs 3424gms respectively) and a greater proportion were low birth weight (<3000gms), 24% and 18%, respectively. There was a significant effect of income, however when this was adjusted for the significant differences still remained. The highest incidence of low birth weight was seen in females born to mothers in a low income group living in a high noise area.

238   The results of these studies have produced some intriguing findings. It is difficult to see why females rather than males should be affected as reported in two of these studies. Methodologically, consideration of other factors which may affect birthweight have not been systematically considered, in

particular smoking and alcohol consumption. One study (Knipschild, Meijer & Sallé 1981) found a strong link between income, albeit assessed from health insurance, and birthweight indicating the importance of social factors in these studies. A major confounding factor is in the definition of low birth weight and prematurity. Low birth weight may result from either a premature infant, i.e. an infant born at 30 weeks of gestational age, or from a infant who is growth retarded, i.e. an infant born at the appropriate gestational age but weighing less than the average weight of the individual at this age. The underlying causation of these two types of low birth weight may be different and may be differentially influenced by aircraft noise. It is important that the two groups be distinguished in future studies.

239   Other studies have looked at the effects of aircraft noise on the incidence of abnormalities. Again the findings have been equivocal. Jones & Tauscher (1978) examined the incidence of birth defects in infants born to mothers living within a 90dB(A) contour of Los Angeles Airport and mothers in the rest of Los Angeles. More birth defects were found in black infants of mothers living near the airport than in black infants born to mothers living in the rest of Los Angeles. Polydactylism was not included in this study. No differences in the incidence of all birth defects considered was observed for white infants. However when the incidence of anencephaly and spina bifida was examined alone in white infants an increased incidence in these specific defects was noted for infants of mothers living near the airport.

240   Edmonds, Layde & Erickson (1979) examined the incidence of 17 catagories of birth defects in two groups of infants whose mothers lived around Atlanta airport. A high noise group (>L_{dn}=65dB) and a low noise group (<L_{dn}=65dB). No significant associations with noise exposure were found. The only positive association with noise was observed for spina bifida with hydrocephalus but this was not found for spina bifida alone. It is unclear why this should be the case.

Summary

241   The effects of prenatal exposure to aircraft noise are equivocal. There is some evidence to suggest prenatal exposure to aircraft noise has an effect on sleeping patterns, but the significance of this for other spheres of activity has yet to be demonstrated. An overall assessment is difficult because of the use of different measures of sound level for high and low noise exposures. Some have used day/night average (Knipschild, Meijer & Sallé 1981), others have used peak values (Schell 1981), whilst still others have used maternal assessment (Ando & Hattori 1973). Sound levels have usually been calculated at certain levels around the vicinity of the airport to produce sound contours. These have been measured outside of houses and thus the effects of attenuation from the subject's house has not been considered. Different dependent variables have been used, e.g. low birth weight (<2500gms or <3000gms). Careful control of social and physiological confounding variables has often not taken place. Other confounding variables, e.g. areas around airports may also have a higher level of pollutants than other areas, or are inhabited by lower SES groups, have not been taken into account.

242   In adults exposure to aircraft noise has proven effects and recent studies suggest these are more pronounced in children (Rosenberg 1991). These effects have yet to be demonstrated in the foetus. The effects of aircraft noise may be mediated by the foetus sensing the noise and the effect caused by direct stimulation the inner ear of the foetus. Alternatively the effects of the aircraft noise may be mediated by the mother's physiological reaction to the sound. Ando & Hattori (1977b) report an increase in human placental lactogen in mothers exposed to aircraft noise in the last four months of pregnancy not evident in mothers who have been exposed to aircraft noise for the whole of pregnancy.

Occupational noise

243   Other studies of the effects of noise on reproductive outcome have not looked at a specific type of noise but rather have observed the effects of working in a noisy environment on the foetus and its future development. Often these

have been performed with a view to studying the effects of a number of occupational factors on reproductive outcome with noise exposure as one variable under consideration.

244   McDonald, Armstrong, Cherry, Delorme, Diodati-Nolin, McDonald & Robert (1986) studied 56,012 births in Montreal, Canada, assessing the effects of a number of occupational factors on the incidence of spontaneous abortion (defined as the loss of pregnancy before 28th week of gestation). Noise level was assessed as either high or low and evaluated by a questionnaire administered to the mother. No indication of what was considered high/low noise was given. Noise was found to have a small association with spontaneous abortion, as was standing and lifting, but stronger associations with spontaneous abortion were found for physical effort, complaints of cold and long hours of work. As no direct measure of noise level was evaluated, it is difficult to specify levels of noise exposure which may be harmful.

245   Using the same data base, McDonald, McDonald, Armstrong, Cherry, Nolin & Robert (1988) examined the effects of various occupational factors on the incidence of prematurity (a birth before 27th week of gestational age) and low birth weight (<2500gms). Noise was found to be associated with low birth weight, but not prematurity, and only in health and manufacturing sectors not managerial, office, sales, or service sectors. The reason for an effect on low birth weight but not prematurity is unclear but may reflect a different underlying mechanism which results in low birth weight or prematurity. This again stresses the importance of discriminating between low birth weight and prematurity. As with the previous study the level of noise exposure causing the effect cannot be determined since noise levels were simply classified as high or low by the mother and absolute sound levels not determined.

246   A study of 1383 women who worked in a heavy metal industry in Poland was reported by Gracyalny & Zdebski (1983). They examined the incidence of preterm birth/spontaneous abortion between the 16-37th week of pregnancy

and found that a 'disadvantageous environment' was associated with an increase in preterm births and low birth weight. Noise, as well as dust and temperature, was a factor producing a 'disadvantageous environment'.

247 Dennler, Diener & Müller (1989) compared pregnancy outcome in a group of textile workers to that of a control group drawn from the surrounding area. The textile workers were exposed to a noise level of greater than 85dB(A). The actual intensity and duration of exposure is unclear. There was no difference between the two groups on any of the variables examined which included, low birth weight, spontaneous abortion, number of sicknesses, number of times off work, length of time off work, frequency of hyperemesis and gestation.

248 Mamelle, Laumon & Lazar (1984) examined the effects of occupational conditions on prematurity, i.e. infants born before 37 weeks of gestational age, in a sample of 3437 women. Information on environmental conditions, including noise was obtained via a questionnaire given to the mother after delivery. Two factors were found to have an effect on preterm delivery, mental stress and the environment. Although noise was not specifically examined it was a contributory factor to a high loading on the environmental factor which also included lighting, ambient temperature, atmospheric pollution, background noise and manipulation of toxic substances.

249 Kurppa, Rantala, Nurminen, Holmberg & Starck (1989) examined the effects of noise during the first trimester on malformations of the foetus. Individuals born presenting with a birth defect were obtained from the Register of Congenital Malformations maintained in Finland and each individual was paired with the birth immediately preceding it. In total 1475 pairs of infants were examined. The incidence of infants with central nervous system defects, orofacial clefts, skeletal defects and cardiovascular defects showed no relationship with noise exposure during the first trimester, either as evaluated by the mother in the form of a questionnaire or by assessing the noise levels in the place of work. Fetuses were considered

exposed to noise if their level of prenatal noise exposure was greater than $L_{Aeq8h}=80dB$.

250   Hartikainen-Sorri, Sorri, Anttonen, Tuimala and Läärä (1988) examined the effects of noise on low birth weight (below the 25th centile for gestational age and born after 37 week of gestation) and prematurity (born before 37 weeks of gestation). Cases of low birth weight and prematurity were paired with matched controls. Mothers were given a questionnaire within one year of delivery and asked to specify exact noise levels during employment (consultation with an occupational health officer was suggested if the mother had difficulty with this). A noisy environment was one defined as $L_{eq(A)8hr}=81dB$ or more. In final analysis 189 premature and 204 low birth weight subjects were studied. Only 26 individuals reported being exposed to noisy environments. There was no significant effect of noise exposure on prematurity or birth weight. Impulse noise, although it was not specified what was regarded as impulse noise, showed a slight positive, but nonsignificant, association with both prematurity and low birth weight. Individuals reporting noise exposure as a problem had a greater frequency of sick leave than those not reporting noise a problem. The duration of leave of the former group was also longer. However the small sample size reporting noise as a problem (only 26 of 393) make risk estimates difficult and may have inflated the effect of noise spuriously. The authors conclude that noise is not a major adverse influence in advanced pregnancy.

251   Nurminen & Kurppa (1989) examined threatened abortion (as determined by vaginal bleeding), pregnancy induced hypertension, length of gestation and birth weight, in a sample of 1190 mothers. Noise levels were assessed by maternal report and independent assessment by industrial hygienists. One area of interest was that whilst 429 mothers reported their working noise level a problem, independent assessors classed only 102 mothers as working in a noisy environment, defined as above $L_{Aeq8h}=80dB$. Threatened abortion was not associated with noise alone, but when associated with shift work the

risk of spontaneous abortion was greatly increased. Similarly noise associated with shift work resulted in a two-fold increase in the incidence of pregnancy induced hypertension (an increase of 20mmHg during pregnancy) and the duration of pregnancy. There was no effect of noise alone on any of these three factors. Noise did exert an effect on its own for low birth weight. The median birthweight of infants of mothers working in a noisy environment was 3470gms and for mothers working in a quiet environment 3550gms. The authors conclude that threatened abortion, pregnancy induced hypertension, and gestation were not related to noise alone.

252   Saurel-Cubizolles, Kaminski, Mazaubrun and Breart (1991) found an increase in arterial hypertension for pregnant women working in a noisy environment but only during the first and second trimester, not during the third.

253   Finally, Duclos, Lafon, Dubreuil, Olivier & Bergeret (1984) in a study of 192 women in the Lyons area of France between 1979-1980 found that females exposed to high noise levels (assessed by maternal questionnaire) complained more about noise but both the noise level and duration of noise had no effect on gestation length, birthweight, Apgar scores at one and five minutes, foetal pH and bi-parietal diameter. Females who complained more about noise had no more time off work than those who did not.

Summary

254   The results from these studies produce conflicting results. In some noise exposure during pregnancy produces an adverse effect on pregnancy outcome whilst in others no effect is observed. However, the few studies performed in the area and the differences in methodology between them prevent direct comparison between those studies which have been done. Definite conclusions regarding the effect of noise on pregnancy outcome must be drawn cautiously.

255   There appears to be little evidence that noise exposure during pregnancy acts a teratogen. Studies which have examined the incidence of birth defects appear not to find an association with prenatal noise exposure.

256   Perhaps the most studied outcomes have been in terms of low birth-weight and premature birth. Here some studies have reported an effect on birthweight and others have found none. Similarly some studies have reported an effect on prematurity and others have found none.

257   There is no doubt however that noise, along with a variety of other occupational and environmental conditions, acts as a general stressor on the mother inducing a variety of physiological and psychological changes which may affect her pregnancy. Given that a psychological component may be involved in the mediation of the stress effect individual differences in the response to noise may account for the differences in findings reported above. This would suggest that it is not the absolute level of noise that is important but rather the subjective impression of noise by the mother which mediates the effects of noise. Further studies are required to fully determine the effects of noise alone. However it should be noted that, in terms of effects produced on reproductive outcome, noise is not special but one of a group of environmental conditions which may induce stress in the mother and so affect the foetus

Protection and legislation

258   Noise exposure may exert its effect directly on the foetus. That is the inner ear of the foetus responds to the sound and this 'sensation' results in an adverse outcome. If this is the case then the provision of protection to the mother is of little value and noise must be prevented from reaching the foetal ear (see paras. 220-221).

259   The effects of noise produce an automatic activation of the autonomic nervous system which may result in changes in the cardiovascular, endocrinological and immunological systems. It is known that noise is

perceived by the foetus and the foetus responds to it. The extent to which this noise also activates the autonomic nervous system of the foetus and produces the changes observed in the adults is unknown.

260   More probably the effects of noise exposure on reproductive outcome are caused indirectly by noise first producing an effect in the mother which then affects the foetus or the mother's ability to maintain her pregnancy. Noise exposure may result in a number of physiological changes in the mother. These may effect reproductive outcome in two ways: the production of chemical substances by the mother, e.g. catecholamines, cortisol, which cross the placenta from the maternal blood stream and into the foetal blood stream; or by altering the mother's physiology and so affecting her ability to maintain her pregnancy. Noise may induce stress in the mother and any effects on outcome are mediated by this stress reaction.

261   If the effects are a result of noise affecting the mother then the provision of appropriate protective head gear can reduce the incidence of noise impinging on the mother and so reduce the effects of noise on reproductive outcome.

Future studies

262   There is a general need for more studies to address the question of whether prenatal exposure to noise effects reproductive outcome. The above discussion indicates that noise levels in such studies have two components which need to be studied and related to outcome. First is the absolute level of noise exposure. This is obviously important for any legislation and the interpretation of direct effects of the foetus. Second however is the mother's perception of the level of noise. If the effects of noise on reproductive outcome are due to the result of stress then the mother's assessment of noise levels is important. These two aspects of noise need separate study as they appear poorly correlated. Many more mothers report noise as a problem than the absolute measurement of sound levels would predict (Nurminen & Kurppa 1989).

263 Many factors however are associated with reproductive outcome, (maternal age, parity, SES, income, previous obstetric history) and studies need to address these to ensure any effects are the result of noise, rather than other factors.

264 Animal studies have similarly found noise exposure during pregnancy may result in poor reproductive outcome (e.g. Nawrot, Cook & Staples 1980; Takigawa, Sakamoto, Murata & Matsumura 1988). However the value of animal studies must be questioned. Whilst they may, in very general terms, indicate that noise has an effect on reproductive outcome they are unable to give details concerning harmful noise levels or durations (see paras. 146-149). Furthermore the use of animals to model exposure in humans is extremely difficult due to the indirect effects of noise, especially with regard to stress. In humans these effects are mediated by the mother and involve her cognitive and psychological processing abilities. Since these will undoubtedly differ from those possessed by animals the use of animals as models of the effects of noise exposure on reproductive outcome in humans should be questioned.

265 Noise may act in conjunction with other factors, e.g. stress, to influence reproductive outcome. Noise may thus be one of a number of environmental factors which when experienced in combination have a much greater effect on reproductive outcome than when experienced in isolation. The role of noise in this context should be addressed.

SECTION X

CONCLUSIONS

Hearing loss

266   Whether prenatal exposure to noise has a damaging effect on subsequent hearing is difficult to confirm or deny due to the lack of experimental evaluation. Only two studies have examined hearing in children after exposure to noise during their mother's pregnancy. Both studies have methodological drawbacks which make definitive conclusions difficult to draw. At best the experimental evidence is suggestive of the fact that exposure to noise during pregnancy may have an effect on later hearing.

267   Theoretically there can be no doubt that noise exposure during pregnancy of sufficient intensity and duration will exert a damaging effect on the foetus and its subsequent hearing abilities. The auditory system of the foetus is functioning from at least as early as 20 weeks of gestation age and has the potential to be stimulated by a variety of external sounds and noise. However the foetal auditory system may function very differently to that of individuals after birth and any estimation of the effects of noise exposure must be based on the characteristics of the foetal auditory system rather than that of the adult. In this respect the type of damage to hearing arising from prenatal exposure to noise may be different from that arising from noise exposure after birth and classically seen in adults.

268   Only one study addressed the issue of exposure to low frequency sounds during pregnancy on subsequent hearing. The study found that such sounds did exert an adverse effect on hearing, but again certain methodological drawbacks with the study must make the acceptance of this conclusion tentative.

269   The characteristics of the maternal abdomen will have a great influence on sound as it passes from the external environment to the foetus within the womb. In particular the low frequencies (around 250Hz and below) within a

particular sound will pass through unattenuated retaining most their acoustic energy. Higher frequencies will be more attenuated. One may predict that the low frequencies within a broad-band noise will be thus more intense (greater acoustic energy) within the womb than higher frequencies and thus the low frequencies have a greater potential for causing harm.

270 The use of the dB(A) scale in the assessment of prenatal levels of noise exposure should be seriously questioned. The scale is based on the response of the adult ear and not that of the foetus, which is likely to be very different. The scale reduces the contribution of low frequency sounds to the assessment of sound intensity, the very frequencies which may be most likely to cause damage to the auditory system of the foetus. The most appropriate scale would be one which equates to the functioning of the inner ear. However this presents many problems, in particular the fact that auditory sensitivity, and the relationship between thresholds at different frequencies, changes as the foetus matures (Shahidullah 1993) making a single scale inappropriate. Until more is known about the sensitivity of the foetal ear and the effect of the abdomen on sound attenuation an equally weighted dB scale may be the most appropriate scale to be used as this will not underestimate the contribution of low frequency sounds.

271 Any effects of noise exposure on foetus are the result of noise directly impinging on the foetal ear. Thus to prevent damage to the foetal auditory system due to prenatal noise exposure the intensity and/or duration of sound reaching the foetal ear must be reduced. Provision of ear protectors to the mother, whilst reducing her exposure to noise, will have no effect on levels of noise experienced by the foetus and consequently no effect of the risk of damage to the hearing of the foetus.

272 There is a clear need for further investigations of the effects of prenatal noise exposure on the subsequent hearing of the foetus and child. Indeed in order for any legislation to provide adequate protection from prenatal noise exposure it is imperative that information is collected and made available on

the effects of exposure of the foetus to noise.

273   Future studies need to address a number of issues, however the most important is **does prenatal noise exposure damage hearing?** To answer this and other important questions, e.g. what are the intensity levels of noise, and duration of noise exposure, which cause damage? are there particular periods during pregnancy when the ear is more at risk to the damaging effects of noise? a prospective long-term epidemiological study of the effects of prenatal noise exposure in humans is crucial. These studies need to assess both auditory threshold and frequency resolution as both may be affected. Consideration should also be given to other possible effects of exposure to noise during pregnancy such as premature aging of the auditory system in adulthood or increased susceptibility to hearing loss from other risk factors. It also needs to be borne in mind that a smaller loss in hearing may be much more handicapping for the newborn and child, especially for the acquisition of language, than for the adult. Thus assessment of language abilities and other social skills in infancy should be used in the examination of the effects of prenatal noise exposure.

Reproductive Outcome

274   A number of different lines of enquiry have examined the effects of noise exposure on reproductive outcome, although the number of investigations in this area is small. Noise does not appear to be a teratogen, but there is some suggestion that noise exposure may influence prematurity and low birth weight. However methodological drawbacks with these studies and a number of conflicting studies which found no effect of noise mean that a link between noise and a specific effect on reproductive outcome is tentative.

275   The effects of noise may be mediated directly by initially stimulating the auditory system of the foetus but are more likely to be the result of an indirect effect arising from noise exerting an effect in the mother which then affects her foetus. In this latter case the provision of protective ear guards to reduce the levels of noise experienced by the mother may reduce the risk to

reproductive outcome arising from effects induced by noise in the mother. This will of course have no effect on outcomes arising from sound directly impinging upon the foetus.

276   There is a clear need for further study to assess the effects of noise exposure during pregnancy on reproductive outcome. In particular whether noise does exert an effect needs to be assessed. In terms of reducing the risk to the foetus it is important to determine whether any effects arise directly from the foetus's exposure to noise or indirectly from effects initially mediated in the mother.

277   In terms of any effects of noise on reproductive outcome these may be mediated, in part or whole, by stress induced in the mother rather than the effect of noise *per se*. Stress is known to exert an adverse effect on pregnancy. In this case is it important to reduce the stress experienced by the mother in response to noise exposure rather than absolute levels of noise experienced.

APPENDIX 1

<u>Terms of Reference</u>

To conduct a critical review of the published scientific evidence relating to the relationship between noise exposure of pregnant women and the foetus and the subsequent postnatal health and function of the child's auditory system. The review should cover:

  i   papers which describe the theoretical basis for harm

  ii   experimental studies in which pregnant animals are exposed to noise and the effect on the offspring examined. In such studies particular comment is requested on the evidence of the noise exposure in comparison with humans and the validity of the animal model.

  iii   experimental studies in which pregnant women are exposed to noise in a controlled situation.

  iv   epidemiological studies based on the exposure of pregnant women to occupational or other environmental noise sources. In such studies comment is required on the reliability of the reported noise exposure, the observed effects and alleged relationship between these, in particular where more than 1 year has elapsed between the birth of the child and the detection of the effect on the auditory system.

  v   languages to include English, French, German, Japanese and others which the reviewer considers appropriate.

The report should comment on the extent to any harm can be attributed to the direct effect of noise on the foetus and to the indirect consequences of the effects of noise on the pregnant woman (this information will help HSE decide how far any harmful effects might be controlled by use of personal protective equipment such as ear protectors worn by the pregnant woman).

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