THE CREATION OF DISTORTION PRODUCTS THROUGH THE INTERACTION OF SPONTANEOUS OTOACOUSTIC EMISSIONS AND EXTERNALLY GENERATED TONES

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As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Linda Weiland Norrix entitled The Creation of Distortion Products Through the Interaction of Spontaneous Otoacoustic Emissions and Externally Generated Tones

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ABSTRACT

A spontaneous otoacoustic emission (SOAE) and external tones (XTs) were used as primaries f_2 and f_1, respectively (frequency of $f_2 > f_1$) to create $2f_1$-f_2 distortion products (DPOAEs). The DPOAEs primarily reflect active processes in the cochlea because low- to medium-level primaries less than 60 dB SPL were employed. DPOAE characteristics were determined and compared to what is known from the literature about DPOAEs created using two XTs. SOAE frequency and amplitude were also described.

One hundred twenty-two women were screened for the presence of SOAEs to obtain 10 subjects for the study. XTs were presented to the ear at SOAE/f_1 ratios between 1.08 and 1.22. Sound pressure levels were incremented in 5 dB steps and extended from levels equal to the initial SOAE amplitudes to levels at which the SOAEs and DPOAEs disappeared into the noise floor. Amplitude and frequency of the SOAE, XT, and DPOAE were recorded by placing a low-noise microphone in the ear canal and extracted using Fast Fourier Transform analysis.

Results indicated that the DPOAE amplitudes and SOAE suppression characteristics were variable across subjects. Despite the variability, the following trends were noted: 1) at larger frequency ratios, DPOAE generation and SOAE suppression were associated with greater XT levels; 2) DPOAE growth functions were characterized by slopes less than 1 dB/dB, a maximum, rollover and disappearance into the noise floor with increasing XT levels; 3) maximum amplitude DPOAEs were observed at frequencies approximately one-half octave lower than the SOAE; 4) DPOAE thresholds were associated with little SOAE suppression whereas the maximum DPOAEs were associated

with a greater amount of SOAE suppression; 5) the most common SOAE frequency shift, in the presence of XT stimulation, was a shift to a higher frequency just prior to total SOAE suppression. Results are interpreted using an electrical circuit model (modified from Yates, 1990) and pictorial representations of the SOAE, XT, and DPOAE vibrational patterns on the basilar membrane (modified from Yates, Geisler, Patuzzi, & Johnstone, 1989).

INTRODUCTION

The discovery of otoacoustic emissions (OAEs) has been associated with dramatic changes in the way in which the cochlea is thought to function. This review highlights some of the important developments in theories of cochlear mechanics that preceded and followed the discovery of otoacoustic emissions. Next, the various types of OAEs are reviewed along with a brief description of the instrumentation needed to record them. Finally, studies important in the development of the present investigation are summarized and the research questions are stated.

Classic Model of Auditory Function

The classic view of sound processing within the cochlea assumes a unidirectional flow of energy from the periphery to the central nervous system. Briefly, acoustic energy in the form of sound waves enters the ear canal and impinges on the tympanic membrane causing it to vibrate. This energy, now in the form of mechanical energy, is transferred to the three middle ear ossicles, the first of which is coupled to the tympanic membrane. The third ossicle, the stapes, is coupled to the oval window of the inner ear. Displacement of the stapes in and out of the oval window results in energy transfer to the cochlea. The hydromechanics of the cochlea produce shearing of the stereocilia of the sensory receptors. This shearing transduces the hydromechanical energy into an electrochemical signal that is introduced to the VIII nerve, which in turn provides a stimulus to the central auditory nervous system.

Passive Cochlear Mechanics

Vibration Patterns of the Basilar Membrane

Many of the early findings describing cochlear function come from the work of von Bekesy completed in the 1950's. Using both models and cadaver specimens of the inner ear, von Bekesy (1960) observed and described what he called a "traveling wave" of the basilar membrane in response to intense vibratory stimulation. He observed that for any frequency of stimulation the movement of the basilar membrane first appears near the base. The vibration reaches a maximum at a particular place along the length of the basilar membrane. After reaching its peak, the traveling wave quickly dissipates due to the viscous and other damping forces within the cochlea. The place of maximum vibration is frequency dependent. High frequency signals correspond with maximum displacement at the basal end of the cochlea while low frequency vibrations drive more apical parts of the cochlea.

The envelope of the traveling wave described by von Bekesy is consistent with a basilar membrane that has broad hydromechanical movement patterns. The rise in amplitude of the traveling wave occurred at approximately 6 dB/octave while the decay of the traveling wave was about 12 dB/octave (von Bekesy, 1960).

Zwislocki (1950) attributed the characteristics of the traveling wave to the passive elements of basilar membrane elasticity and damping within the cochlea. The base, which is narrow and stiff, responds preferentially to high frequencies and begins vibrating before the apex, which is wide and flaccid and thus cannot respond as rapidly as the basal region. In addition, high levels of damping within the cochlea cause the traveling wave to decay rapidly after

reaching its maximum. However, the passive and linear elements of cochlear mechanics, described by von Bekesy (1960) and Zwislocki (1950), cannot account for two phenomena: the dynamic range and frequency discrimination of normal hearing.

Dynamic Range of Normal Hearing

Von Bekesy's (1960) recordings of the vibration of the basilar membrane at high intensities have been used to estimate the movement of the basilar membrane near threshold levels. However, linear extrapolation from high intensity responses to threshold level responses results in estimates that are far below molecular dimensions (e.g., 0.003 angstroms at threshold for a 3 kHz stimulus) (Moller, 1983). In contrast, the threshold of stimulation of inner hair cells is approximately 3.5 angstroms movement of the basilar membrane (Sellick, Patuzi, & Johnstone, 1982). Therefore, it is implausible that linear changes in the movement patterns of the basilar membrane can account for threshold level responses.

Frequency Resolution

The auditory system demonstrates exquisite frequency resolution. For example, in the mid-frequency region (0.5 - 4 kHz for humans), normal-hearing individuals are able to detect a 0.3% change in frequency. The discrepancy between a broad pattern of basilar membrane movement and good frequency discrimination motivated von Bekesy (1967) to speculate that the central nervous system sharpened the response. In the 1960's and 70's, researchers discovered that VIII nerve fibers, which are part of the peripheral auditory system, were themselves very sharply tuned (Evans & Wilson, 1975; Kiang, Watanabe, Thomas, & Clark, 1965). Frequency tuning curves from VIII nerve

fibers that receive innervation from the basal end of the cochlea exhibit very steep high frequency slopes (up to 600 dB/octave) and low frequency slopes of approximately 180 dB/octave (Evans, 1972). However, VIII nerve fibers have direct one-to-one connections with inner hair cells and are therefore not anatomically positioned to serve a sharpening function (Dallos, 1988). This led Evans (1972) to speculate about the existence of a "second filter" within the cochlea that sharpens the tuning of the basilar membrane.

Modified Passive Cochlear Mechanics

Nonlinearities and Sharp Tuning Within the Cochlea

Studies using the Mossbauer or capacitance probe techniques to measure cochlear mechanical activity at low to medium sound pressure levels (SPLs) have reported that nonlinearities occur in the vibration patterns of the basilar membrane in chinchillas (Robles, Ruggero, & Rich, 1986), squirrel monkeys (Rhode, 1971; Rhode & Robles, 1974) and guinea pigs (LePage & Johnstone, 1980; Sellick et al., 1982). Studies employing such techniques generally describe the vibration amplitude of a small length of the basilar membrane under "in vivo" conditions. Rhode (1971) presented evidence that the amplitude of vibration was enhanced at medium-intensity levels when the characteristic frequency (CF) of a specific position along the basilar membrane was employed as a stimulus. He displayed input/output functions relating basilar membrane displacement to malleus displacement as a function of frequency for three SPLs (70, 80, 90 dB). The increased gain at the CF produced displacements that were 10 to 20 times greater than the displacement of the malleus.

The amplification of the response at the CF is also related to precise basilar membrane tuning. In 1978, Russell and Sellick identified stimulus levels needed to produce a criterion intracellular voltage (i.e. 2 mV) as a function of frequency. They found sharp frequency tuning curves for potentials recorded from healthy inner hair cells. As methodologies such as the Mossbauer technique were developed and improved, researchers were able to measure movement of the basilar membrane using live specimens and low SPLs. As predicted by Evans (1972), studies using the Mossbauer technique, low intensities, and experimental procedures that minimized cochlear trauma have demonstrated that the envelope of the basilar membrane displacement pattern is sharper than the Bekesy traveling wave envelope (Khana & Leonard, 1982; Robles et al., 1986; Sellick et al., 1982; Sellick, Yates, & Patuzzi, 1983).

The vibration patterns of a point on the basilar membrane are characterized by saturating nonlinearities when stimulated with a frequency corresponding to its CF. Saturating nonlinearities are often examined by studying velocity of the basilar membrane rather than displacement. Sellick et al. (1982) suggested that basilar membrane velocity can be appropriately compared to afferent activity or neural tuning curves because inner hair cells, which are innervated by 95% of the afferent neural supply rising from the cochlea, appear to respond to velocity of motion rather than amplitude of displacement. Robles et al. (1986) measured basilar membrane velocity at various SPLs in the chinchilla cochlea. They found that the input-output functions were nonlinear for frequencies near CF with a slope less than one. Frequencies lower or higher than CF had linear input-output functions.

Another type of nonlinearity results when a physical system is overdriven, the change in response amplitude fails to follow the change in input amplitude, and new oscillations occur at frequencies other than those produced by the driving force. These new oscillations are called distortion products (DPs). DPs produced by the auditory system have been studied for many years. One of the first types of distortion products to be observed was *harmonic distortion.* Helmholtz (1870) recorded harmonic distortion when the ear was stimulated with high SPLs. According to Wever and Lawrence (1954) Helmholtz attributed this distortion to mechanical overloading within the middle ear. He believed that aural distortion was caused by the actions of the eardrum and the malleoincudal joint. At low and moderate SPLs *intermodulation distortion* is observed (Goldstein, 1967). Intermodulation distortion may be detected when two pure tones of particular frequencies and intensities are presented to the ear. The output components have frequencies that consist of the sums and differences of the original pure-tone frequencies and multiples of those frequencies. The most prominent intermodulation distortion tone is the $2f_1$-f_2 cubic difference tone, where f_1 and f_2 are the frequencies of the two external tones and f_2 is higher in frequency than f_1. This DP has been detected psychoacoustically (Goldstein, 1967), revealed in recordings obtained from VIII nerve fibers (Goldstein & Kiang, 1968; Kim, 1980), and detected in the cochlear microphonic (Dallos, 1969). More recently $2f_1$-f_2 has been recorded acoustically in the ear canals of individuals with normal hearing sensitivity (Kemp, 1979). These findings support the idea that the cochlea operates in an inherently nonlinear manner at low to moderate stimulus levels.

It is impossible to explain the sharp tuning and nonlinearities by considering only passive basilar membrane characteristics such as mass and stiffness. The sharp tuning of the basilar membrane, due to the increased gain at CF, and the nonlinearities are vulnerable to mechanical insults. This has been shown in chinchillas (Robles et al., 1986), squirrel monkeys (Rhode & Robles, 1974), and guinea pigs (LePage & Johnstone, 1980; Sellick et al., 1982). In dead or unhealthy ears the vibration patterns of the basilar membrane become very broad resembling the Bekesy traveling wave and the nonlinearities disappear. In addition, the most effective stimulus for the point of measurement shifts to a lower frequency in damaged cochleae (Rhode & Robles, 1974; Sellick, Patuzzi, & Johnstone, 1983). The findings of sharp tuning and nonlinearities within the healthy cochlea led researchers to speculate about the existence of a filter within cochlear micromechanics (Evans, 1972; Hall, 1980). Furthermore, because it is extremely dependent on the health of the cochlea, researchers speculated that this filter would involve an active, metabolic process (Dallos, 1988; Davis, 1983).

Active Cochlear Mechanics

The hypothesis that an active mechanical system exists within the cochlea was advanced by Gold in 1948. However, researchers first began investigating and describing such a system in 1978, when Kemp discovered OAES. The discovery of OAEs led to modifications of the classic model of cochlear function. The cochlea is now viewed as a system associated with bidirectional flow of energy (Davis, 1983). Not only is energy transmitted from the periphery to the central nervous system but also a reverse traveling wave is generated within the cochlea, transmitted through the middle ear as mechanical

energy and through the ear canal as sound waves and measured as OAEs. These OAEs are not precursors to sensation but rather represent epiphenomena of the hearing process.

Davis (1983) called the active mechanism within the cochlea believed to be responsible for OAEs the "cochlear amplifier. " According to Davis, the cochlear amplifier (CA) acts at low stimulus levels "to enhance the vibration of a narrow segment of the basilar membrane" (p. 81). This enhancement overcomes some of the viscous damping forces within the cochlea and increases sensitivity by about 45 dB for the CF (Davis, 1983). It occurs at a position along the basilar membrane corresponding to a lowering of stimulus frequency by approximately one-half octave with a corresponding shift of the traveling wave envelope toward the apex of the cochlea.

Models of cochlear function must include mechanisms to account for the enhanced basilar membrane vibrations observed at low stimulus levels. Several models of cochlear function incorporate such mechanisms (Geisler, 1986; Kim, 1980; Neely & Kim, 1983; Zweig, 1991; Zwicker, 1986a). Most models propose some form of feedback and/or negative damping. For example, Zweig speculated that outer hair cells (OHCs) are force generators, with fast positive feedback, that create negative damping. If these active forces are in phase with the excitation, they will oppose the damping forces within the cochlea (Brass & Kemp, 1993) and feed back energy into the cochlear partition at a position corresponding to the driving frequency. That is, mechanical energy is supplied to the traveling wave near its maximum to counteract the damping effects present within the cochlea. The result is a selective boost in basilar membrane vibration near the maximum. A larger vibration amplitude in

a very narrow segment of the basilar membrane would be a more effective stimulus for the corresponding inner hair cells (IHCs), which receive about 95% of the afferent innervation. Anderson and Kemp (1979) suggested that a larger traveling wave could increase the energy available to the IHCs either directly through greater fluid flow within the subtectorial space or indirectly by a reduction in basilar membrane damping at CF. Several characteristics of OHCs make them ideally suited for the role of amplifying the Bekesy traveling wave.

Anatomical Evidence

OHCs, unlike IHCs, are located on the flexible basilar membrane. The tallest of their stereocilia are embedded within the underside of the tectorial membrane and provide a direct mechanical coupling between the tectorial membrane and basilar membrane. A local length change in the OHCs could supply energy to the basilar membrane/tectorial membrane system and contribute to amplification of the traveling wave over a very narrow segment.

Physiological Evidence

Brownell, Bader, and de Ribaupierre (1985) found that isolated OHCs from the guinea pig changed shape in response to chemical, electrical, and mechanical stimulation. These authors, however, were unable to observe movements from an IHC or supporting cell. Although OHC length changes have not been observed "in vivo", it has been postulated that their motility could alter the micromechanics of the organ of Corti (Dallos, 1988, 1992; Geisler, 1986).

Psychoacoustical Evidence

Other evidence that suggests OHCs are necessary for the CA to function comes from animal studies that have involved systematic destruction of OHCs.

Loss of OHCs is associated with a loss of hearing sensitivity of about 40-50 dB in the frequency region that corresponds to the cochlear position of missing OHCs (Dallos & Harris, 1978; Ryan & Dallos, 1975).

Electrophysiological Evidence

Electrophysiological evidence links OHCs with the CA. Destruction of these cells in the guinea pig and chinchilla is associated with decreased frequency selectivity as evidenced by broad cochlear nerve tuning curves with missing tips (Evans & Harrison, 1976; Liberman & Dodds, 1984).

Acoustical Evidence

By-products of the amplification process such as intermodulation distortion ($2f_1$-f_2) and OAEs provide evidence that OHCs are associated with the CA. These byproducts are reduced or eliminated when the cochlea is exposed to metabolic toxins or noise that presumably destroy or damage OHCs (Anderson & Kemp, 1979; Lonsbury-Martin, Martin, Probst, & Coats, 1987). More direct evidence associating the disappearance of OAEs with missing OHCs has also been reported (Zurek, Clark, & Kim, 1982).

The Measurement of OAEs

OAEs appear to be a significant by-product of the amplification process, a process that results in good auditory sensitivity and fine frequency resolution at low and moderate levels of stimulation (Lim, 1986). They are easily recorded by placing a miniature microphone in the ear canal and provide an objective and relatively noninvasive tool for probing cochlear function at low to moderate stimulus levels.

OAEs are low-intensity sounds that are embedded in larger amplitude noise within the ear canal. Instrumentation used to record OAEs is designed to

extract them from the ear-canal noise. The technology shares many features with technology used to record evoked potentials, which are also low amplitude signals embedded in larger amplitude noise.

The recording probe assembly consists of miniature transducers, one microphone and one or two receivers. A low-noise microphone is necessary for the recording of the OAEs. Earphone receivers may or may not be employed during the recording session depending on the OAE of interest. Only those OAEs that are evoked by external sounds require use of the earphones. The probe assembly is coupled to the ear canal via a rubber or foam meatal plug.

The acoustic signals are detected by the microphone and converted to electrical signals. The electrical signal is typically amplified and filtered prior to analog-to-digital (A-to-D) conversion and averaging. Both high-pass and low-pass filters are used. High-pass filters with cut-off frequencies around 400 Hz are necessary to reduce low-frequency background noise. Low-pass filters (with cut-offs set at one-half the A-to-D sampling rate) are necessary to reduce artifacts related to aliasing. Averaging can be performed in the temporal or frequency domain, depending on the OAE of interest.

Types of OAEs

There are two general categories of OAEs: spontaneous OAEs and evoked (or stimulated) OAEs (Glattke & Kujawa, 1991). Characteristics of these two categories of emissions will be briefly described. The reader is referred to several excellent reviews of the various classes and characteristics of OAEs (Glattke & Kujawa, 1991; Martin, Probst, & Lonsbury-Martin, 1990; Probst, 1990).

Spontaneous Otoacoustic Emissions (SOAEs)

SOAEs are narrowband signals with amplitudes generally less than 20 dB SPL, that are recorded in the absence of external stimulation. Dallos (1992) suggests that SOAEs provide the strongest evidence for an amplification process occurring within the cochlea. These emissions are detected in approximately 40% of normal-hearing individuals (Bright & Glattke, 1986), and have been recorded more frequently in women than in men (Probst, 1990; Talmadge, Long, Murphy & Tubis, 1993). SOAEs tend to be observed more commonly in the mid frequencies, possibly because of the reverse transfer function of the middle ear which is maximum in the region between 1 and 2 kHz (Kemp, 1980; Wilson, 1980). Most ears with SOAEs have multiple SOAEs rather than just one (Martin et al., 1990). SOAE frequency is quite stable over time (Probst, 1990). In contrast, SOAE amplitude is more variable (Probst, 1990). Because SOAEs are present in close to half of the population with normal audiometric thresholds, they are not sensitive as a test for peripheral hearing loss.

Although the exact origin of SOAEs is unknown, several theories exist. Martin et al. (1990) and Probst (1990) suggested that because of their frequency stability SOAEs are generated by a structural feature at a specific place on the basilar membrane. In one non-human primate that exhibited SOAEs, Lonsbury-Martin, Martin, Probst, and Coats (1988) found irregularities in OHC arrangement especially in the apical part of the cochlea. These irregularities included disorganization in the arrangement of OHC stereocilia and the presence of a fourth row of OHCs. Lonsbury-Martin et al. suggested that normal structural irregularities are in some way associated with SOAEs. In

human studies, SOAEs are generally not detected in frequency regions that exhibit hearing loss. Therefore, if structural irregularities are associated with the generation of SOAEs in humans, these irregularities are not associated with audiometric hearing loss detected by conventional threshold audiometry.

Structural irregularities along the cochlear partition do not necessarily imply poor cochlear function. In fact, some authors have suggested that SOAEs are a sign of a healthy cochlea (Bonfils, 1989; Moulin, Collet, Delli, & Morgon, 1991). Moulin et al. (1991) obtained audiometric results for 126 ears of 63 subjects and screened them for the presence of SOAEs. Findings indicated that subjects with SOAEs, in contrast to those without SOAEs, exhibited less hearing loss at all octave frequencies between 0.25 and 8 kHz. In addition, SOAEs were never found when hearing loss at 1 kHz exceeded 10 dB HL. Alternatively, SOAEs may be due to an unstable micromechanical control system. In referring to the moment between cancellation and amplification of energy within the cochlea, Kemp (1986) suggested that "the human ear often crosses the line and becomes unstable at specific frequencies resulting in spontaneous emissions" (p. 99). The source of an unstable micromechanical control system remains unclear. Some of the possible sources include structural irregularities within the cochlea or the auditory efferent system that comprises the majority of OHC connections.

Evoked OAEs

Evoked OAEs require external stimulation and are related to the stimulus. There are three classes of evoked emissions: transiently evoked otoacoustic emissions (TEOAEs), stimulus-frequency otoacoustic emissions (SFOAEs), and distortion product otoacoustic emissions (DPOAEs). The amplitude of all three

of these types of emissions depends partly on stimulus level, size of ear canal, frequency response of the middle ear and presence of SOAEs. For example, evoked OAEs are usually larger at frequencies that also exhibit an SOAE (Furst, Rabinowitz, & Zurek, 1988; Wilson, 1980).

Transiently evoked otoacoustic emissions (TEOAEs)

TEOAEs were first described by Kemp in 1978. They can be recorded after stimulating the ear with a transient stimulus such as a click or toneburst. Temporal averaging of the acoustic waveform after the brief stimulus helps to reduce the ear canal noise by a factor of the square root of the number of samples. The response reflects spectral characteristics of the eliciting stimulus. For example, click-evoked TEOAEs contain energy that is distributed across the frequency spectrum. TEOAEs resulting from tone bursts contain energy around the frequencies of the eliciting stimulus. Additionally, the latency of the emission is related to the frequency composition of the emission, which, in turn, mirrors the distribution of energy in the stimulus and the time delay imposed by cochlear mechanical events. In general, when compared to the high frequency components of a response, the low-frequency components appear after a greater latency . TEOAEs exhibit nonlinear input/output functions that saturate at moderate levels of stimulation. These emissions can be recorded in almost all individuals for whom audiometric thresholds are better than 30 dB HL (Probst, 1990). TEOAEs are stable in frequency and replicable within an individual. Many TEOAE patterns have been reported among individuals with normal hearing thresholds, each exhibiting dominant peaks at various frequencies (Robinette, 1992).

Stimulus frequency otoacoustic emissions (SFOAEs)

SFOAEs are due to energy originating in the cochlea that is added to the energy of the stimulus. The evoking stimulus for this type of emission is a continuous low-level pure tone swept in frequency. The emission occurs at the frequency of the stimulus and at approximately the same intensity. Sweeping the stimulus frequency produces phase changes between the stimulus and the emission. This results in SFOAEs that are individually and uniquely characterized by peaks and valleys with amplitudes between -20 and +10 dB SPL (Schloth, 1982). SFOAEs are present in approximately 90% of the population with normal hearing and saturate at moderate stimulus levels (Probst, 1990). Because SFOAEs are difficult and time consuming to record they are not used clinically.

Distortion product otoacoustic emissions (DPOAEs)

DPOAEs are evoked by presenting two primary pure tones, that are close in frequency, into the ear canal (f_1 and f_2 where f_2 is the primary tone higher in frequency than f_1). Two earphones are needed to generate this OAE. A spectral analysis of the resulting signal, such as a fast fourier transform (FFT), is used to detect DPOAEs. Energy is present at a frequency that was not present in the stimulus. The most robust DPOAE is the cubic, or $2f_1 - f_2$ distortion product. The typical amplitude of the $2f_1$-f_2 DPOAE, when the level of f_1 equals f_2, is 40 dB or more below the level of the primaries. However, the absolute amplitude depends on the f_2/f_1 ratio, and levels of the primary stimuli. These emissions can be detected for frequencies between 1 and 8 kHz (Martin et al., 1990) and can be recorded in almost all individuals for whom audiometric thresholds are less than 30 dB HL (Probst, 1990). Because individual

frequencies can be tested, DPOAEs are potentially useful for evaluating the function of discrete regions of the cochlea.

To associate the function of particular areas in the cochlea with the behavior of $2f_1$-f_2, the site of generation of $2f_1$-f_2 must be known. Currently it is believed that $2f_1$-f_2 is generated near the primaries, then propagates to its characteristic place which is apical to the cochlear region that is responsive to the primaries. Martin, Lonsbury-Martin, Probst, Scheinin, and Coats (1987) studied $2f_1$-f_2 generation in rabbits, using primaries at 65 dB SPL or less, and a f_2/f_1 ratio of 1.25. They performed three separate manipulations to help determine the origin of DPOAEs. First, suppression tones at various frequencies and intensities were introduced into the ear canal along with the two primaries. The magnitudes of external tones that reduced DPOAEs by 6 dB were plotted as "suppression contours." Results indicated that suppression of the DPOAE was greatest when the suppressor tone was located near the frequencies of the primary tones. The authors stated that smoothing of the isosuppression function revealed that the position corresponding to the geometric mean of the two primaries was the most sensitive region of the cochlea, where the suppressor tone could have its greatest impact on DPOAE amplitude. A second manipulation involved the addition of a moderate level tone (interfering tone) to the ear canal along with the two primaries. In this case the interfering tone, at 5.962 kHz, was held constant and the primary frequencies were varied. DPOAEs were produced for primary tones in the region from 1 to 12 kHz both in the absence and in the presence of the interfering tone. Results revealed the DPOAE amplitude was greatly reduced when the primary tones were centered around the interfering tone. In contrast,

DPOAE amplitude was only slightly reduced when the distortion product itself fell within the frequency region of the interfering tone. A final manipulation involved exposing the ear to pure tone fatiguing stimuli at 90 dB SPL for 3 minutes at the frequency of either f_1, f_2, the DPOAE, or 1/2 octave below each of these frequencies. Presenting the tones 1/2 octave below f_1, f_2, or the DPOAE was expected to position the fatiguing signal on the cochlear partition corresponding to the frequency of interest (i.e., f_1, f_2 or the DPOAE). DPOAEs at 2 and 4 kHz were examined. For the 2 kHz DPOAE, tone exposure 1/2 octave below f_2 was associated with the greatest amplitude reduction of the DPOAE. For the 4 kHz DPOAE, tone exposure 1/2 octave below f_1 or f_2 equally reduced the amplitude of the DPOAE. The least effective condition for reducing the amplitude of both the 2 and 4 kHz DPOAEs was tone exposure at f_2 and 1/2 octave below the DPOAE of interest. The authors suggested that the cochlear position between the locations driven by the primaries contributed primarily to the generation of $2f_1$-f_2. Results for the 4 kHz DPOAE suggested that mechanical disturbances of both primaries may contribute to DPOAE generation while the f_2 place appeared to have contributed more to the 2 kHz DPOAE.

Brown and Kemp (1984) also examined DPOAE generation in gerbils and in human subjects using levels of stimulation between 40 and 60 dB SPL and f_2/f_1 ratios approximately 1.32:1. They examined three frequency regions within the gerbil cochlea corresponding to f_2 (i.e., 1.75, 3.5, and 5.8 kHz). For human subjects, one frequency region near 1.85 kHz was studied. Brown and Kemp found that tones near f_2 were more effective in suppressing the $2f_1$-f_2

than tones near f_1. The authors suggested that f_2 is the most important site for $2f_1$-f_2 generation at the levels of stimulation used in their study.

DPOAE Characteristics as a Function of Primary Level

According to Martin et al. (1990) the DPOAE response grows linearly until the primary tones reach 60-70 dB SPL. The DPOAE response saturates for stimuli approximately 70 dB SPL and above. Bonfils, Avan, Londero, Trotoux, and Narcy (1991) examined DPOAE growth in humans using equilevel primaries less than 1130 Hz and f_2/f_1 ratios between 1.18 and 1.26. They used an iso $2f_1$-f_2 paradigm ($2f_1$-f_2 frequency was held constant while f_1 and f_2 were varied). The growth functions exhibited saturating behavior when the primaries were between 30 and 60 dB SPL and exhibited a linear growth pattern when the primaries were above 66 dB SPL. Although high level primaries (78 dB SPL) were associated with the largest amplitude DPOAEs, the authors noted that the DPOAE amplitudes appeared to be independent of f_2/f_1 ratios. In contrast, 48 dB SPL primaries were associated with largest DPOAE amplitudes when the ratio of f_2 to f_1 was between 1.22 and 1.26. Bonfils et al. suggested that low-frequency DPOAEs generated by stimuli below 60 dB SPL are probably associated with the saturating properties of OHCs while those generated by high-level primaries reflect the passive properties of the cochlea. Koppl, Manley, and Johnstone (1993), studying the bobtail lizard, also found evidence that DPOAEs generated by low-level and high-level primaries respond differently when external tones are applied to the ear canal. The tones that were most effective in suppressing DPOAEs generated with low-level primaries (<50 dB SPL) were close in frequency to f_1. In contrast, the most

effective suppressor tones for distortion products generated with high-level primaries (>50 dB SPL) were shifted to about 1/2 octave below f_1.

Whitehead, Lonsbury-Martin, and Martin (1992) found evidence for more than one source contributing to DPOAE generation in rabbits. The source with the largest contribution depended not only on intensity levels of the primaries but also on the frequency separation of f_2 and f_1. One source was dominant above 60-70 dB SPL provided there was a moderate frequency distance between f_1 and f_2 (e.g., a f_2/f_1 ratio of 1.37) or at all stimulus levels with a closely spaced f_2/f_1 ratio (e.g., 1.05). The second source contributed to DPOAE generation when primaries were less than 60 dB SPL and moderately spaced or at all stimulus levels when the primaries were widely spaced (e.g., 1.57). Whitehead et al. based their conclusions on several findings. First, they noticed notches in DPOAE growth function around 55-70 dB SPL but not below these levels. They concluded that irregularities in the growth functions were due to phase cancellation of two sources with approximately equal amplitudes. The absence of irregularities below 55 dB SPL indicated that these DPOAEs were dominated by a single distortion generating source. A second line of evidence for more than one source came from three-dimensional graphs that illustrated DPOAE amplitude as a function of the level of f_1 and f_2. Distinct maxima were observed to be associated with low-level primaries and high-level primaries. The boundary between the two maxima was approximately 55-70 dB SPL. Discontinuities in the patterns of growth of DPOAEs and phase functions have also been observed in humans. For example, Popelka, Osterhammel, Nielsen and Rasmussen (1993) suggested that the nonmonotonicities that were present

for 9% of the subjects in their study were due to two sources of distortion product generation.

Several authors have observed DPOAEs generated using high-level primaries in damaged or dead cochleae. Lonsbury-Martin et al. (1987) examined DPOAEs generated with 65 and 75 dB SPL primaries in the rabbit. They found that with anoxia and death, DPOAEs generated with 75 dB SPL primaries persisted over an hour longer than those generated with 65 dB primaries. Zurek et al. (1982) found DPOAEs in a chinchilla with a noise-damaged cochlea when primary levels were 85 or 90 dB SPL. In contrast, DPOAEs were absent for primary tones at lower levels.

Optimal F_2/F_1 Frequency Ratio using Medium- to High-Level Primaries

Although little is known about DPOAEs generated with low-level primaries, many researchers have examined DPOAE characteristics using primary levels above 40 dB SPL and more commonly above 60 dB SPL. When DPOAEs are generated using medium- to high-level primaries, the maximum DPOAE level depends on the f_2/f_1 ratio as well as on the level difference between the primaries.

There is general agreement that the optimal f_2/f_1 ratio for generating the $2f_1$-f_2 is approximately 1.2:1. Gaskill and Brown (1990) found that this optimal ratio only varied slightly with changes in frequency, stimulus level or subject (for stimulus levels from 40 to 65 dB SPL). When DPOAE amplitude is plotted as a function of f_2/f_1 ratio, holding SPL constant, the curves are nonmonotonic (Gaskill & Brown, 1990; Harris, Lonsbury-Martin, Stagner, Coats, & Martin, 1989). If the frequency separation is increased or decreased beyond the

optimal ratio, the DPOAE amplitude declines. This decline occurs at a greater rate for f_2/f_1 ratio changes in the high-frequency region than in the low-frequency region (Gaskill & Brown, 1990; Harris et al., 1989). Several theories exist to explain this nonmonotonic behavior. A decrease in DPOAE amplitude with a large frequency separation has been explained by insufficient interaction between basilar membrane traveling wave envelopes associated with the primaries (Gaskill & Brown, 1990). Kemp (1986) suggested that a small frequency separation results in a single traveling wave peak which is modulated by the beat frequency (f_2-f_1). The DPOAE in this case is very close in frequency to the primaries and is inefficiently propagated toward the base of the cochlea. Alternatively, with a small ratio, f_1 and f_2 could be mutually suppressing each other or suppressing activity at the place where the DPOAE is generated (Manley & Koppl, 1993). A third suggestion was offered by Brown and Gaskill (1990). These authors speculated that for primaries close in frequency, either the basilar membrane passively filters the DPOAE or that some other structure, such as the tectorial membrane, filters the response before it reaches the ear canal.

At high levels of stimulation (e.g., 85 dB SPL) the optimal frequency ratio becomes slightly larger than 1.2 (Harris et al., 1989) and the attenuation of the DPOAE, as a function of f_2/f_1, ratio becomes more gradual than at low levels of stimulation (Gaskill & Brown, 1990; Harris et al., 1989). Gaskill and Brown suggest that high-level primaries are associated with broadly-tuned traveling wave envelopes. Motion patterns to the two primaries would therefore be able to interact for larger frequency separations.

Optimal Level Difference Between Primaries

DPOAE amplitudes depend on the differences in SPL of the primaries. When the level of f_2 is set at about 40 dB SPL, the largest DPOAEs are observed when the level of f_1 (L1) exceeds the level of f_2 (L2) by 10-15 dB SPL. As the level of L_2 increases, maximum DPOAE amplitudes are observed with smaller primary level differences. In contrast, lower-level primaries require larger L1-L2 differences (Gaskill & Brown, 1990). Kemp (1986) suggested that the largest DPOAE amplitudes are observed when the two stimuli result in nearly equal vibrational amplitudes over a particular area of the basilar membrane. Therefore, the optimal L1 to L2 difference depends on the frequency separation of the primaries. The optimal level difference between the primaries increases as the f_2/f_1 ratio gets larger (Brown & Gaskill, 1990).

The optimal level difference and optimal ratio change when using primary tone levels that are between 40 and 90 dB SPL. Very little is known about DPOAE characteristics created using primary levels less than 40 dB SPL. For example, Gaskill and Brown (1990) found it difficult to generate DPOAEs using L2 levels less than 25 dB SPL and L1 levels less than 40 dB SPL. However, a unique situation occurs in ears with SOAEs. DPOAEs can be generated when two low-level SOAEs act as primaries.

DPOAEs Resulting From Two SOAEs

A small number of investigations have involved the study of DPOAEs that appear to be created by the interaction of cochlear mechanical activity patterns that produce two SOAES. Because the SOAEs are very low in amplitude (generally < 20 dB SPL) the DPOAEs created likely represent active nonlinear processes within the cochlea. Jones, Tubis, Long, Burns, and Strickland (1986)

examined this phenomenon in seven subjects and referred to the intermodulation DPs resulting from two SOAEs as "satellite" SOAEs. The satellite SOAEs were distinguishable from independent SOAEs because their frequencies followed frequency shifts in the primaries. Satellite SOAEs could be eliminated by suppressing f_1 or f_2. Conversely the satellite SOAE itself was much more difficult to suppress by an external tone without also suppressing f_1 or f_2. Frequencies of f_1 ranged from 1225 to 1745 Hz while f_2 ranged from 1331 to 2171 Hz. The f_2/f_1 SOAE ratios ranged from 1.06 to 1.24 with a median of 1.11. Talmadge et al. (1993) also observed 18 emissions which could have been DPOAEs resulting from the interaction of two SOAEs. In most cases the f_2/f_1 ratio was less than 1.2. Jones et al. (1986) found "satellite SOAEs" that were 6 to 18.5 dB below the level of the lower frequency SOAE (f_1) while Talmadge et al. found DPOAEs to be 4.7 dB below the f_1 SOAE. This is in contrast to DPOAEs created using two XTs. As noted previously, DPOAEs created by two XTs are generally 40 dB or more below the level of the primaries.

When examining DPOAEs generated by two SOAEs, parameters such as the frequency ratios and levels of f_2/f_1 cannot be manipulated and descriptive data are based on circumstances that are beyond control of the investigator. By substituting an XT for one SOAE, nonlinear process related to cochlear function can be evaluated more systematically. If the SOAE is designated f_2 and one XT is applied to the ear canal, DPOAEs can be created using low to medium levels of f_1. Furthermore, this paradigm (the SOAE for f_2 and an XT for f_1) probes the active processes of the cochlear amplifier for the following reasons:

1. The cochlear amplifier is on (albeit in a state of feedback) at the f_2 site. Because energy is not being added at the frequency of f_2, the contribution from f_2 to DPOAE generation is a result of the active cochlear amplifier rather than the Bekesy traveling wave.
2. Low- to medium-level primaries are more likely to probe the active cochlear amplifier prior to saturation than are high-level primaries that generate passive hydro-mechanical response.

Several researchers have examined the interactions of SOAEs and XTs (Frick & Mathies, 1988; Long, Sun, & Talmadge, 1993; Zurek, 1981). Long et al. (1993) suggested that XTs applied to the ear canal can interact with a SOAE in at least three ways. They can beat with the SOAE, force the SOAE to oscillate at the frequency of the XT, or suppress the SOAE.

Acoustic Beats and Synchronization

Flottorp (1953) and Ward (1955) reported on psychoacoustic beats produced by an XT and a hypothesized internal tone (which they called an "idiotone"). They described this phenomenon as "monaural diplacusis." Monaural diplacusis is a perceptual phenomenon in which, at low SPLs, a pure tone elicits sensations of beats, roughness, and multiple tones. Zurek (1981) was one of the first researchers to demonstrate objectively and record acoustic beats due to the interaction of an XT and a SOAE. Beats of 6/s were detected by Zurek when he applied an XT of 1899 Hz to his ear, in the presence of a SOAE at 1906 Hz. The SOAE and XT were nearly equal in amplitude. Beat rate decreased to 3/s when the XT was increased by 7 dB. Long et al. (1993) also reported that beating is maximal when the stimulus level is similar to the SOAE level and decreases with XT level increases. These authors also

reported that as the frequency of the XT approached the SOAE frequency, partial synchronization occurred and finally the SOAE synchronized to the external tone.

Suppression of a SOAE

Suppression, measured as amplitude reduction, has been demonstrated for SOAEs (Frick & Matthies, 1988; Long, Sun, & Talmadge, 1993; Rabinowitz & Widin, 1984; Ruggero, Rich & Freyman, 1983; Schloth & Zwicker, 1983; Tooley, 1993; Zizz & Glattke, 1988; Zurek, 1981). It is common to depict results of suppression studies by plotting the frequency/intensity combinations required to suppress a SOAE by a certain criterion relative to its amplitude in quiet. These plots are known as suppression tuning curves and are indications of the frequency specificity of the cochlea (Figure 1).

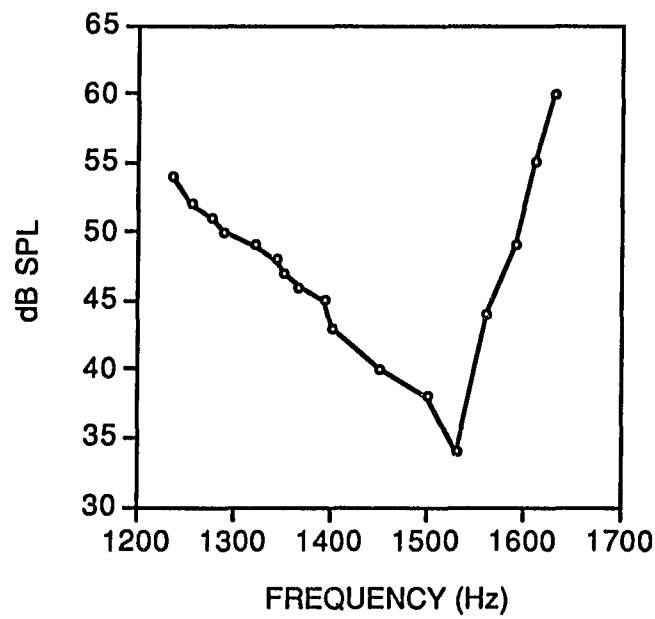

Figure 1. Example of a SOAE suppression tuning curve. Plotted are the frequencies and sound pressure levels (SPLs) of XTs required to suppress a SOAE at 1531 Hz by a criterion (e.g. 6 dB) relative to its SPL in quiet.

Alternatively one can plot the SOAE amplitude reduction as a function of an XT level to determine the patterns of suppression for XTs of various frequencies (Figure 2).

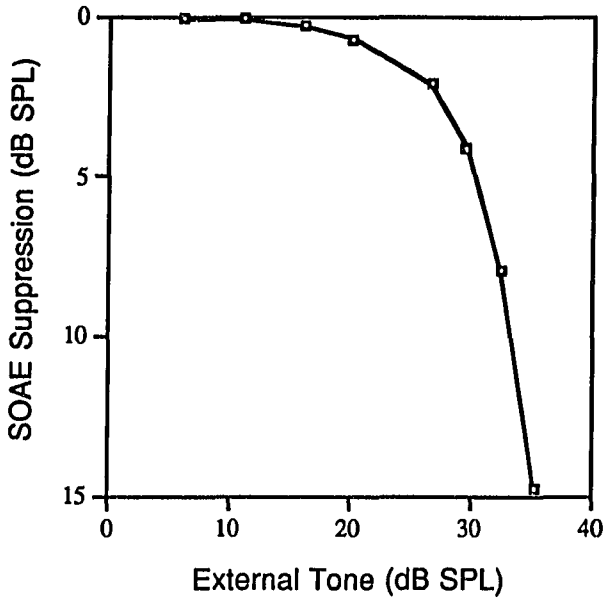

Figure 2. Example of the growth of suppression for a SOAE at 1267 Hz when an XT at 1092 Hz is applied to the ear canal.

Studies that have examined suppression of SOAEs are summarized in Table 1. Zurek (1981) reasoned that SOAE suppression tuning curves should be highly tuned if OAEs originate from a narrow segment of the basilar membrane. Studies that have examined suppression tuning curves have found them to be highly tuned, resembling the shapes of neural and psychophysical tuning curves. The tip of a suppression tuning curve is located slightly higher than the SOAE frequency (Rabinowitz & Widin, 1984; Schloth & Zwicker, 1983; Zizz & Glattke, 1988; Zurek, 1981). Suppression tuning curves exhibit a steep high frequency slope and a more gradual low frequency slope, regardless of suppression criterion (Frick & Matthies, 1988; Schloth & Zwicker,

Table 1. Studies That Have Examined Suppression of Spontaneous Otoacoustic Emissions (SOAEs)

	Frequency	amplitude (dB SPL)	suppression criterion	high slope (dB/octave)	low slope (dB/octave)	growth of suppression by a low frequency	growth of suppression by a high frequency
Zurek (1981)	1400 1910 2103	9 6.5 6	4	steep	gradual	abrupt	gradual
Schloth & Zwicker (1983) Calculated from their figure 4.	2830	12	3 10 6	76.4 88.0 similar to above slopes	61.7 61.8	n/a	n/a
Rabinowitz & Widin (1984)	1893 1380 ≈1530	15 ≈7 ≈13	10 6 6	333 168 201	66 95 111	≈5dB/dB	≈1dB/dB
Zizz & Glattke (1988)	1112 1225 1400 1400 1650	9.2 13.1 12.6 15.5 19	4	154.4 134.4 135.2 95.6 104.5	48.4 56.4 52.4 50.4 55.8	n/a	n/a
Frick & Matthies (1988)	1486 1932 1924	10.9 2.6 6.8	total suppression 3	123 73 79 similar to total slopes	77 34 36	n/a	n/a
Long et al. (1993)	4300	14		n/a	n/a	steep near SOAE (≈4dB/dB)	gradual
Tooley (1993)	4555 2740 1751 1662	-6.15 -2.35 -5.4 2.9	3-5	311 73 111 60.5	85 86 57 81.3	n/a	n/a
Long et al. (1991)	2070 1800 1630 3275	7 7 12 5	3	steep	gradual	steep	gradual

1983). The addition of an XT in suppression studies also has been shown to influence the frequency of the SOAE. Schloth and Zwicker (1983) reported SOAE frequency shifts of ± 3 Hz in their suppression studies. Rabinowitz and Widin (1984) reported frequency "pulling" for suppressors ± 20 Hz of the SOAE. That is, XTs were associated with shifts of the frequency of the SOAE toward the frequency of the XT. Suppressor frequencies further away and higher in frequency were associated with more complex shifts while XTs further away but lower in frequency produced small (<2 Hz) and unsystematic changes. Long et al. (1991) also demonstrated frequency pulling for XTs close in frequency to the SOAE. They suggested frequency pulling is related to synchronization of the SOAE by the XT. Frequency changes in SOAEs have also been observed in the absence of an XT. For example, Zurek found a slow decrease in the frequency of his SOAE (about 10 Hz) after inserting the probe into his ear canal and recording for a period of 45 minutes. Likewise, Frick and Matthies reported fluctuations up to 10 Hz in four subjects over a 1 hour period of time. Even though SOAE frequency has been shown to vary slightly both with and without external tonal stimulation, suppression tuning curves have been shown to be very replicable over time (Tooley, 1993; Zizz & Glattke, 1988).

Studies also have examined the rate of growth of suppression of SOAEs. For XT frequencies lower than the SOAE, suppression occurs rapidly once a critical suppressor tone level is reached. This is also true for suppressors higher in frequency, provided that the suppressor is very close in frequency to the SOAE. High frequency suppressors further from the SOAE exhibit growth curves with more gradual slopes (Long et al., 1993; Rabinowitz & Widin, 1984; Zurek, 1981).

The term suppression can have a number of meanings. An intriguing interpretation of the word was suggested by Zwicker (1986b). Zwicker interpreted suppression to be a "reduction of an enhancement" (p. 163) that is caused by saturating the feedback loop of the CA. Zwicker also offered an interesting view on the generation of DPOAEs. He suggested that suppression plays an important role in the development of DPOAEs.

DPOAEs Resulting From a SOAE and an XT

When DPOAEs are created using a SOAE and an XT, usually the SOAE is treated as f_2 and the XT as f_1. It appears very difficult to generate observable DPOAEs when the SOAE is designated f_1. For example, Glattke and Harris (unpublished data) were not able to generate any $2f_1$-f_2 DPOAEs when the SOAE was lower in frequency (f_1) than the XT (f_2). In contrast, they were able to generate $2f_1$-f_2 DPOAE in five out of five subjects when the SOAE was f_2 and the XT was f_1.

Zurek (1981) reported on the generation of $2f_1$-f_2 and $3f_1$-$2f_2$ when he applied a 1713 Hz tone to his right ear that had an SOAE at 1910 Hz (a ratio of 1.12). Increasing the intensity of the XT resulted in elimination of a detectable SOAE and the combination tones. However, more specific details regarding SPL levels of the DPOAEs and XT were not provided. Frick and Matthies (1988) were able to generate DPOAEs in two of three ears. Using a subject with a SOAE at 1486 (mean amplitude of 10.9 dB SPL) the authors found a very small range of XT frequencies (approximately 50 Hz) that, when applied to the ear canal, resulted in a detectable $2f_1$-f_2 . In this same subject an SOAE at 1932 Hz (mean amplitude 2.6 dB SPL) was associated with a greater range of XT frequencies (approximately 250 Hz) that resulted in a $2f_1$-f_2 DPOAE. Data

from their one subject suggest that SOAE/f_1 ratios between 1.1 and 1.25 are necessary to generate a DPOAE at $2f_1$-f_2. More recently, Long et al. (1993) reported on this phenomenon. Using a small group of subjects, Long et al. studied DPOAEs generated in three subjects all of whom had an SOAE approximately 17 dB above the noise floor. SOAEs were located at 2362 Hz, 4272 Hz, and 1654 Hz and at least one subject had more than one SOAE in the test ear. However, it is unclear from their study which subject had more than one SOAE. Details regarding the characteristics of the additional SOAEs in this ear were not provided. Long et al. found that when the SOAE level was equal to the XT level, a $2f_1$-f_2 DPOAE was detected. In two subjects, this DPOAE initially grew monotonically with increasing levels of the XT. Maximum DPOAE levels were observed when the SOAE was suppressed by the XT. With further suppression, the $2f_1$-f_2 DPOAE level decreased and finally disappeared when the SOAE was no longer observed. The third subject (having a SOAE at 4272 Hz) exhibited an initial nonmonotonic growth function with increasing levels of the XT. Similar to the other two subjects, the maximum amplitude was observed when the SOAE was just suppressed by the XT. The $2f_1$-f_2 DPOAE then decreased in amplitude and finally disappeared with further increases in the level of the XT. Long et al. examined DPOAEs using f_2/f_1 frequency ratios between 1.05 and 1.35 in .02 or smaller steps. The levels of their XTs were not fixed but rather, the authors sampled points near the expected maximal DPOAE. For one of their subjects they found that the f_2/f_1 ratio yielding the largest $2f_1$-f_2 was 1.2. The ratio yielding the largest DPOAE is not reported for the other two subjects. Levels of f_1 that were associated with the largest $2f_1$-f_2 DPOAE (using a frequency ratio around 1.22) varied across subjects but were generally

between 35-45 dB above the level of the SOAE. Long et al. also reported that $3f_1$-$2f_2$ was present in some cases. The maximum amplitude occurred using an f_2/f_1 ratio around 1:1. When DPOAE amplitude was plotted as a function of its own frequency, both DPOAEs showed a maximum about 1/2 octave below the frequency of the SOAE. This pattern is similar to that reported for DPOAE evoked by two XTs (Brown & Gaskill, 1990). That is, DPOAE amplitude (for DPOAE = $2f_1$-f_2 and $3f_1$-$2f_2$) is maximal at a frequency approximately 1/2 octave below the frequency of f_2.

In a pilot study, Glattke and Harris (unpublished data) generated $2f_1$-f_2 using one SOAE and one XT in five subjects. SOAEs ranged from 1232 to 4100 Hz with amplitudes between -5.68 and +3.16 dB SPL. The ratios that were examined ranged from 1.1 to 1.2 in .02 steps. The intensity levels of the XTs varied in 5 dB steps up to 50 dB SPL. Glattke and Harris found complex relationships among DPOAE amplitude, SPL of the XT, f_2/f_1 ratio and SPL of the SOAE. In contrast to Long et al., $2f_1$-f_2 initially appeared when the XT was approximately 20 dB above the level of the SOAE. The amplitude of $2f_1$-f_2 increased with increasing levels of the XT. When the XT reached 30-40 dB above the initial SOAE amplitude, the XT began to suppress the SOAE and the DPOAE amplitude began to decrease and finally disappeared with further increases in XT level. For SOAE/f_1 ratios of 1.1, 1.12 and 1.14, the DPOAE maximum occurred when the XT was 30-35 dB above the initial SOAE amplitude. For ratios of 1.16, 1.18 and 1.2 the DPOAE maximum occurred when the XT was 40 dB above the initial SOAE amplitude. In addition, larger DPOAE amplitudes were observed at the larger frequency ratios.

Summary

Otoacoustic emissions are noninvasive tools used to examine cochlear function. OAEs can be elicited using a range of stimulus levels. High SPLs likely probe processes that include the passive mechanics of the Bekesy traveling wave while low SPLs likely probe the active micromechanical elements of the cochlea, most probably the OHCs. The amplitude characteristics of DPOAEs, when generated using medium- to high-level primaries, have been well characterized. However, previous investigators have found it difficult to generate DPOAEs using lower-level primaries. When one primary is a SOAE and the other an XT, DPOAEs can be created using low- to medium-level primaries. The present investigation describes DPOAEs created through the interaction of a SOAE and an XT. Because low- to medium-level primaries were employed, the DPOAE characteristics reflect primarily active mechanisms within the cochlea.

Noninvasively obtained acoustic data, like those obtained in the present investigation, represent mechanical events within a healthy, living cochlea, a cochlea that is physiologically vulnerable to more invasive measurements. These acoustic data are important because they can be used in the development and refinement of cochlear models.

Research Questions

The purpose of this study was to describe the amplitude characteristics of the $2f_1$-f_2 DPOAE created using primary tones less than or equal to 60 dB SPL. The specific questions addressed were:

1. What are the amplitude characteristics of $2f_1$-f_2 DPOAE when f_2 is a SOAE?
2. What is the relation of SOAE suppression to $2f_1$-f_2 DPOAE generation?

METHOD

Instrumentation

Distortion product otoacoustic emissions (DPOAEs) reported in this study were at the frequency $2f_1$-f_2. Spontaneous otoacoustic emissions (SOAEs) and DPOAEs were recorded using the instrumentation diagrammed in Figure 3. The instrumentation incorporates a probe assembly that includes an Etymotic Research (ER-10) microphone and two miniature earphones (ER-2). Only one earphone was used in the present study. The probe assembly was coupled to the ear canal by a disposable foam tip. It was calibrated in response to a 1 kHz, 94 dB SPL tone generated by a B & K 4230 Calibrator. At the beginning of each test session, a 1.5 kHz tone at a fixed attenuator setting (previously measured in a 2 cc coupler as 13 $\pm$ 1dB SPL) was presented to one earphone of the probe assembly. This signal was measured in the ear canal of each subject. If necessary the probe was repositioned in the ear canal to attempt to reach a 13 dB SPL output. This procedure helped to minimize variability due to placement of the probe and intersubject differences related to the physical dimensions of each ear canal.

The microphone transduced the acoustic signals in the ear canal to electric signals which were then amplified (100 times) and high-pass filtered (cutoff of 400 Hz, 30 dB per octave) by a laboratory constructed unit. This signal was led to a signal analyzer (Stanford Research System-SR760) for averaging and Fast Fourier Transform (FFT) analysis. Spectrum averaging was done using 0.75, 1.56 or 3.125 kHz windows centered approximately at f_1 (the lower-frequency primary). This resulted in frequency resolution of 1.95, 3.9, or 8 Hz, respectively. The smallest window that allowed the DPOAE, external tone (XT),

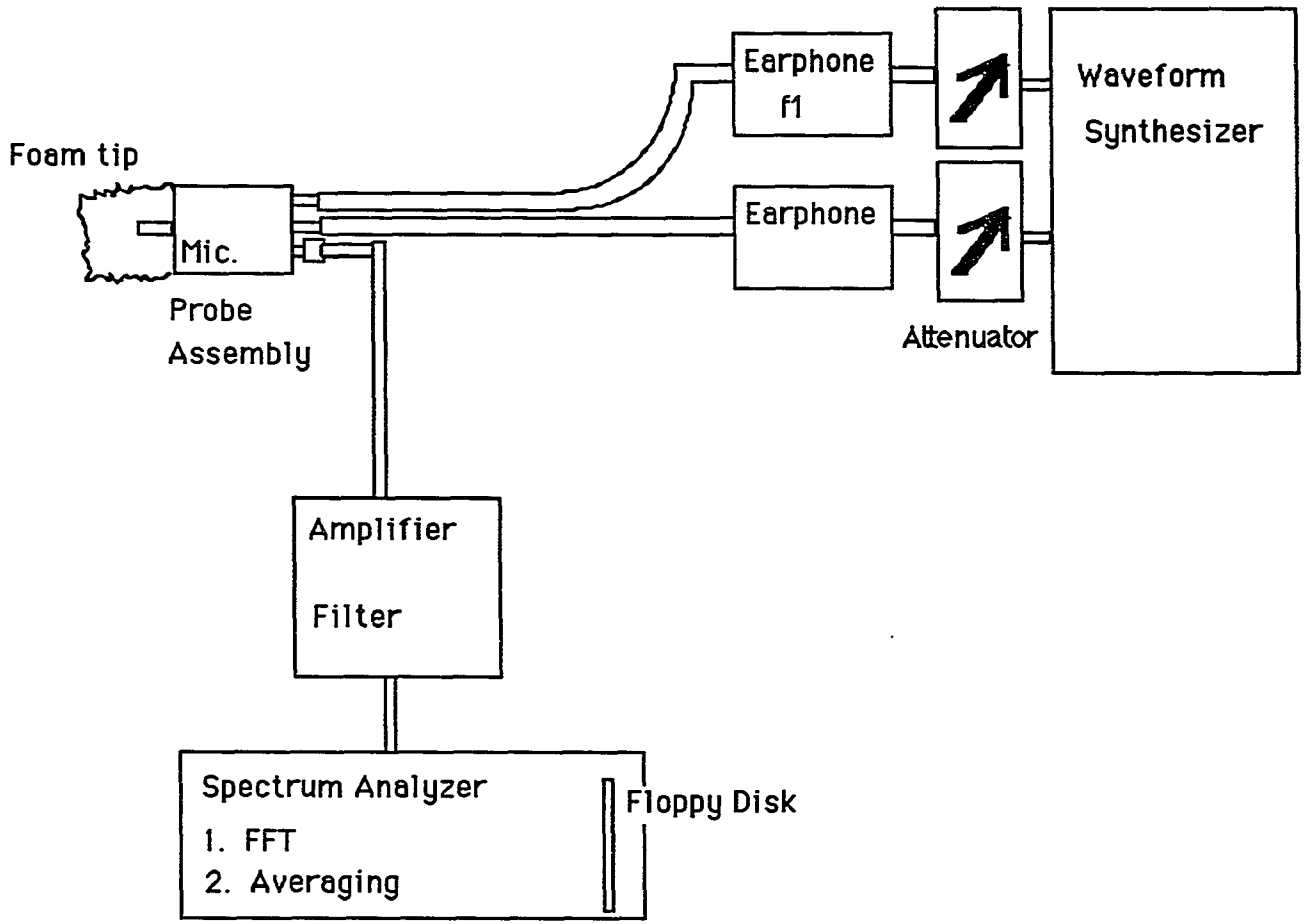

Figure 3. Instrumentation for XT generation and response detection and measurement. Only one earphone was used in the present study to generate the XTs (f1).

and SOAE to be visualized was used. The amplitude of each frequency bin was determined using a Hanning window function. Six hundred samples were averaged to determine the amplitudes of the signals and noise. All data were stored on floppy disk for later analysis.

Subject Selection

Young women between the ages of 18 and 40, who reported a history of normal hearing, were recruited from the University of Arizona. They read and signed a human subjects consent form (Appendix A) and were then screened for the presence of SOAEs. An SOAE was defined as a narrow band signal having a peak amplitude at least 3 dB SPL above the noise floor. An otoscopic exam was performed prior to testing to insure unoccluded ear canals and a visible tympanic membrane.

SOAE Screening

After placing the microphone in the ear canal, the signal from the microphone was analyzed without presenting an intentional acoustic stimulus. The SOAE search extended from 500 to 5000 Hz in 750 Hz windows with a resolution of 1.95 Hz. Windows were centered initially at 1000 Hz and then at increments of 500 Hz. Approximately 600 spectra were averaged to search for SOAEs.

Other Subject Selection Criteria

If SOAEs were observed during the screening session they were required to meet the following criteria for the subject to participate in this study.

1) The target SOAE frequency was between 1.1 and 5 kHz.
   The purpose of the lower frequency limit was to avoid recording DPOAEs in high levels of noise. The upper frequency limit was

employed because of speculation that OHCs in the basal and apical regions of the cochlea function differently (Dallos, 1988).

2) Only one SOAE could be present in an ear. Or, if there was more than one SOAE, the extraneous SOAE had to be separated from the target SOAE or anticipated DPOAE by at least one-half octave. This criterion was employed to avoid possible interactions between the extraneous SOAE and the DPOAE, XT and target SOAE (Burns, Arehart & Campbell, 1984; Cianfrone, Mattia, Altissimi & Turchetta, 1990; McFadden & Pasanen, 1994; Wier, Pasanen & McFadden, 1988).
3) The SOAE amplitude was at least 10 dB SPL above the noise floor. The noise floor was measured at a frequency approximately 40 Hz lower than the SOAE. This criterion was employed because pilot studies revealed that it was difficult to obtain DPOAEs when a SOAE was less than 10 dB above the noise floor.
4) The SOAE was stable in frequency and intensity for a period of 10 minutes. A stable SOAE was defined as one that did not shift in frequency by more than $\pm$ 4 Hz and $\pm$ 2 dB SPL during the 10 minute observation period.

Additionally, to be included in the study, subjects had to demonstrate

1) a negative history for significant medical conditions as assessed by a questionnaire (Appendix B). Subjects were excluded from the study if any medical condition was checked in Part I or a "yes" was checked in Part II of Appendix B.

2) normal hearing sensitivity (15 dB HL or better) for the octave frequencies 0.25 - 8 kHz (ANSI, 1969);
3) normal middle ear function demonstrated by a symmetric tympanogram with compensated equivalent volume of 0.3 to 3 cc and contralateral acoustic reflexes present at 0.5, 1, and 2 kHz.

Stimuli

Tones were individually synthesized using a 12-bit programmable waveform synthesizer (Quatech, WSB-10C). The levels of the tones were adjusted using an attenuator (Hewlett Packard 350 D).

General Procedures

Subjects participated in two recording sessions. Session Two was a replication of session One. Subjects were asked to refrain from activities that could be damaging to the cochlea for a period that began 72 hours prior to the first session and continued through the completion of the second session (see Appendix B). For both sessions, subjects were comfortably seated in a sound-treated booth. Each session lasted approximately one hour. Prior to session Two, subjects completed a questionnaire about their recent experiences, that could cause damage to the cochlea (see Appendix C). If any of the conditions were checked in this questionnaire the subject was disqualified from the study.

Session One

The purpose of this session was to create DPOAEs using low- to medium-level primaries in ears with SOAEs, using the SOAE as f_2. An XT (f_1) at various frequencies and SPLs was applied to the ear canals.

Procedures

A pure tone (f_1) at one of 11 randomly sequenced SOAE/f_1 ratios (ranging from 1.1 to 1.20, in .01 steps) was applied to the ear canal. A new random sequence was determined for each subject. If DPOAEs were observed at the ratio of 1.1, 1.11, 1.19 or 1.20, the ratio was decreased or increased, respectively, in 0.01 steps. Ratios did not extend lower than 1.08 or higher than 1.22. Initial level of the XT was set to approximately match the original level of the SOAE. The level of the XT was then incremented in 5 dB steps until the SOAE and DPOAE disappeared into the noise floor. Prior to testing with each new frequency ratio, the SOAE was recorded in the absence of external acoustic stimulation.

Session Two

This session was conducted within a minimum of four hours and a maximum of 28 hours of session One and was a replication of session One. The instrumentation, method for generating stimuli, procedures, and sequence of SOAE/f_1 ratios were the same as in session One.

Data analysis

Measures obtained from data for both sessions consisted of the following:

1. Initial SOAE level, in the absence of acoustic stimulation, prior to presentation of the first XT.
2. SOAE level, in the absence of acoustic stimulation, prior to studying each frequency ratio.
3. SPL of the noise floor at a point approximately 40 Hz lower in frequency than the SOAE.

4. SPL of the SOAE during stimulation.
5. SPL of each DPOAE.
6. SPL of the XT.
7. SPL of the noise floor at a point approximately 40 Hz lower in frequency than each DPOAE.

Variability between subjects was expected regarding the individual noise floors and observed levels of SOAEs. Therefore, for group data analysis the observed DPOAE and SOAE values were recalculated relative to their respective noise floors to yield the following measures:

8. Relative SPL of the SOAE -- defined as the observed SOAE value minus the noise floor of the SOAE.
9. Relative XT level--defined as the observed XT level minus the initial SOAE level.
10. Relative SPL of the DPOAE -- defined as the observed DPOAE value minus the noise floor of the DPOAE.

Data from session Two were used to assess the reliability of session One. The data from session One were analyzed in detail. If any data were missing from session One, the results from session Two were described in detail.

RESULTS

SOAE Search

One hundred and twenty-two women who reported normal hearing sensitivity in both ears were screened bilaterally for the presence of spontaneous otoacoustic emissions (SOAEs). Table 2 displays the outcome of this screening. Fifty-four percent of the women and 42% of the ears screened exhibited SOAEs that were at least 3 dB above the noise floor. Considering only the 66 women who had SOAEs, 80% of the 132 tested ears revealed SOAEs. Fifty-six percent of the women with SOAEs had them in both ears while 33% had right ear unilateral SOAEs and 11% had left ear unilateral SOAEs. Only 29% of the 122 women screened had a single SOAE in either ear. If an ear revealed SOAEs, the median number of emissions per ear was two with a maximum of eleven. Figure 4 displays scatterplots of the emission characteristics detected in subjects' right and left ears. SOAEs were more commonly observed in the right than in the left ear (169 and 118 SOAEs, respectively). The greatest concentration of SOAEs was found between 1.2 and 1.79 kHz, as illustrated by the histogram in Figure 5. Figure 6 displays the amplitudes of the SOAEs. The majority of SOAEs had amplitudes between -12.9 and 3.8 dB SPL.

Subject Selection and Data Retention

Nineteen of the women who were screened met the criteria for subject selection. However, seven women did not participate in the study for the following reasons: three of the women moved from the area before testing began; three women had very few distortion products as a result of the interaction of their SOAE and the XTs; one woman's SOAE was markedly

Table 2. Prevalence and Characteristics of SOAEs Determined From the Screening Session. A Total of 122 Women Were Screened.

	Number of Women	% of Women Screened	% of Women With SOAEs
SOAE(s) present	66	54	-
Left Ear SOAEs	44	36	67
Right Ear SOAEs	59	48	89
Bilateral SOAEs	37	30	56
Right ear, unilateral SOAEs	22	18	33
Left ear, unilateral SOAEs	7	5	11
A single SOAE in either ear	35	29	53

changed from the screening session to the first test session. The SOAE of interest decreased in amplitude while four new emissions appeared. Data for the remaining 12 women were included if the reliability of the results of session One and Two was .75 or greater and if the DPOAE mean amplitudes of session One and Two were not significantly different from each other.

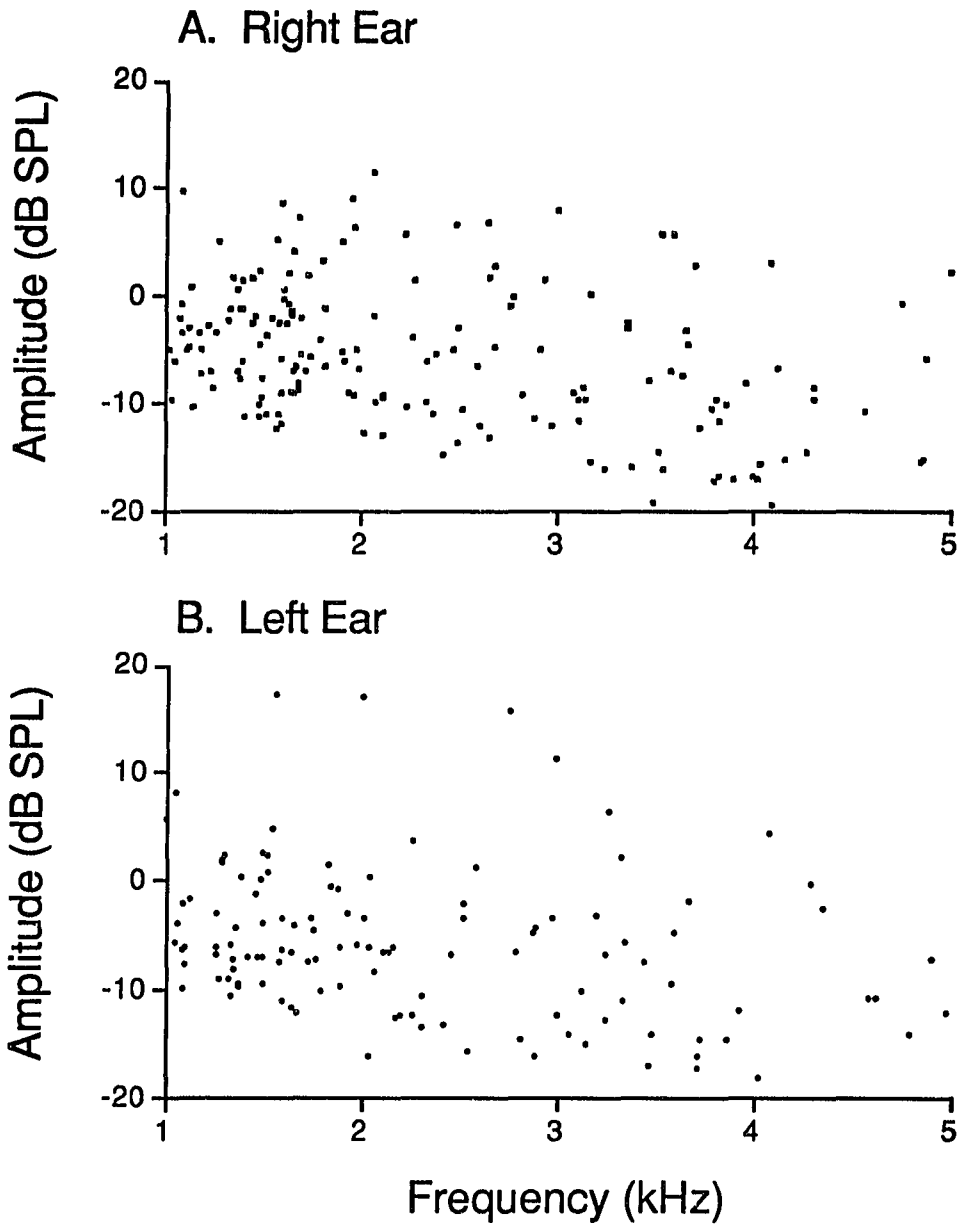

Figure 4. SOAE amplitude as a function of frequency recorded from 66 women. Figure 2A: 169 SOAEs were recorded from right ears. Figure 2B: 118 SOAEs were recorded from left ears.

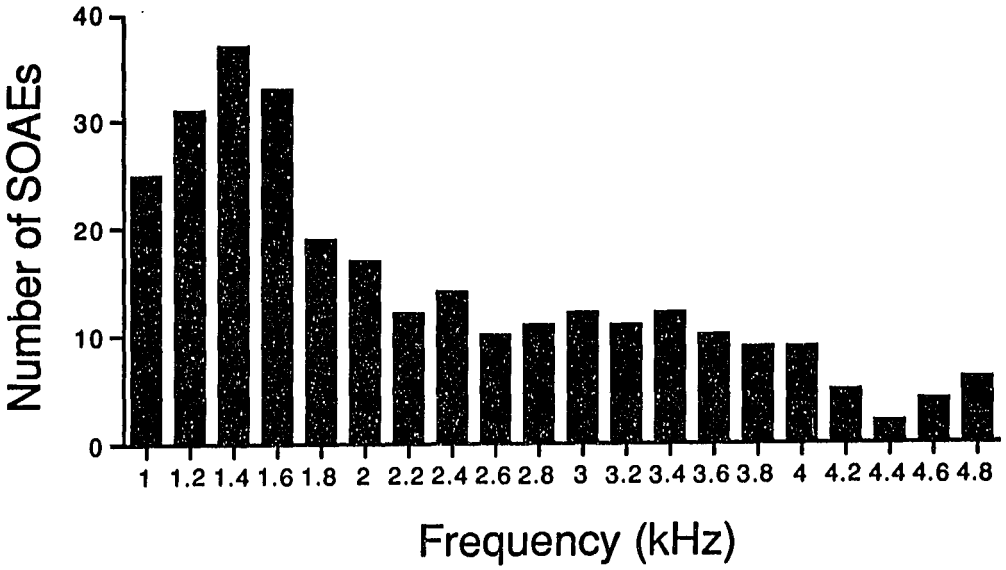

Figure 5. Frequency distribution of SOAEs. Bin widths are 0.199 kHz re: the beginning frequency indicated.

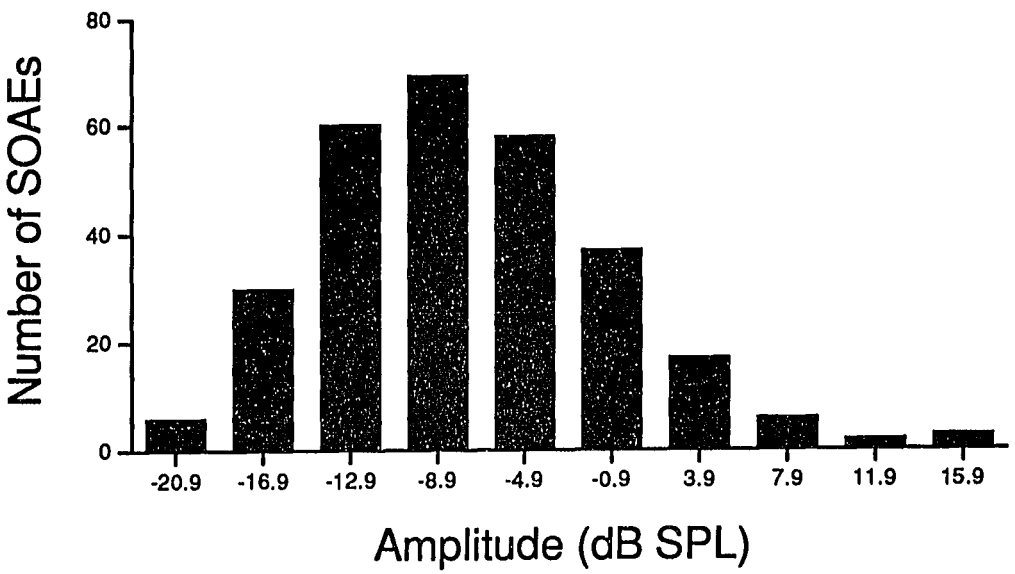

Figure 6. Amplitude distribution of SOAEs. Bin widths are 4 dB re: the beginning amplitude indicated.

Reliability Between Sessions One and Two

A Pearson Product Moment Correlation (r) was used to assess the test-retest reliability of DPOAE amplitude as a function of the relative XT level. An overall correlation was calculated for each subject by converting r at each of the 15 ratios to a Fischer Z value. The Fischer Z values were then averaged and converted back to an overall r. Data for two women exhibited overall correlations less than .75 and were therefore excluded. Overall correlations for the 10 remaining subjects ranged from .77 to .97 and are displayed in Table 3.

Table 3. Overall Correlations For DPOAE Amplitudes as a Function of XT Level (Relative to Initial SOAE Amplitude) Between Sessions One and Two.

Subject	Overall Correlation
KM	.77
MH	.91
AR	.87
RA	.97
JS	.84
IR	.93
MK	.89
JV	.78
BR	.93
LH	.88
KS	.92

Mean DPOAE Amplitudes For Sessions One and Two

Paired t-tests were performed for each subject and at each ratio to determine if the mean DPOAE amplitude was the same for both sessions. Because of the large number of t-tests performed, a significance level of .003 was used (alpha = .05/15 contrasts). Results indicated that none of the t-tests were significant. No other women were excluded and therefore, 10 subjects participated in the study.

SOAE, Noise Floor, and Recording Characteristics

Table 4 displays the results of the calibration for each subject and the recording window used to obtain the data for sessions One and Two. The amplitude of the 1.5 kHz calibration tone, presented at a fixed voltage to the earphone and recorded in subjects' ear canals, ranged from 9.9 to 15.2 dB SPL. The differences in calibration values between subjects is likely due to the differences in the sizes of the subjects' ear canals. For any particular subject the difference in calibration from session One to session Two was small. The maximum variation was 1.7 dB SPL for subject JS.

The SOAEs, in the 10 subjects, used for f_2 ranged from 1266 to 4906 Hz, and from -7.2 to 7.5 dB SPL. Eight of the 10 SOAEs were recorded from the right ear. The SOAE characteristics as well as the average SOAE noise floor (a point taken for all recordings approximately 40 Hz below the frequency of the SOAE) for each subject and for both sessions are displayed in Table 5. The session from which data were analyzed is indicated in this table by an asterisk. The minimum and maximum SOAE initial frequency shift across sessions was 1 and 16 Hz for subjects KS and MK, respectively. SOAE initial

amplitude change across sessions ranged from 0 to 3.5 dB SPL for subjects BR

Table 4. Recording Window Used and Sound Pressure Level (SPL) Recorded in Subjects' Ear Canals in Response to a 1.5 kHz Calibration Tone Presented at a Fixed Voltage to the Earphone.

Subject	Window (kHz)	Session 1 (dB SPL)	Session 2 (dB SPL)
KM	0.78	13.7	13.2
AR	1.50	13.8	13.8
RA	0.78	12.8	12.6
JS	1.50	9.9	11.6
IR	0.78	14.2	14.1
JV	1.50	14.6	14.7
MK	3.125	14.9	14.1
BR	0.78	13.8	14.1
LH	3.125	15.0	14.2
KS	0.78	15.2	14.6

and AR, respectively. The average SOAE noise floors ranged from -13.8 to -18.0 dB SPL. For any particular subject there was little change in the average noise floor across sessions. The maximum change was 0.6 dB SPL for subject BR.

When no XT was presented, SOAE amplitude and frequency characteristics varied within a test session. Changes relative to the initial SOAE characteristics are displayed in Table 6. The largest frequency change within a session was 19 Hz observed for the SOAE of subject JS. The largest amplitude

Table 5. Initial SOAE Characteristics

Subject	Ear	INITIAL SOAE (Hz)		INITIAL SOAE (dB SPL)		Mean SOAE NOISE FLOOR (dB SPL)	
		Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2
KM	R	*1266	1270	5.5	8.0	-13.8	-13.9
AR	R	*2637	2645	6.1	9.6	-15.1	-15.3
RA	L	*1486	1490	1.7	1.3	-15.1	-14.8
JS	R	*4074	4063	4.3	3.1	-17.7	-18.0
IR	R	*1529	1533	7.5	7.8	-15.0	-15.1
JV	R	*2215	2211	4.6	5.7	-14.2	-14.2
BR	R	2057	*2064	4.2	4.2	-16.1	-16.7
MK	L	*4906	4922	-7.2	-6.9	-14.3	-14.4
LH	R	*4297	4312	-3.2	-3.6	-15.3	-15.0
KS	R	*1604	1605	5.9	4.0	-15.1	-14.7

Table 6. Maximum SOAE Frequency and Amplitude Changes Without XT Stimulation (Prior To Each New Ratio) Relative to the Initial SOAE Frequency and Amplitude.

Subject	Session	Initial SOAE Frequency	Initial SOAE Amplitude	Frequency change (Hz)	Amplitude change (dB SPL)
KM	1	1266	5.5	-2	4.4
	2	1270	8.0	-2	-1.5, 1.3
AR	1	2637	6.1	-4	6.3
	2	2645	9.6	-4	-0.2, 4.2
RA	1	1486	1.7	0	-0.8, 0.6
	2	1490	1.3	-4	-1, 0.1
JS	1	4074	4.3	8	-0.2, 4.5
	2	4063	3.1	19	3.5
IR	1	1529	7.5	-2	-0.8, 0.7
	2	1533	7.8	-6	-0.9, 0.7
JV	1	2215	4.6	-12	1.4
	2	2211	5.7	-4	2.5
MK	1	4906	-7.2	0	-0.1, 1.4
	2	4922	-6.9	0	-0.8, 0.6
BR	1	2057	4.2	-2	-2, 0.5
	2	2064	4.2	-9	2.9
LH	1	4297	-3.2	8	-1.0, 1.3
	2	4312	-3.6	8	3.7
KS	1	1604	5.9	-2, 1	-1.5, 0.6
	2	1605	4.0	0	-0.1, 1.5

change within a test session was 6.3 dB SPL observed for the SOAE of subject AR. A regression analysis was done to determine if there was a significant correlation between the frequency and amplitude of the SOAEs without XT stimulation. The results of this analysis are displayed in Table 7. A significant correlation was obtained for only 1 of the 10 subjects (r=0.60, $F_{(1, 14)}$=7.15, p=.02). However, with a significance level of .05 it is expected that 1 out of 20 correlations would be significant by chance.

Table 7. t-test Results of the Comparison Between SOAE Frequency and Amplitude Without XT Stimulation.

Subject	r	df	F	p
KM	.39	1,14	2.36	.15
AR	- .31	1,14	1.42	.26
RA	- .09	1,14	0.11	.75
JS	- .32	1,14	1.45	.25
IR	.60	1,14	7.15	.02
MK	.32	1,14	1.46	.25
BR	- .50	1,14	4.22	.06
JV	- .29	1,14	1.18	.30
KS	- .02	1,14	0.005	.95
LH	- .12	1,14	0.18	.67

DPOAEs Created Using a SOAE and XTs

With an SOAE as f_2 and XT as f_1, and SOAE/f_1 ratios between 1.08 and 1.22, DPOAEs were created for all 10 subjects. The optimal ratio (ratio at which the largest DPOAE was observed) varied across subjects. Table 8 displays the optimal SOAE/f_1 ratios for all subjects. These ranged from 1.12 to 1.22 with a mean of 1.16 (SD = .03).

Table 8. Optimal Ratio (Ratio at Which the Largest DPOAE Was Observed) and DPOAE Growth and Decline Rates at the Optimal Ratio.

Subject	Optimal Ratio	Slope of DPOAE Growth Function (dB/dB)	Rate of DPOAE Decline (dB/dB)
KM	1.12	0.75	-3.39
AR	1.14	0.98	-1.25
RA	1.18	0.93	-2.76
JS	1.15	0.86	-2.07
MK	1.18	0.56	-1.58
IR	1.22	0.54	-3.34
JV	1.13	0.90	-1.29
BR	1.18	0.84	-1.36
LH	1.15	0.50	-1.09
KS	1.17	0.65	-3.51
Group Mean	1.16 (s.d.=.03)	0.75 (s.d.=.18)	-2.16 (s.d.=.94)

Complex relations were observed between the XT levels, DPOAE amplitudes and SOAE characteristics. Figure 7 displays these relations for one representative subject and at one representative SOAE/f_1 ratio. The upper panel of this figure depicts the SOAE frequency as a function of the relative XT amplitude (XT amplitude relative to the initial SOAE amplitude). The lower panel depicts the DPOAE and SOAE amplitudes (relative to their respective noise floors) as a function of the relative XT amplitude. Although the general characteristics of the amplitude functions plotted in the lower panel of this figure were similar across subjects and ratios, the relative and absolute SPLs associated with the functions were variable. Furthermore, the variability was not simply related to the initial frequency or amplitude of the SOAE.

XT Levels and DPOAE Amplitudes

The relation of XT levels to DPOAE amplitudes was nonmonotonic. DPOAEs grew to a peak with increasing levels of the XT and then declined with further increases in XT level (lower panel of Figure 7). The slope of the growth function and decline rate, at the optimal ratio, were calculated for each subject and are shown in Table 8. These were calculated using a linear least squares fit of four to seven and two to three data points, respectively. The slopes of the growth function ranged from 0.5 to 0.98 dB/dB with a mean of 0.75 dB/dB (SD = .18). The rate of DPOAE decline was much steeper than the growth rate and ranged from -1.09 to -3.51 dB/dB with a mean of -2.16 dB/dB (SD = .94).

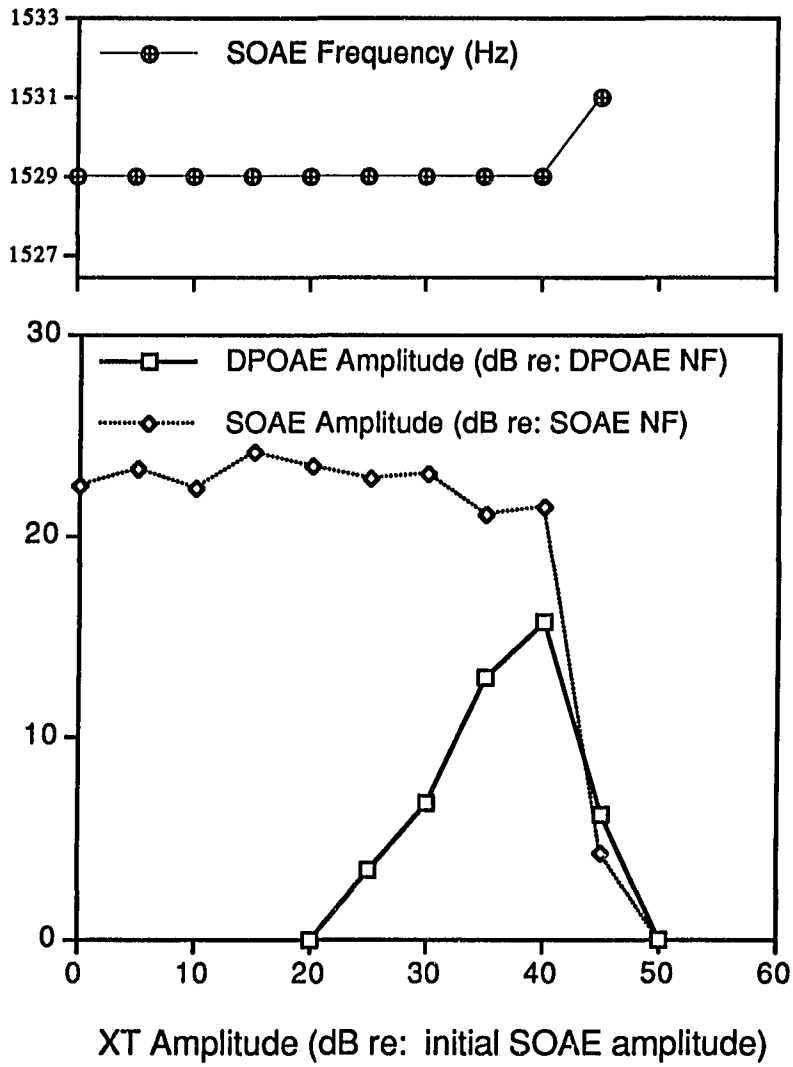

Figure 7. Data from one representative subject at one representative ratio illustrating the most common trends. Upper panel reveals SOAE frequency as a function of XT amplitude. Lower panel reveals SOAE and DPOAE amplitudes, relative to their respective noise floors (NFs), as a function of XT amplitude.

The variability in XT levels associated with the creation of DPOAEs at the various SOAE/f_1 ratios is displayed in Figures 8 and 9. Figure 8 illustrates the average XT SPLs (upper panel) and XT levels relative to the SOAE initial amplitude (lower panel), along with their standard deviations, required for the DPOAEs to reach a threshold of 3 dB SPL above the noise floor. DPOAEs created by the interaction of SOAEs and XTs reached threshold values when the average XT level was 23 to 33 dB SPL or 20 to 30 dB above the initial level of the SOAE. These XT levels varied as a function of SOAE/f_1 ratio. In general, as the ratio increased more intense XTs were needed before DPOAEs were observed. Figure 9 displays the average XT SPLs (upper panel) and XT levels relative to the SOAE initial amplitude (lower panel), along with their standard deviations, associated with the maximum DPOAEs. The maximum DPOAEs were obtained when the average XT levels were 30 to 43 dB SPL or 27 to 40 dB above the initial level of the SOAE. As the ratio increased, more intense XTs were needed before the maximum DPOAEs were observed.

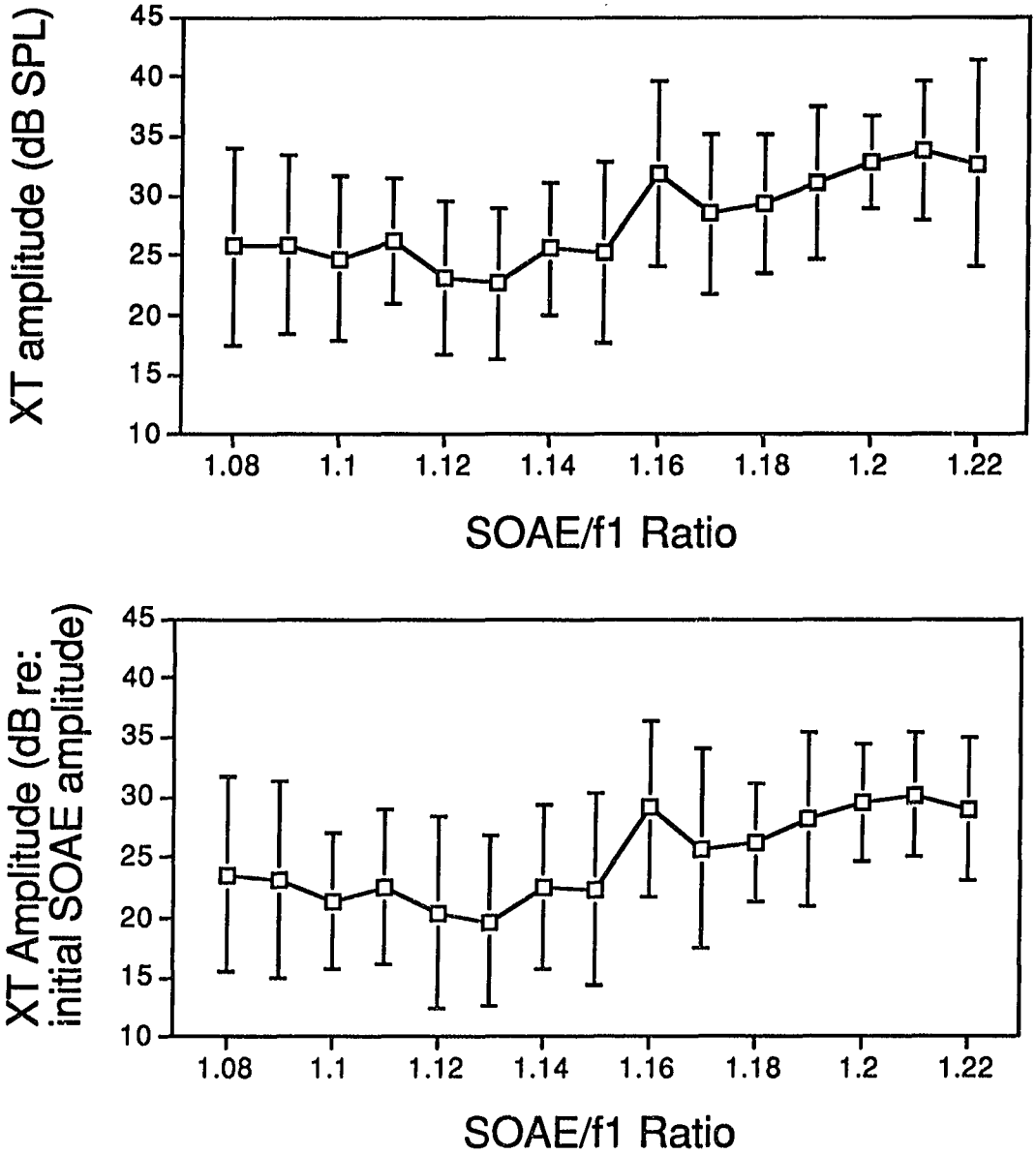

Figure 8. Mean XT amplitudes and standard deviations associated with DPOAE thresholds. XT amplitudes are depicted in dB SPL (upper panel) and relative to the initial SOAE amplitude (lower panel)

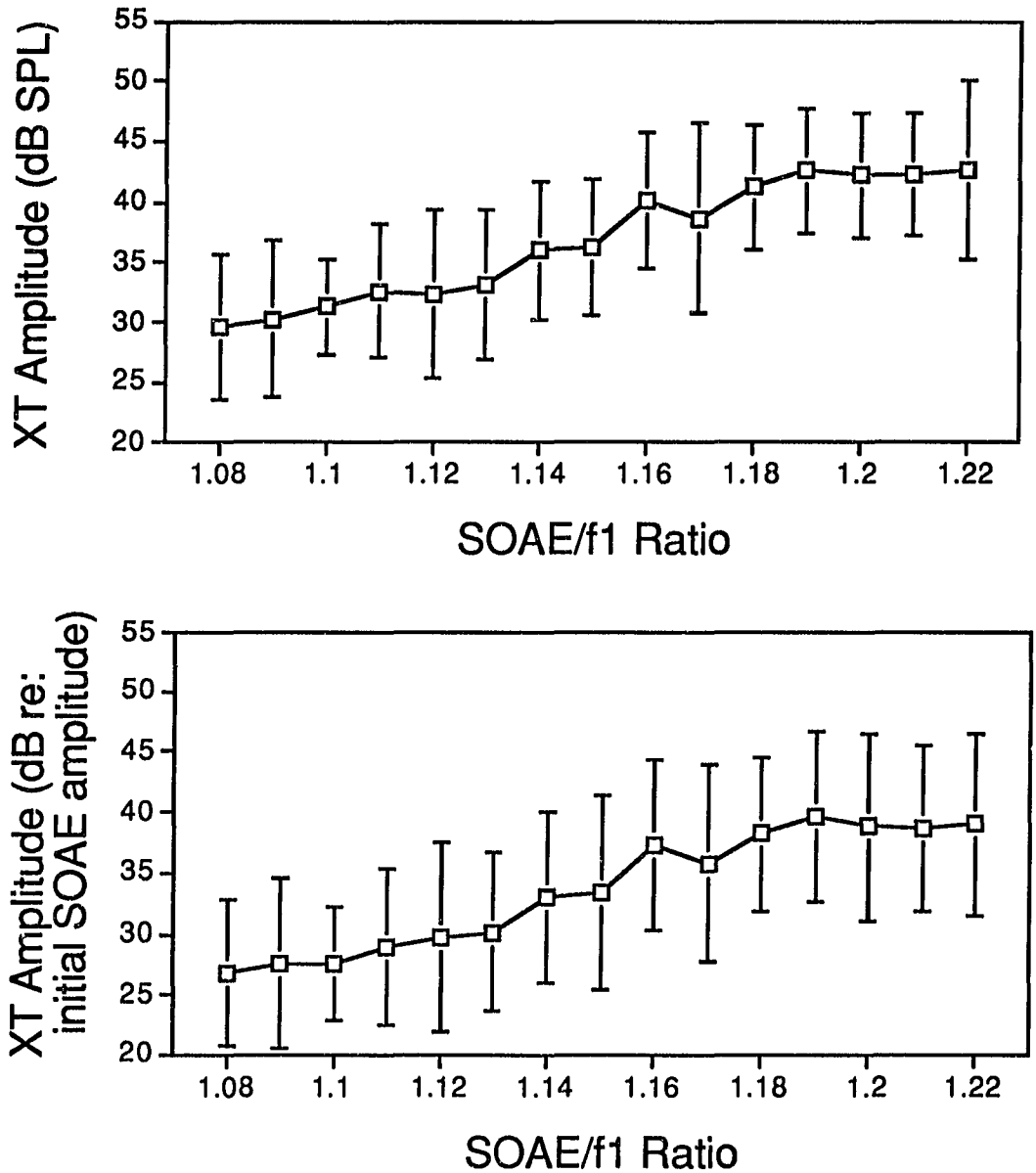

Figure 9. Mean XT amplitudes and standard deviations associated with the maximum DPOAEs. XT amplitudes are depicted in dB SPL (upper panel) and relative to the initial SOAE amplitude (lower panel). The latter is also known as the optimal level difference.

XTs and SOAE Characteristics

The relation of XTs to SOAE amplitudes was characterized by two distinct phases. The lowest levels of the XTs had very little impact on the amplitude of the SOAE. However, once a critical XT level was reached, the SOAE rapidly disappeared into the noise floor (refer back to Figure 7, lower panel). The rate of this rapid suppression was approximately 1 to 2 dB/dB. The means and standard deviations of the XT levels required for total suppression (the point at which the SOAE disappeared into the noise floor) are plotted in Figure 10. As the ratio increased, the XT levels required for total suppression also increased.

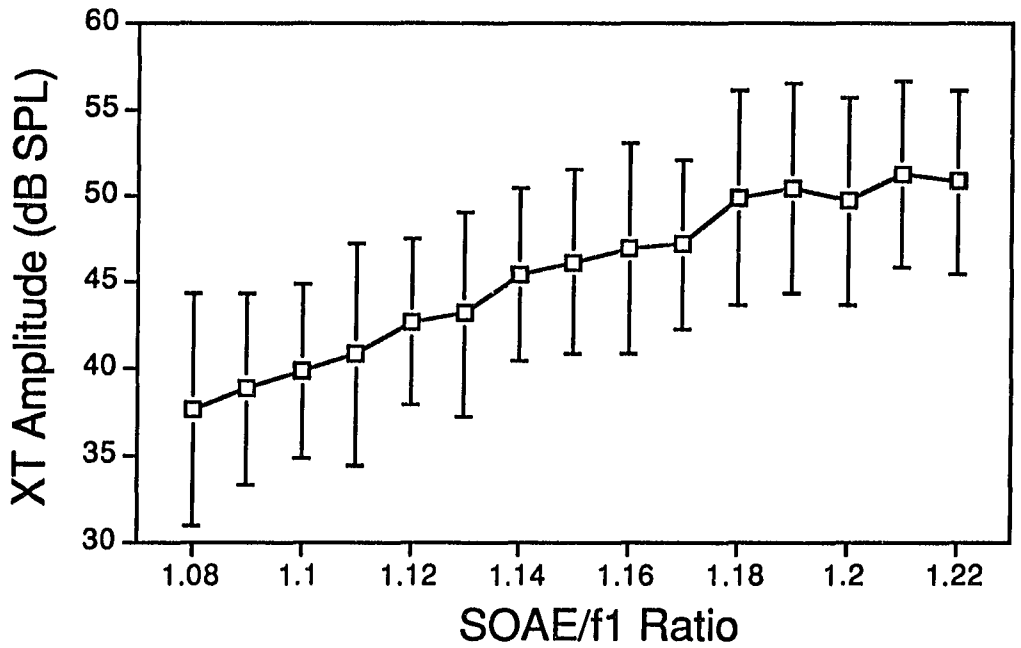

Figure 10. Mean XT amplitudes and standard deviations associated with total suppression of the SOAEs.

A SOAE suppression tuning curve is a plot of the XT levels and frequency combinations required to reduce the amplitude of an SOAE by a criterion amount (refer back to Figure 1). For this study only frequencies lower than the SOAE were applied to the ear, therefore only that portion of the tuning curve (frequencies 1.08 to 1.22 of the SOAE frequency) was calculated for each subject. The slope for total suppression was calculated using a logarithmic fit of the 15 data points. The slopes of these functions ranged from 61.5 to 104.7 dB/octave and are depicted in Table 9. These values are very similar to low frequency slopes reported by others (Rabinowitz & Widin, 1984; Tooley, 1993).

Table 9. Equation and Slope of the Low-frequency Tail of the SOAE Suppression Tuning Curve for Total Suppression for Each Subject. Slopes were Calculated After the Data Were Fit With a Logarithmic Function y=m(log x)+b, Where m is the Slope and b is the y-Intercept.

Subject	Slope equation	Slope (dB/octave)
KM	y= -298.59 Log(x) + 947.17	89.9
AR	y= -266.79 Log(x) + 941.65	80.3
RA	y= -204.35 Log(x) + 676.29	61.5
JS	y= -312.24 Log(x) + 1159.28	94.0
IR	y= -233.14 Log(x) + 781.06	70.2
JV	y= -347.76 Log(x) + 1187.97	104.7
MK	y= -318.58 Log(x) + 1203.43	95.9
BR	y= -214.88 Log(x) + 739.37	64.7
LH	y= -273.64 Log(x) + 1017.64	82.4
KS	y= -249.50 Log(x) + 835.70	75.1

The relation of XT levels to SOAE frequency was complex. The SOAE often shifted its frequency in the presence of XTs. The most common pattern was an upward shift in frequency just prior to total suppression of the SOAE (Figure 7, upper panel). For three of the subjects (IR, BR, AR) this shift occurred for at least 10 of the 15 ratios. For subject IR, the only time the frequency of the SOAE shifted to a higher frequency was just prior to its disappearance into the noise floor.

SOAE Characteristics and DPOAE Amplitude

The relation of SOAE amplitudes to DPOAE amplitudes was nonmonotonic. As the SOAE was reduced in amplitude, the DPOAE amplitude reached a maximum. With further reduction of the SOAE, the DPOAE disappeared into the noise floor.

The average amount of SOAE reduction concurrent with the appearance of the first DPOAE ranged from 0 to 2.8 dB SPL for the various subjects (collapsed across ratio). The average amount of SOAE reduction collapsed across ratios and subjects was 0.8 dB SPL. The SOAE was further reduced when the maximum DPOAE was observed. The amount of reduction ranged from 0.67 to 6.0 dB SPL for the various subjects with an overall average of 2.8 dB SPL.

Paired t-tests (one-tailed) were performed to compare the SOAE amplitude, without XT stimulation, to the SOAE amplitude observed at the DPOAE threshold and at the maximum DPOAE (Table 10). Using a significance level of .05, it is expected that 1 of the 20 t-tests performed would be significant by chance. The results indicated that in five subjects, the SOAE was significantly reduced when the first DPOAE appeared (DPOAE threshold).

In all subjects, the SOAE amplitude associated with the maximum DPOAE was significantly smaller than the SOAE amplitude recorded without XT stimulation.

Table 10. t-test Results of the Comparison of Initial SOAE Amplitude and SOAE Amplitude Observed at DPOAE Threshold and Maximum.

	DPOAE Threshold			DPOAE Maximum		
Subject	t	p	df	t	p	df
KS	-.09	.460	14	2.90	.005	14
LH	1.55	.080	9	5.82	< .001	9
JV	2.48	.020	10	4.78	< .001	10
BR	3.23	.003	14	4.35	< .001	14
IR	.70	.250	14	3.37	.002	14
MK	3.05	.004	14	3.15	.004	14
JS	.60	.280	14	3.06	.004	14
RA	3.40	.002	14	6.17	< .001	14
AR	1.55	.070	12	5.06	< .001	12
KM	5.12	< .001	13	9.53	< .001	13

Total SOAE suppression almost always resulted in the disappearance of the DPOAE into the noise floor. However, in one subject (MK), the DPOAE was occasionally apparent even when the SOAE disappeared into the noise floor (see ahead to ratios 1.14 and 1.19 of Figure 20A & B, respectively).

Group Data

The mean relative DPOAE and SOAE amplitudes as a function of relative XT level, collapsed across all subjects, were calculated (Figure 11). There was much individual variability in terms of the optimal ratios (ratios that resulted in the maximum amplitude DPOAEs) as well as the XT levels needed to suppress SOAEs and generate DPOAEs. Thus, averaging the data had the effect of decreasing the relative level of the DPOAE and smoothing out the data plotted for any particular stimulus level. The individual data were characterized by isolated data points that included higher amplitude DPOAEs and a fine structure consisting of peaks and troughs.

Individual Data

Data for the individual subjects is displayed in Table 11. Individual ratio graphs, that depict the DPOAE and SOAE relative amplitudes as a function of relative XT level, and 3-dimensional (3-D) graphs of the relative DPOAE amplitude as a function of DPOAE frequency and relative XT level are also provided. Figure numbers associated with each subject can be found in Table 11. Six of the 10 subjects exhibited DPOAEs at all frequency ratios. The frequency distance between the maximum DPOAE and the SOAE ranged from 0.38 to 0.65 octave. The maximum DPOAE amplitudes ranged from -2.8 to 5.9 dB SPL while the relative XT levels associated with these maximums ranged from 25 to 50 dB. During the recording session or at the beginning of the test session, new SOAEs often appeared that were not apparent during the screening session. Sometimes these new SOAEs grew in amplitude from the beginning to the end of the test session. For example, subject AR's new SOAE, at a frequency of 2906 Hz, had an initial amplitude of -14 dB SPL and grew to -4

dB SPL by the end of the test session. Any new SOAE having a frequency between the SOAE and DPOAE frequencies is depicted on the 3-D graphs by an arrow. An asterisk on these graphs indicates the frequency of the largest DPOAE observed for that particular subject.

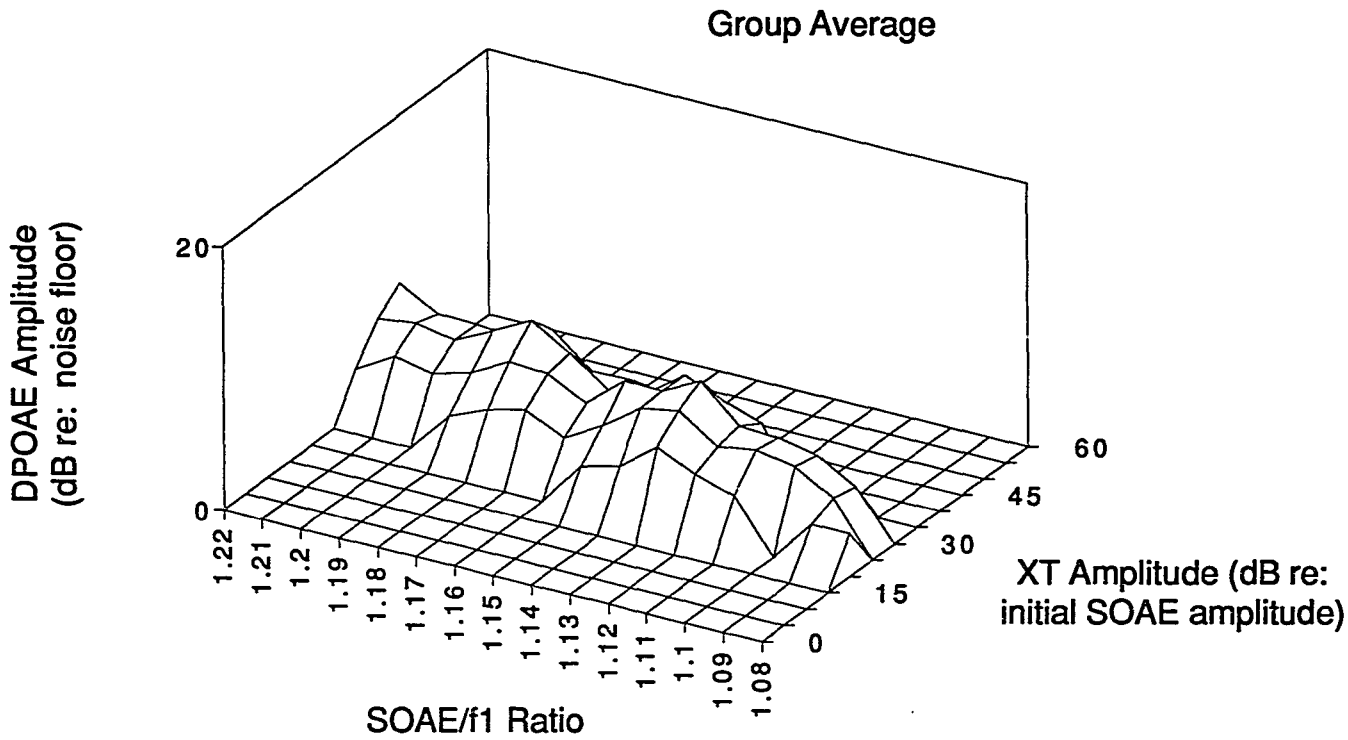

Figure 11. DPOAE amplitude group means as a function of SOAE/F1 ratio and XT amplitude.

Table 11. Individual Subject Data Including Ratios That Failed to Produce DPOAEs, Distance Between Maximum (Max) DPOAE and F_2, Characteristics of New SOAEs not Observed During the Screening Session but Present During the Test Session, Relative XT Levels Associated With the Maximum DPOAE and Figure Numbers for Each Subject's Ratio Graph and 3-D Plot.

Subject	Ratio(s) without DPOAEs	Distance between Max DPOAE & f_2 (octave)	Max DPOAE amplitude (dB SPL)	New SOAEs (Hz)	New SOAEs (dB SPL)	Relative XT levels for DPOAE Maximum	Figures
KM	1.16	0.35	4.7	1369	11	25	12A & B, 13
AR	1.12, 1.20	0.40	1.0	2906	-14 to -4	30	14A & B, 15
RA	---	0.52	1.2	---	---	30	16A & B, 17
JS	---	0.44	4.7	3207	-15 to -8	40	18A & B, 19
MK	---	0.53	1.8	---	---	50	20A & B, 21
IR	---	0.65	5.9	1184	-9	45	22A & B, 23
JV	1.08, 1.09 1.15, 1.17	0.38	-0.5	1805 2512	-5 -10	30	24A & B, 25
BR	---	0.52	1.0	1838	-12 to -2	35	26A & B, 27
LH	1.10, 1.11 1.20, 1.21 1.22	0.44	-2.8	3312 3187	-12 to -7 -12	30	28A & B, 29
KS	—	0.50	5.4	—	—	40	30A & B, 31

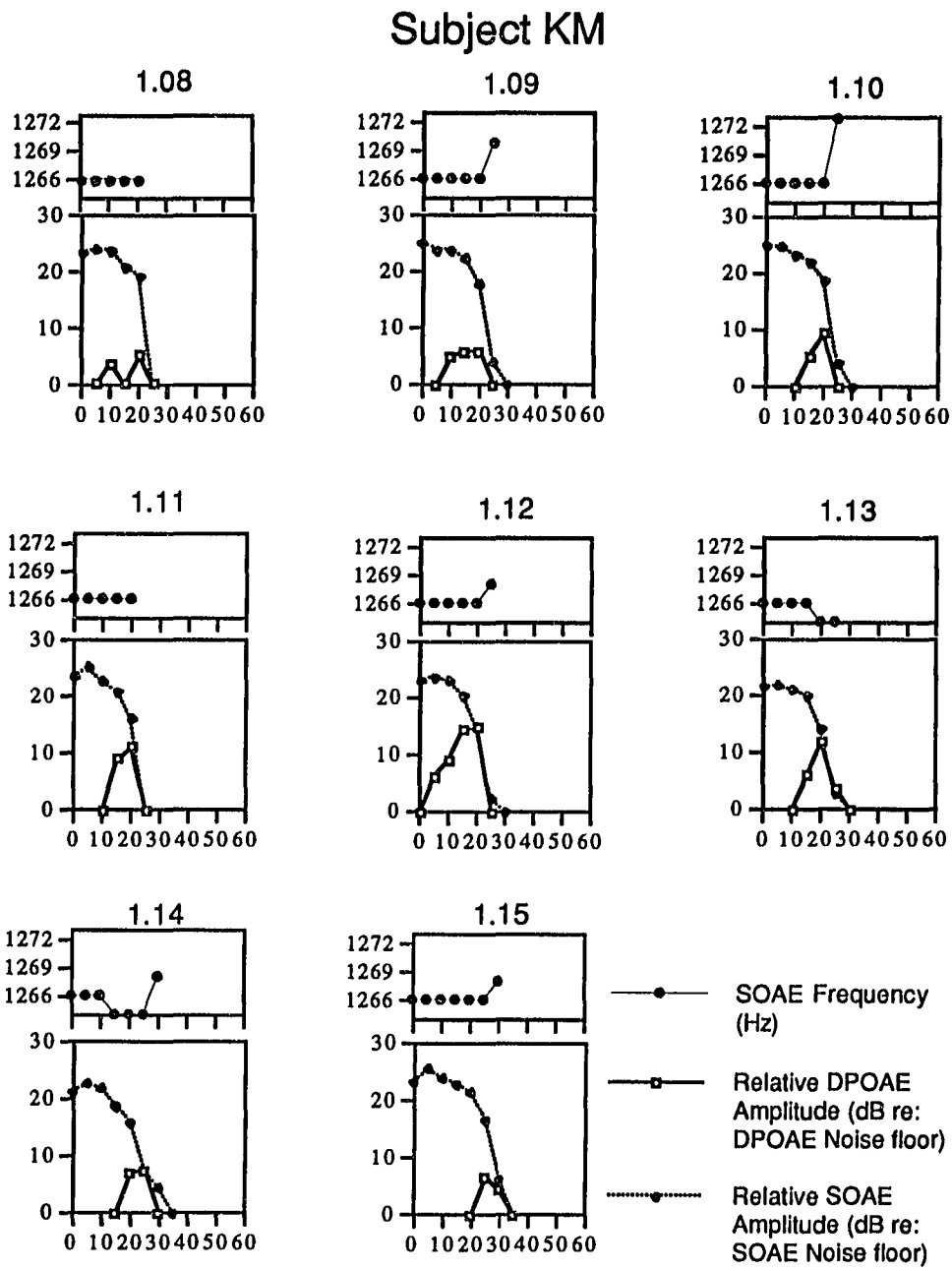

Figure 12A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re:Initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

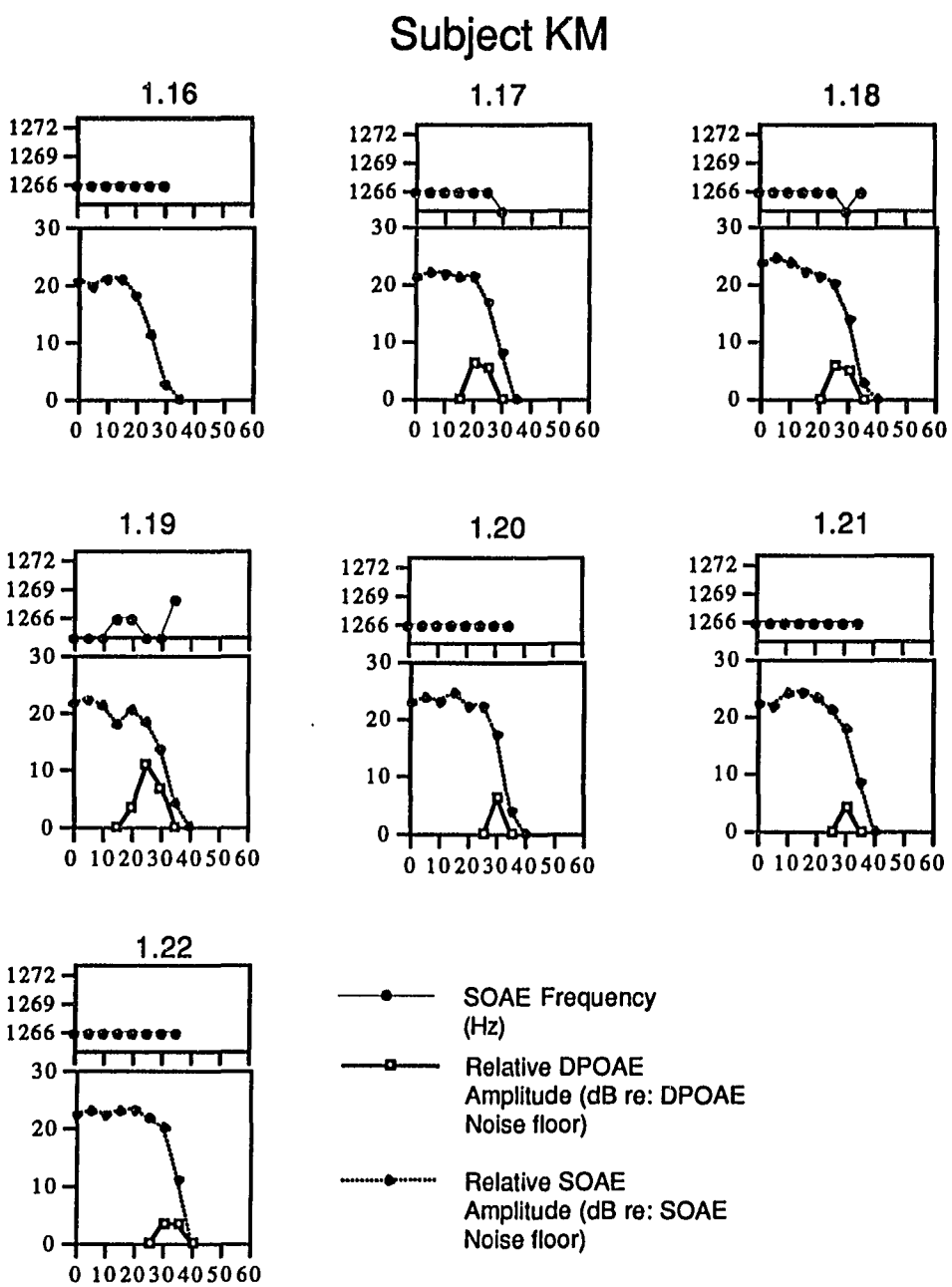

Figure 12B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X- axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: DPOAE and SOAE amplitudes as a function of relative XT level.

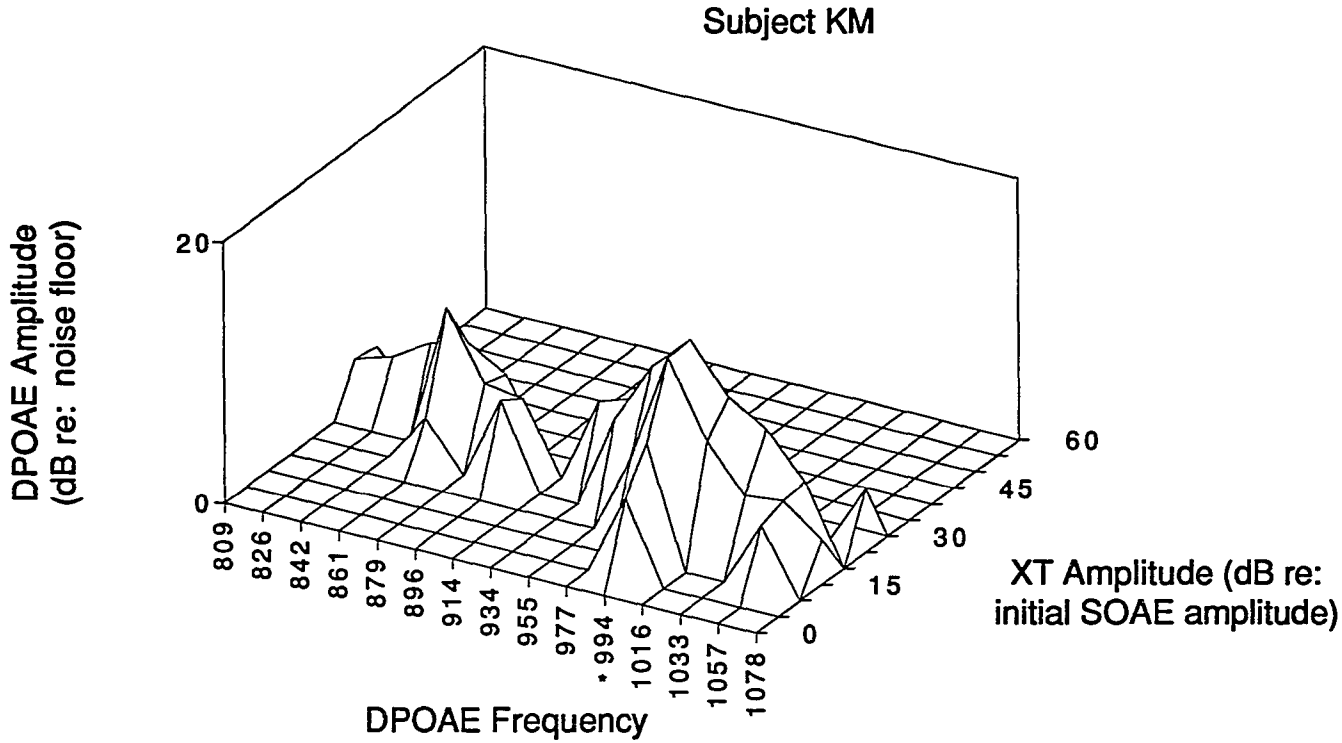

Figure 13. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

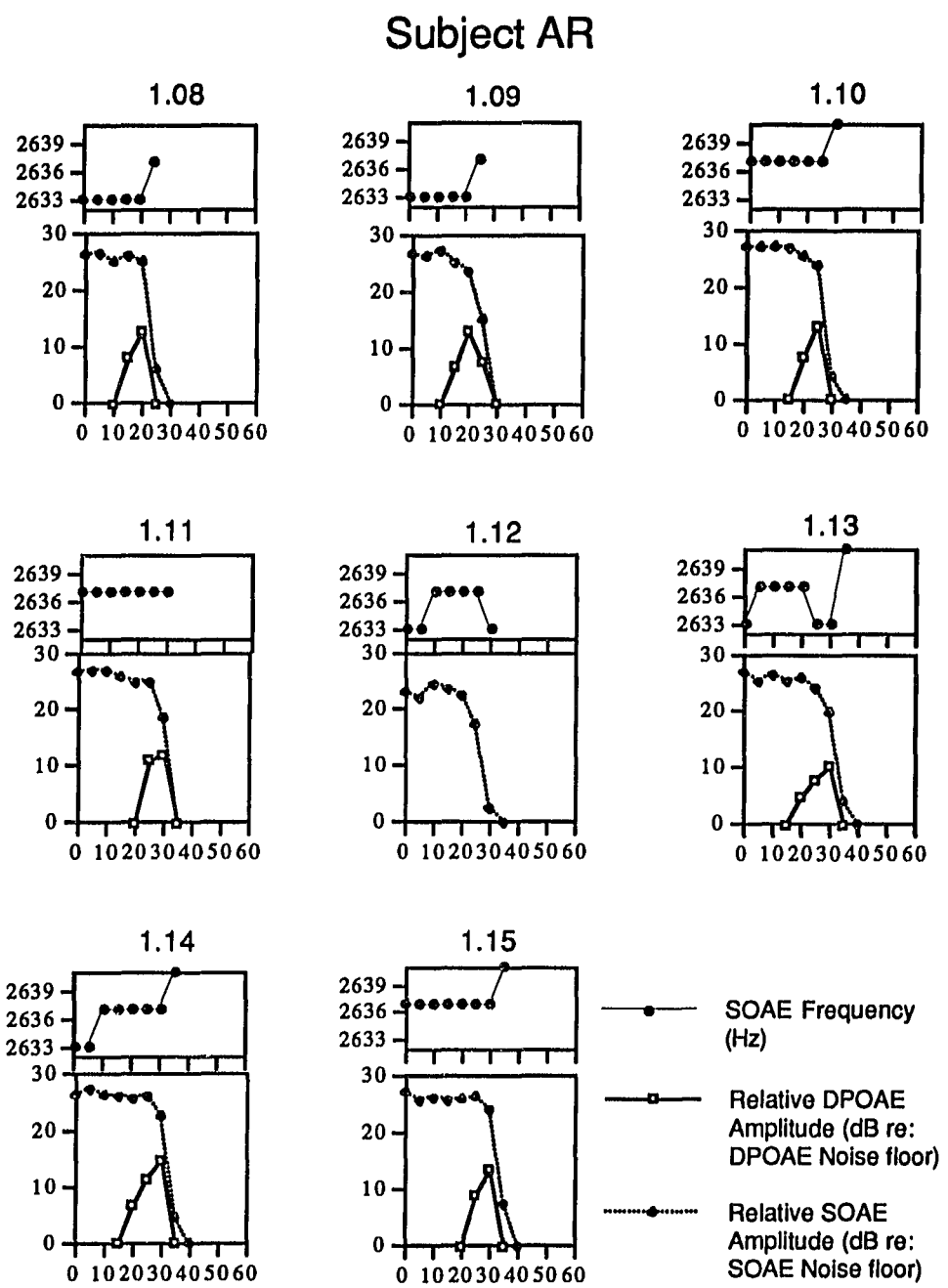

Figure 14A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

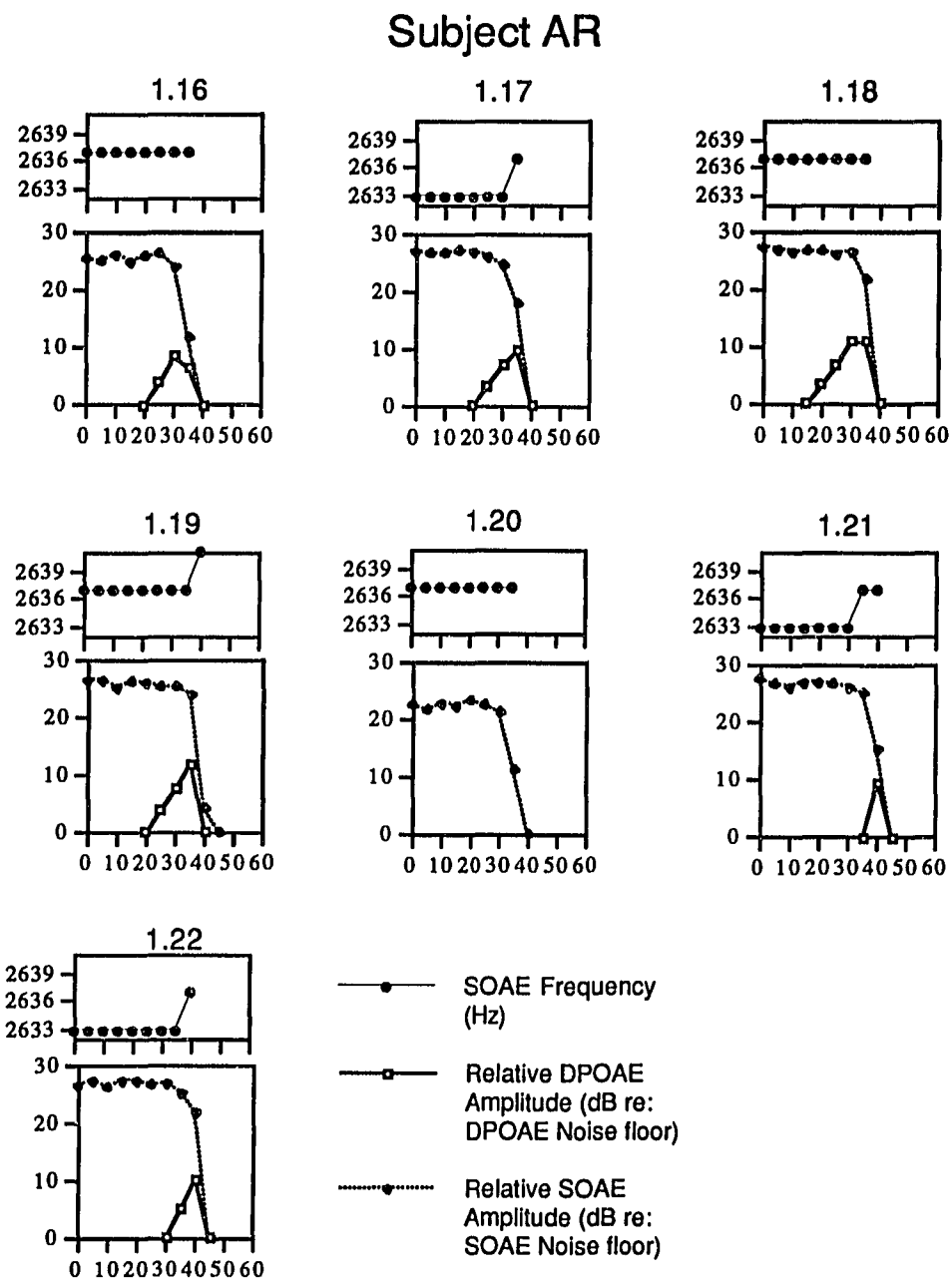

Figure 14B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: DPOAE and SOAE amplitudes as a function of relative XT level.

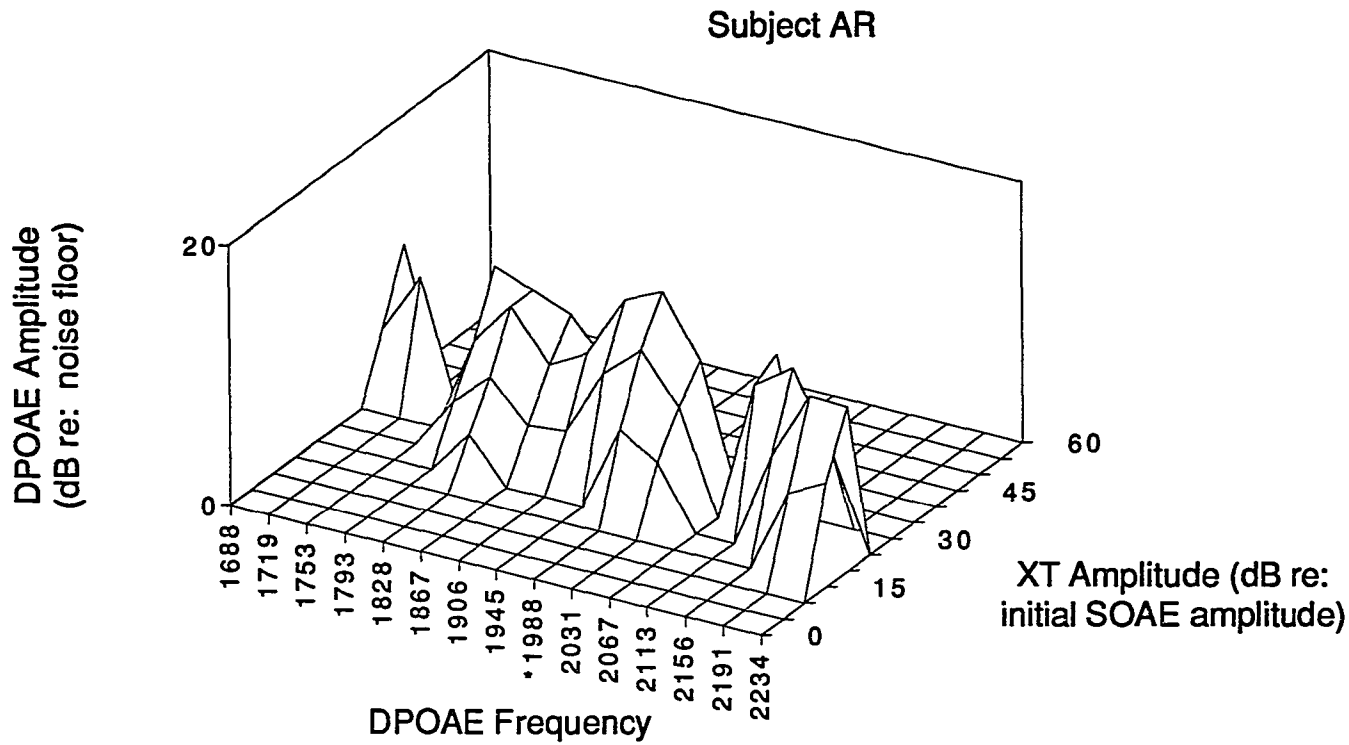

Figure 15. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

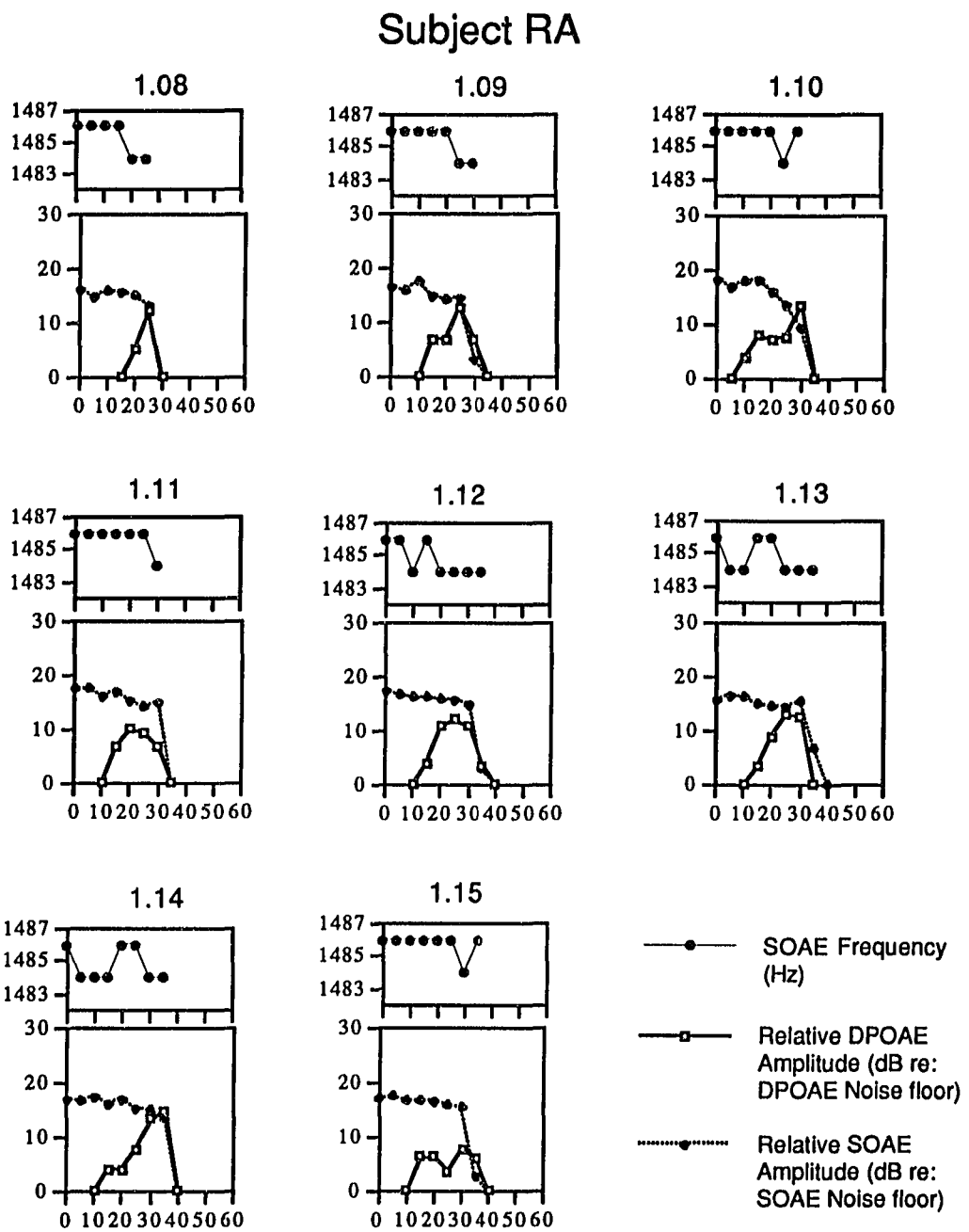

Figure 16A. Individual ratio graphs for SOAE/I1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

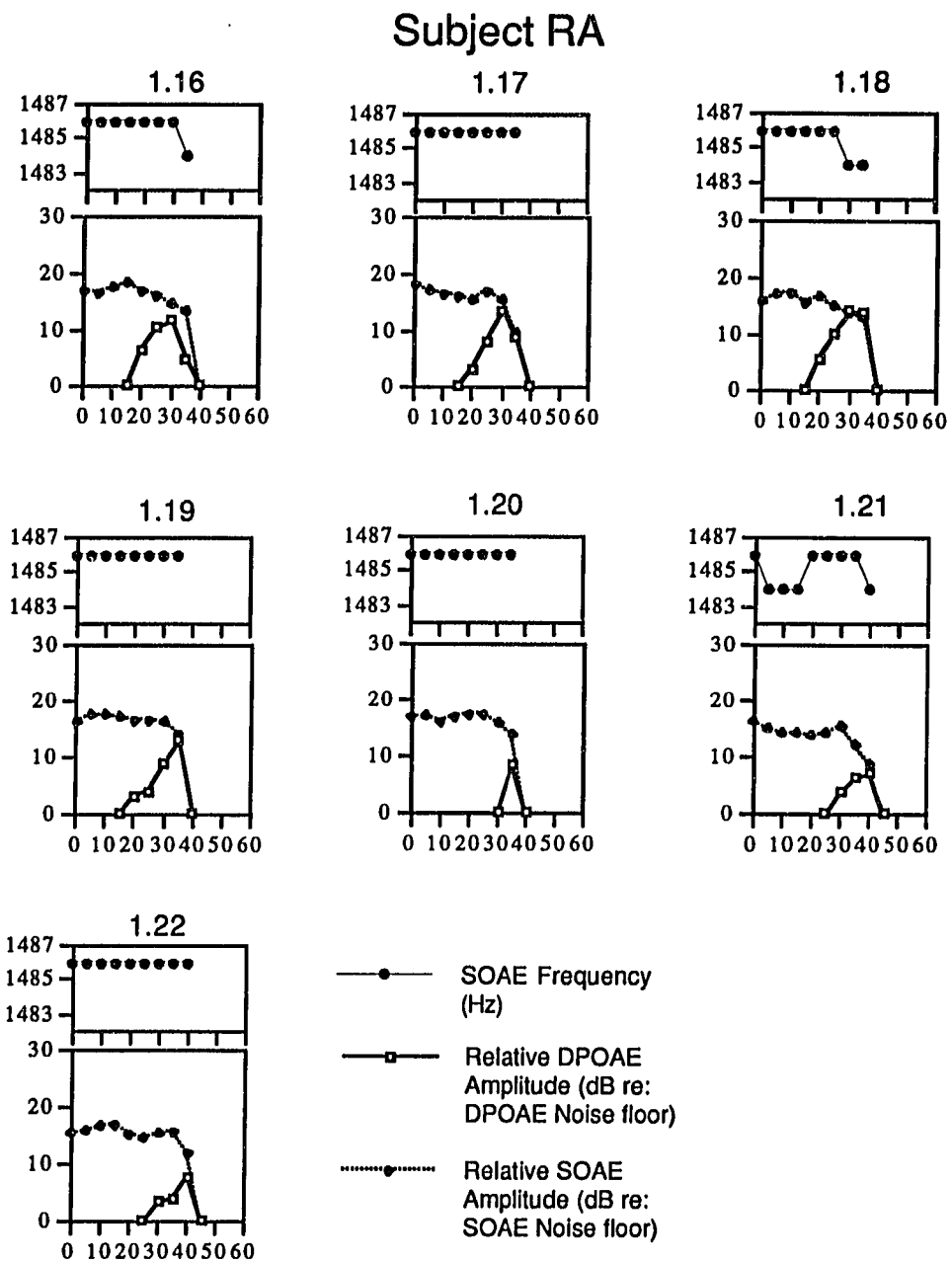

Figure 16B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

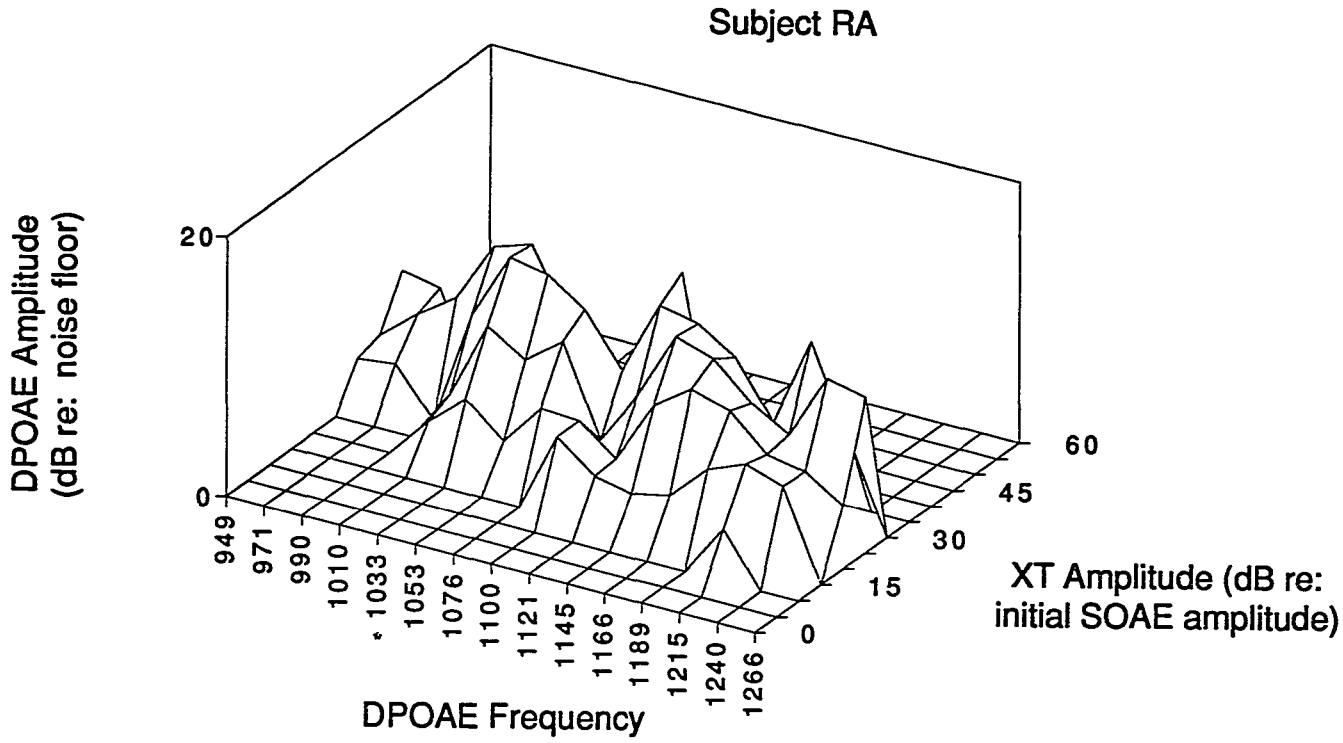

Figure 17. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

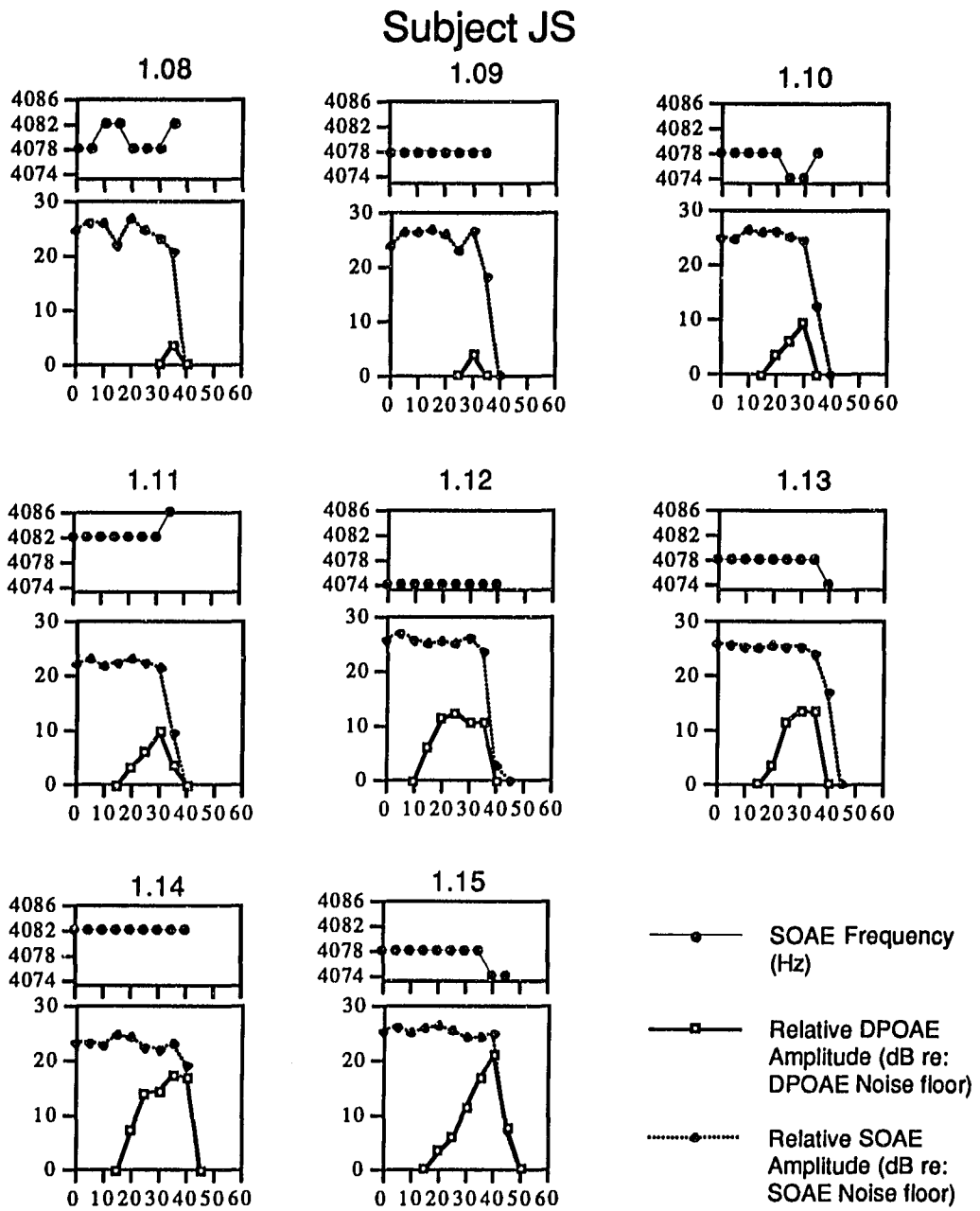

Figure 18A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

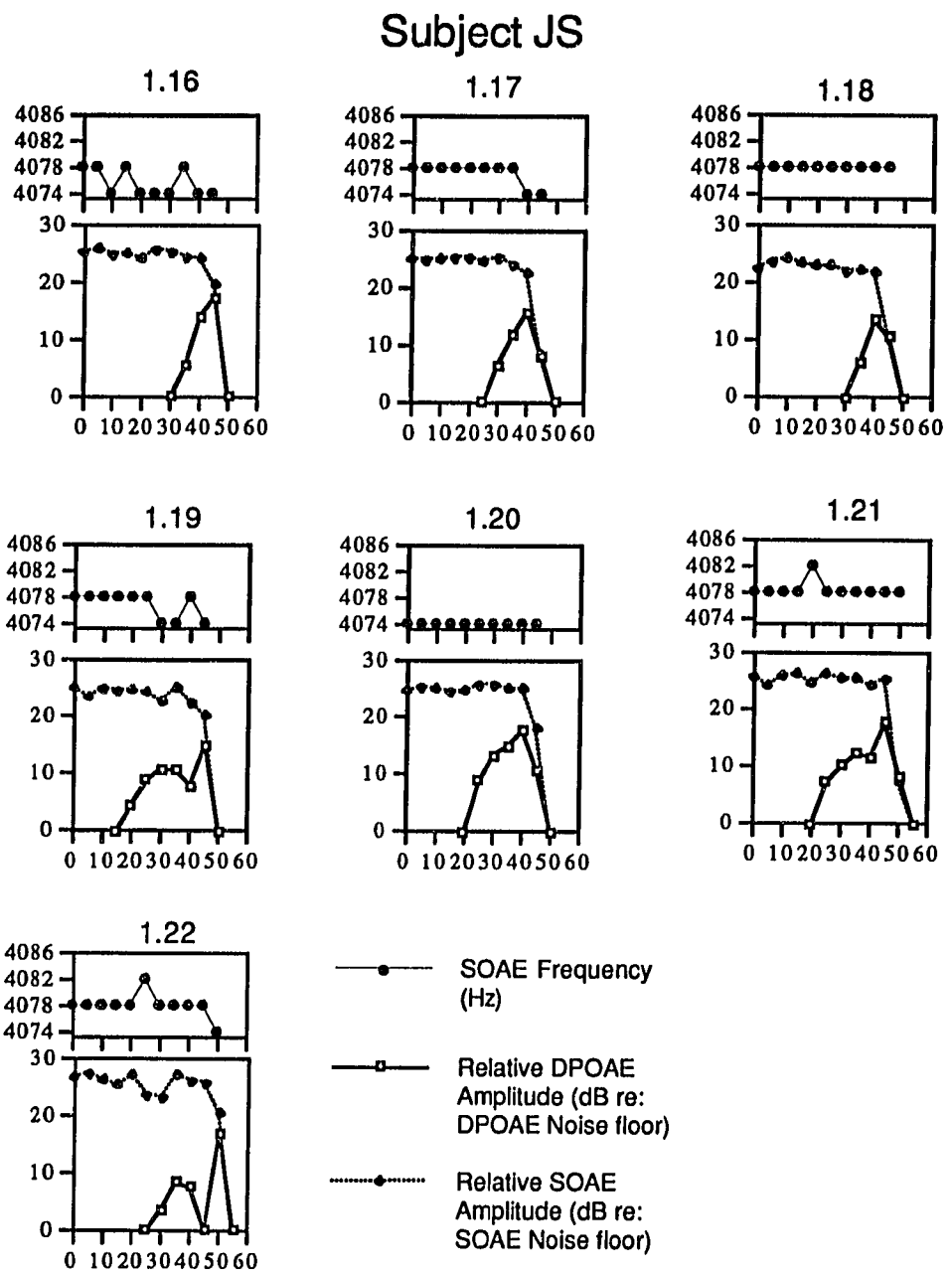

Figure 18B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

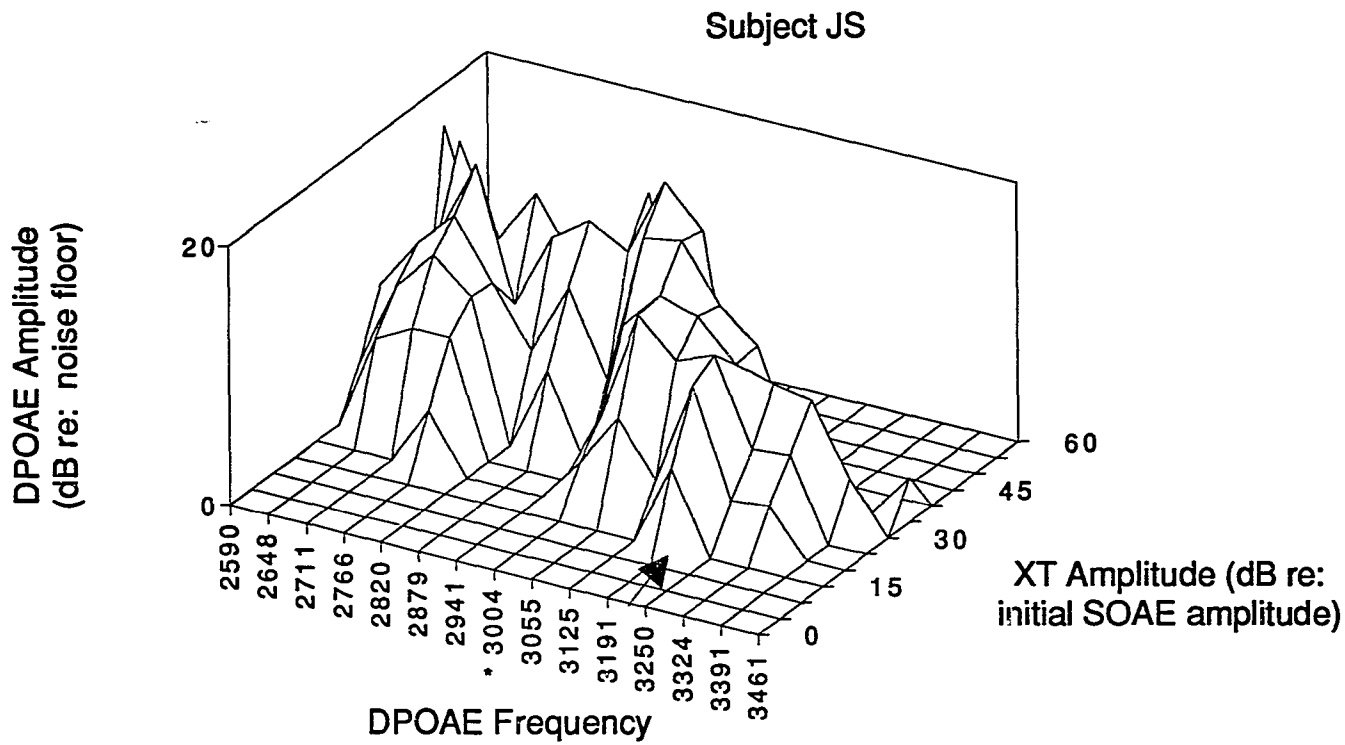

Figure 19. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE. An arrow indicates the frequency of a new SOAE.

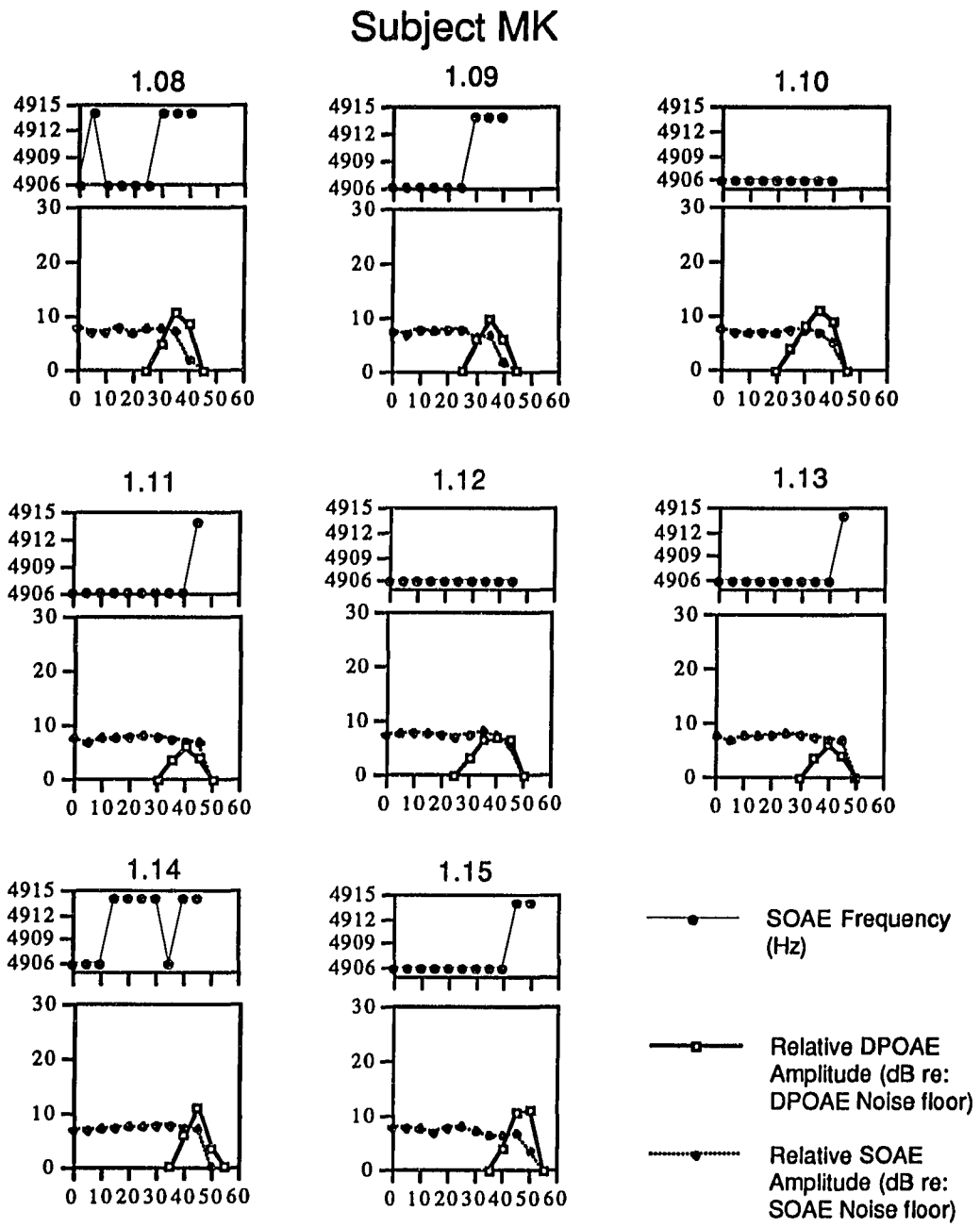

Figure 20A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

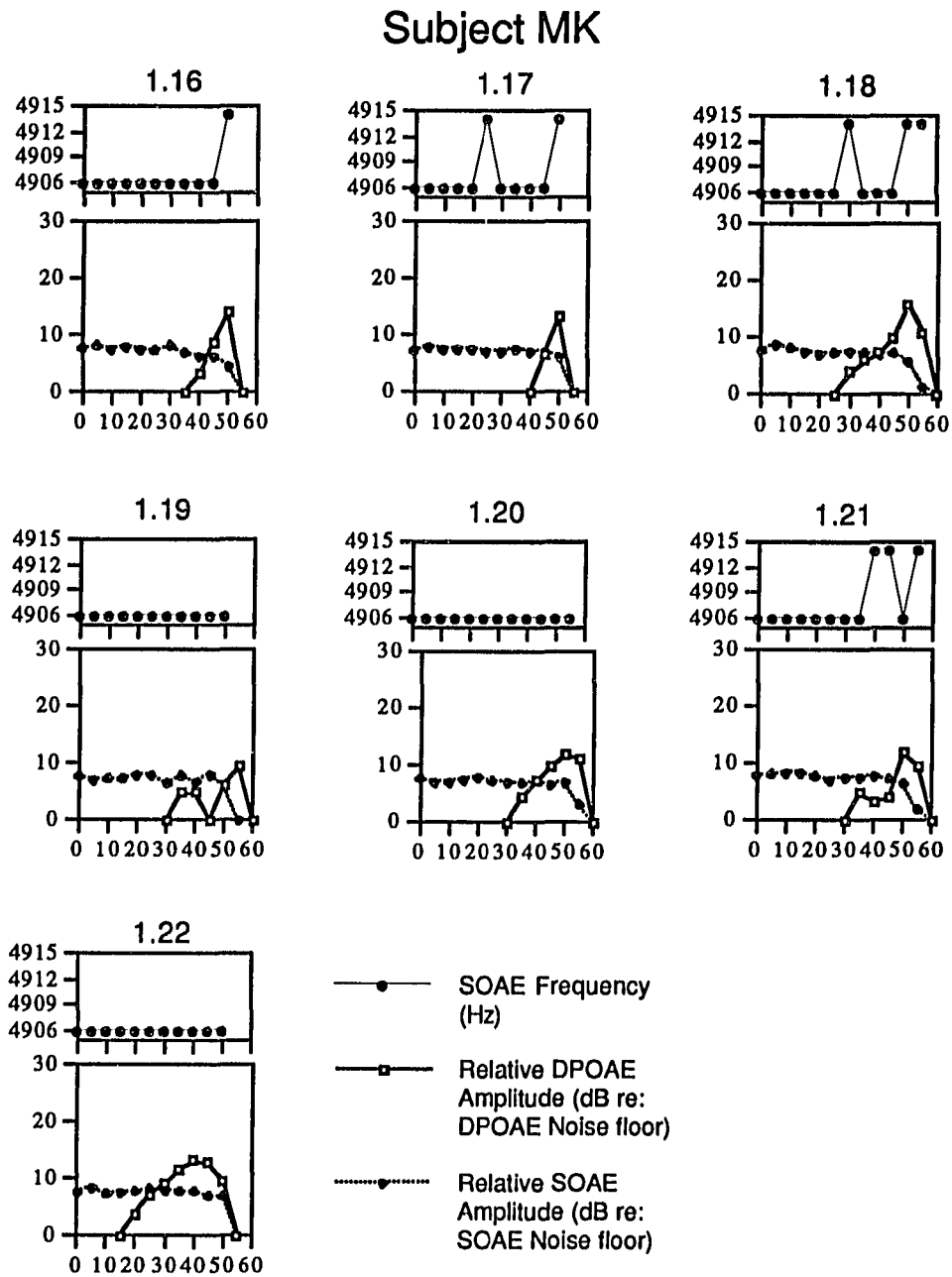

Figure 20B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

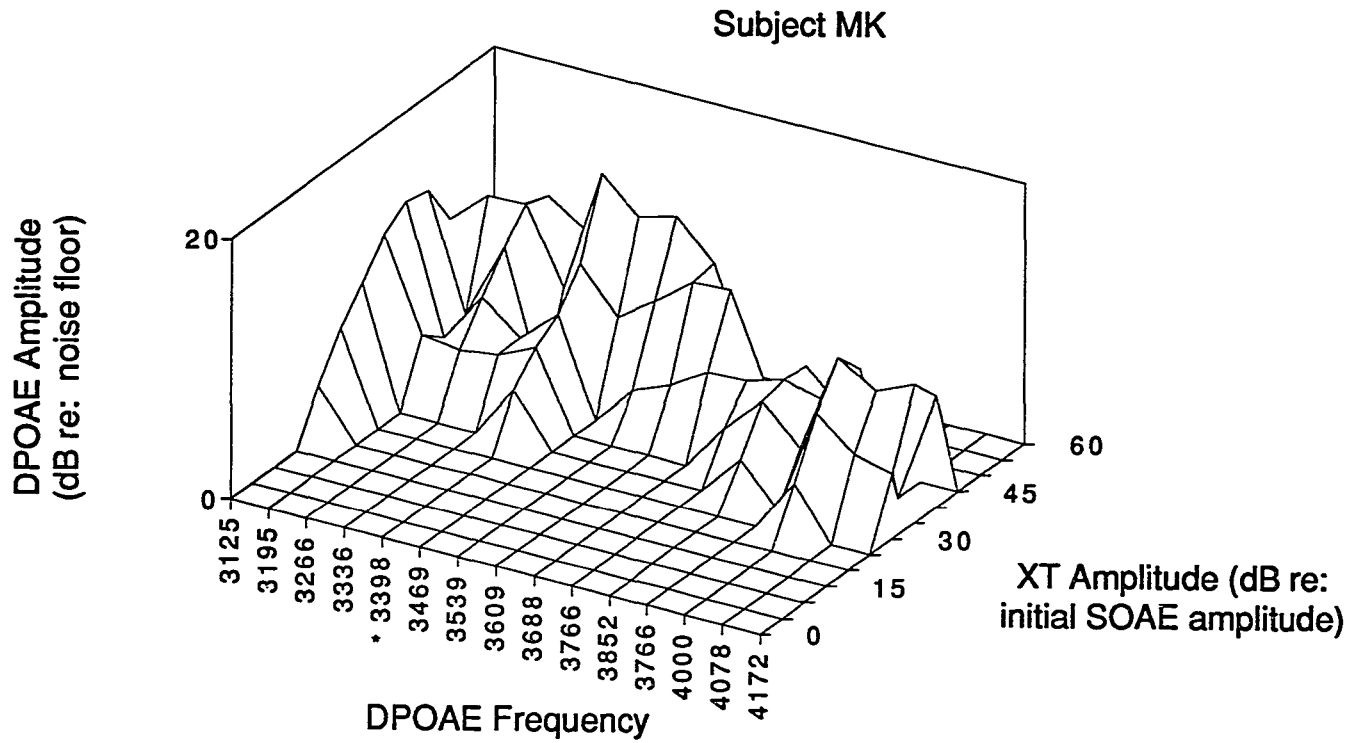

Figure 21. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

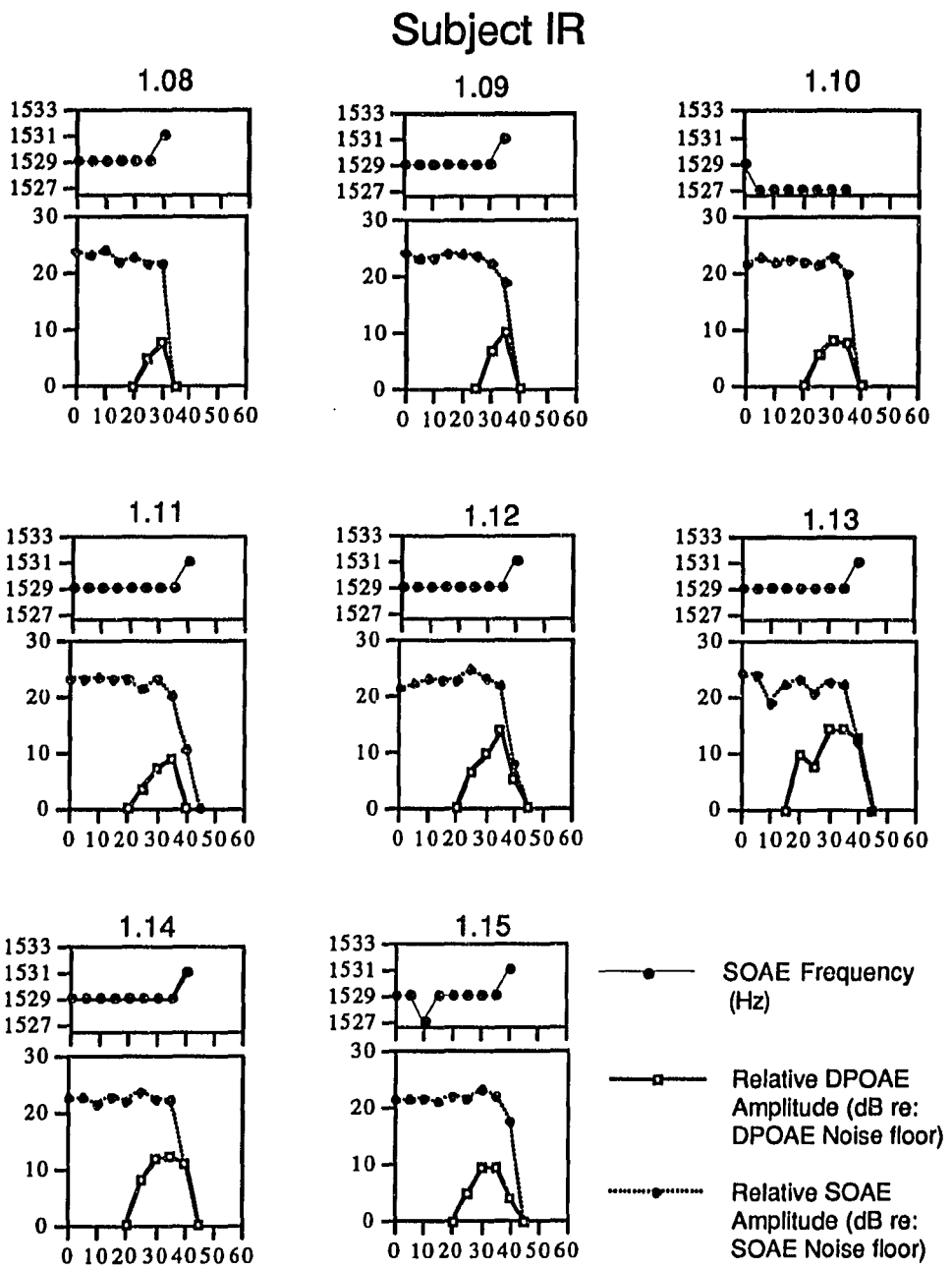

Figure 22A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

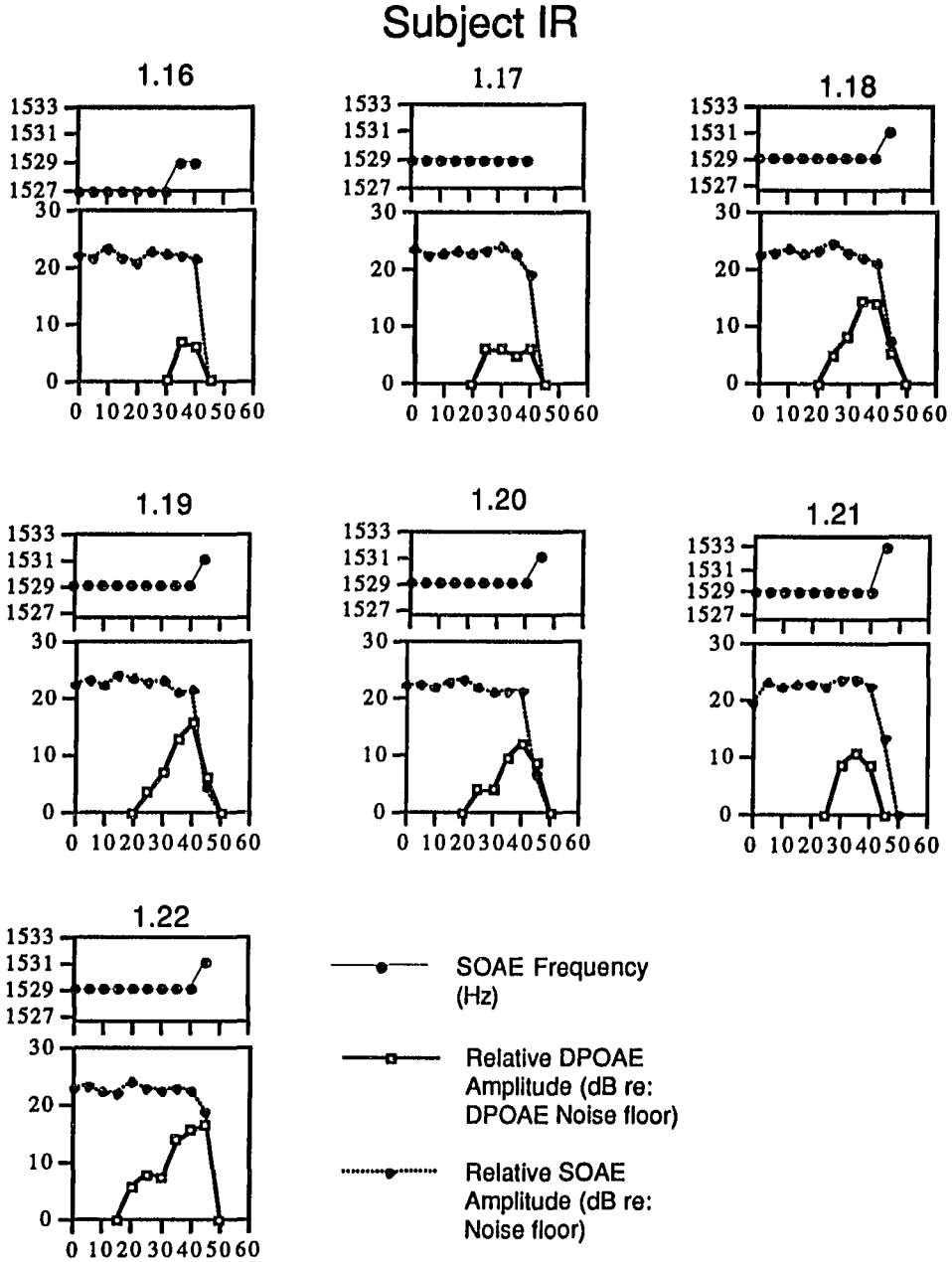

Figure 22B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

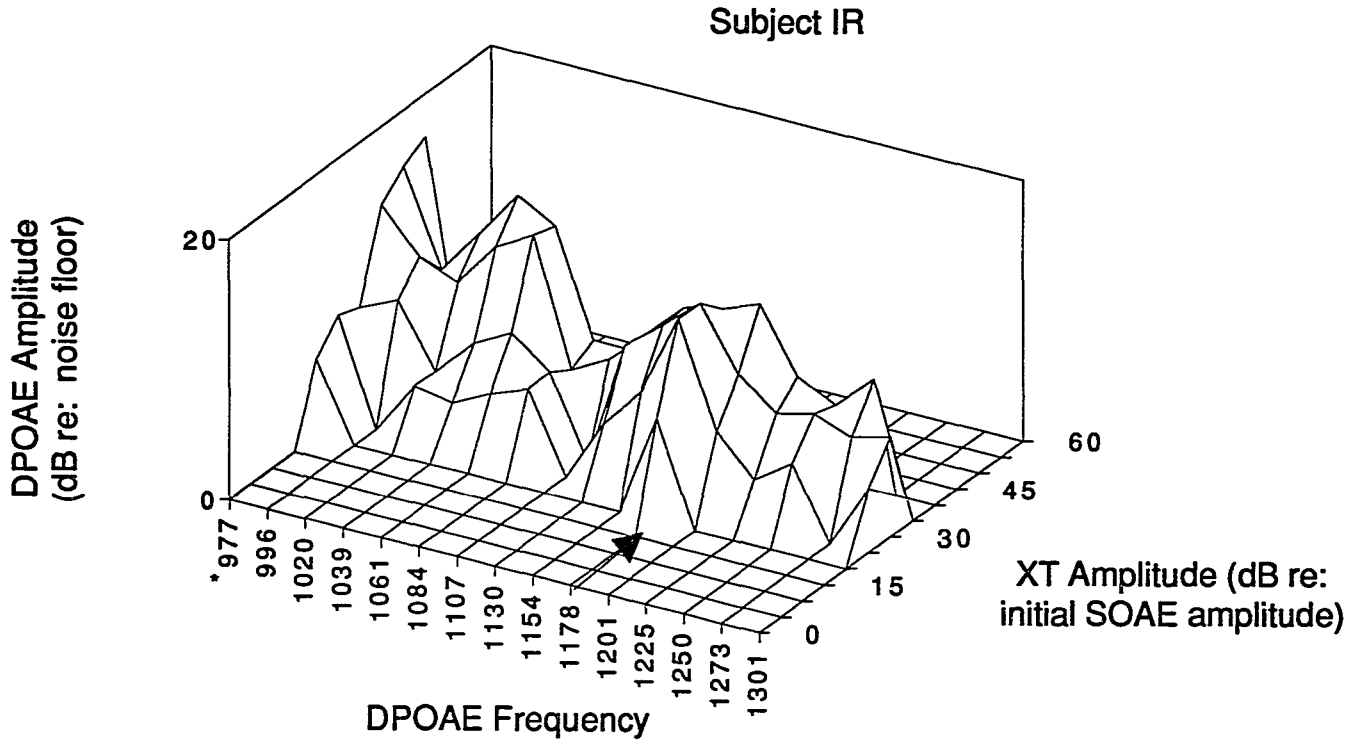

Figure 23. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE. An arrow indicates the frequency of a new SOAE.

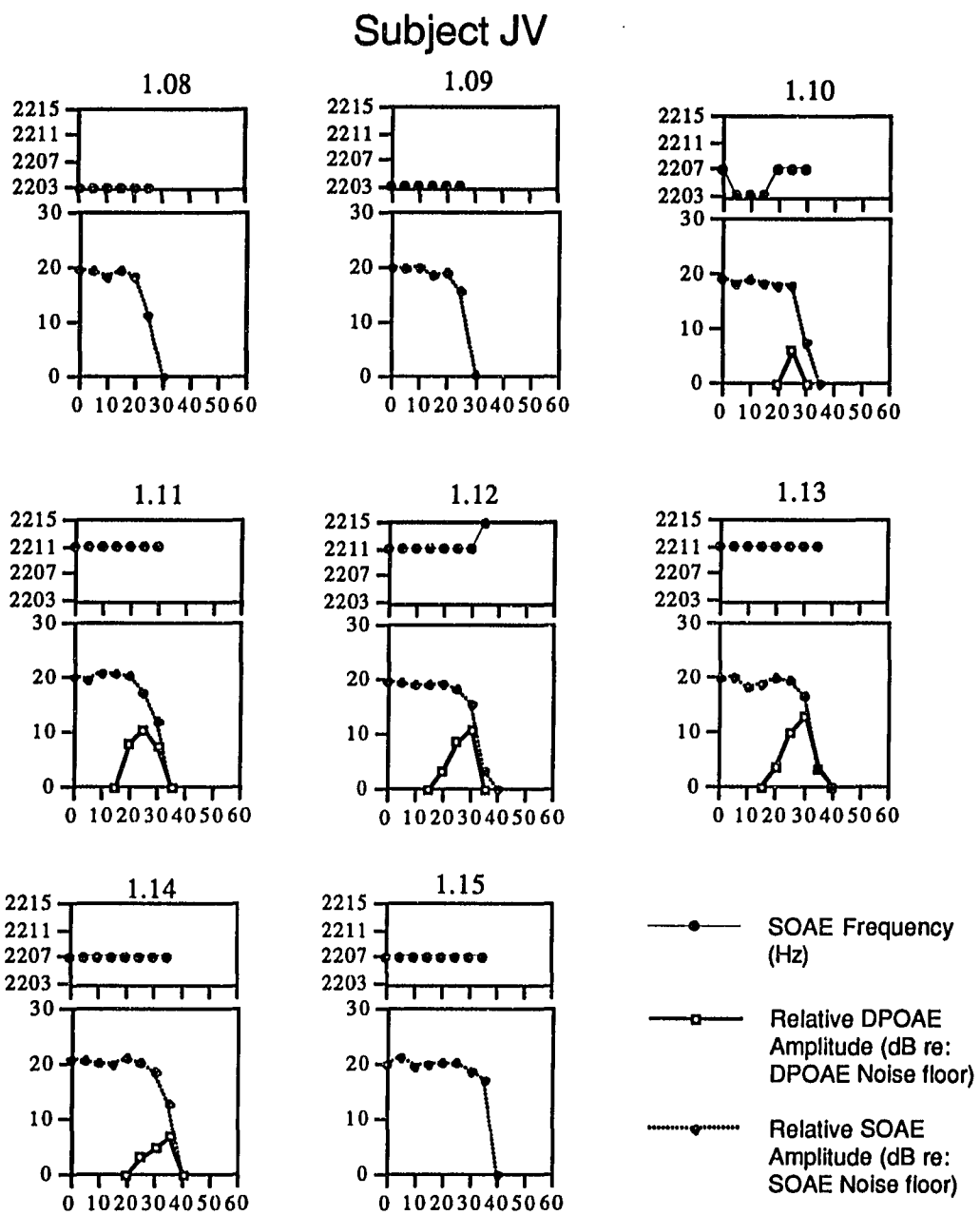

Figure 24A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

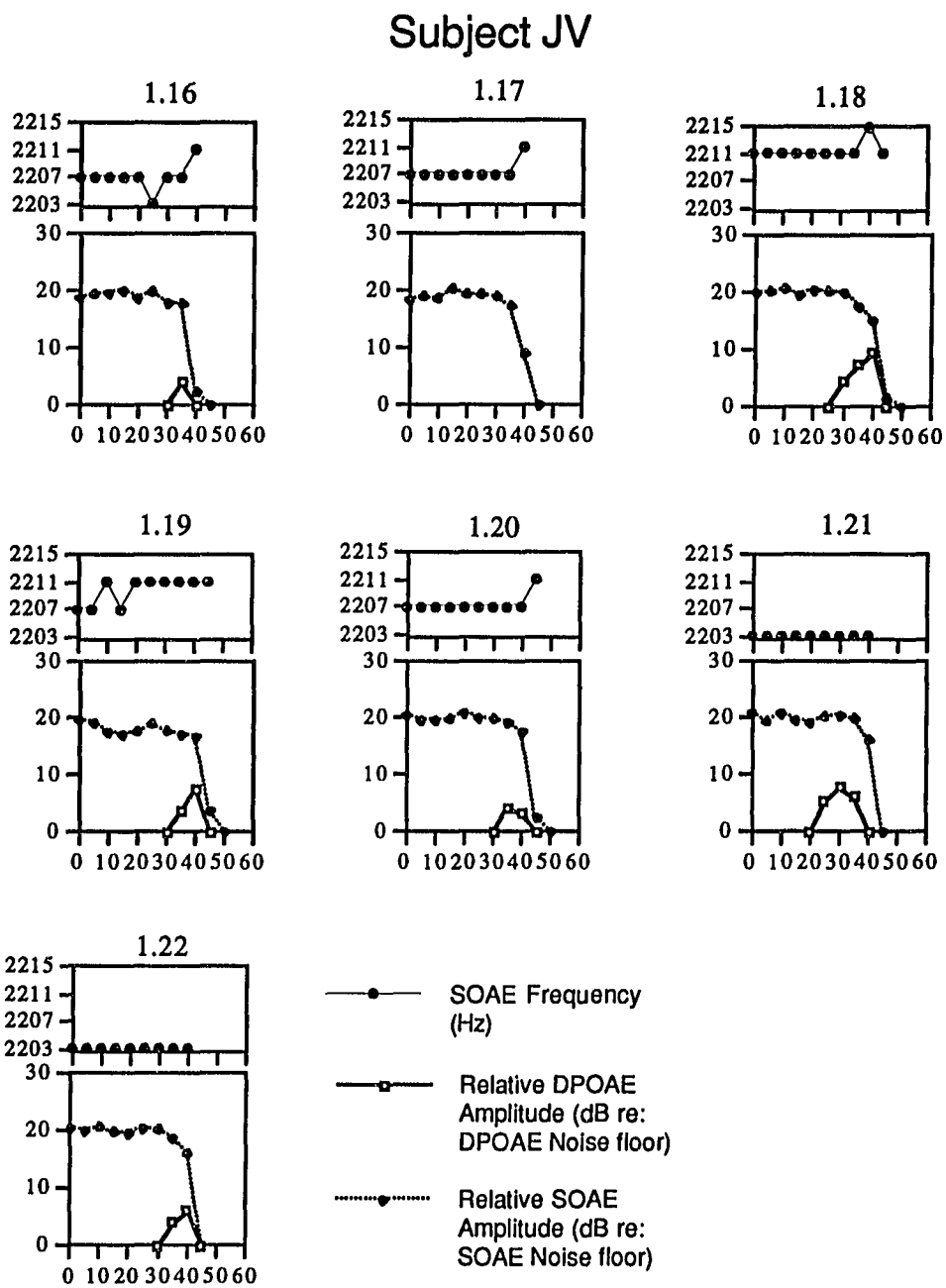

Figure 24B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

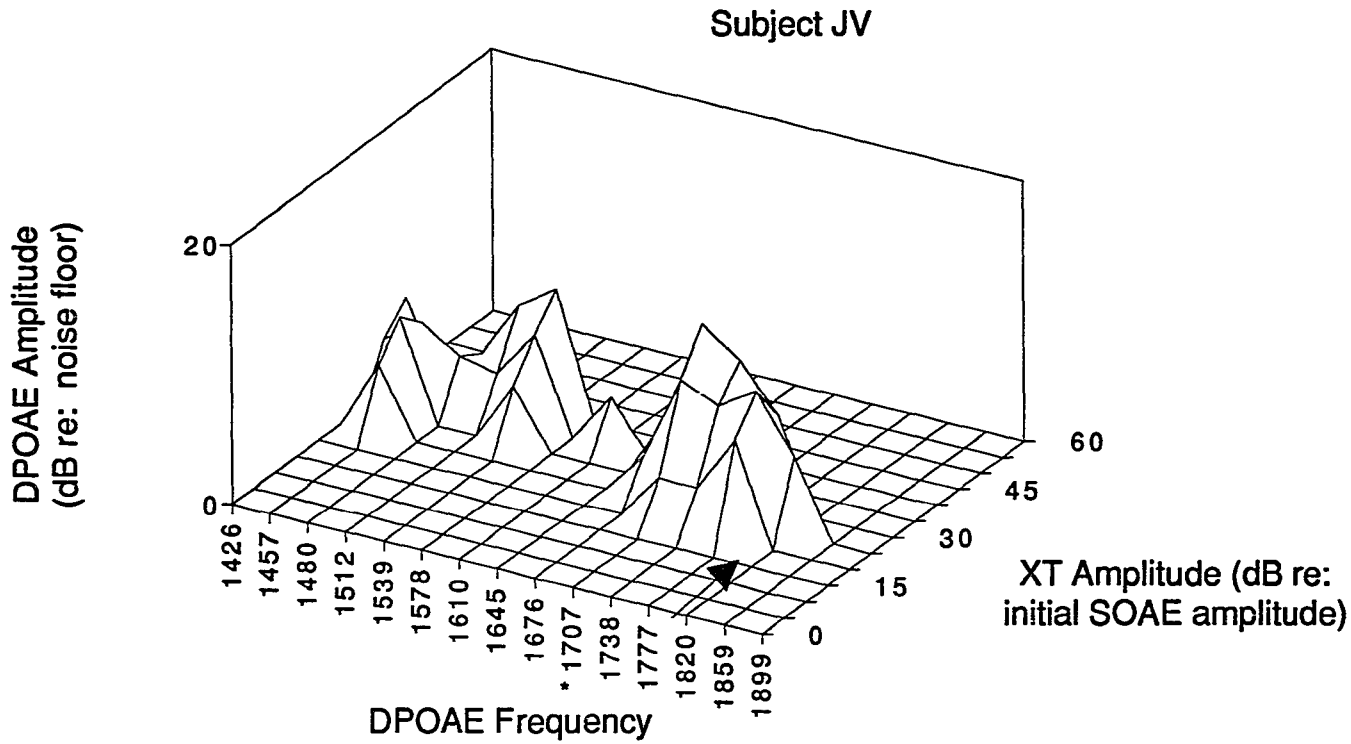

Figure 25. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE. An arrow indicates the frequency of a new SOAE.

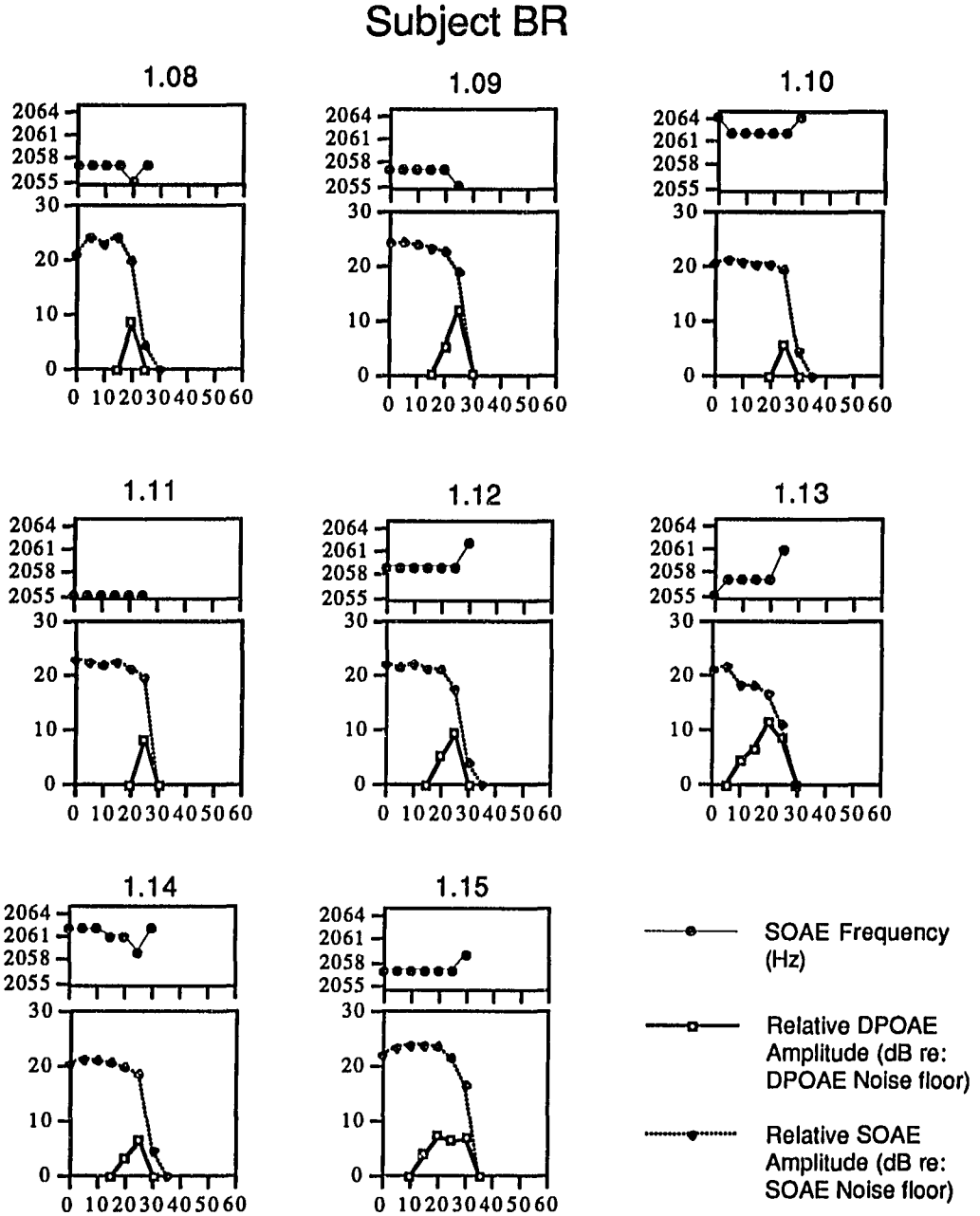

Figure 26A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

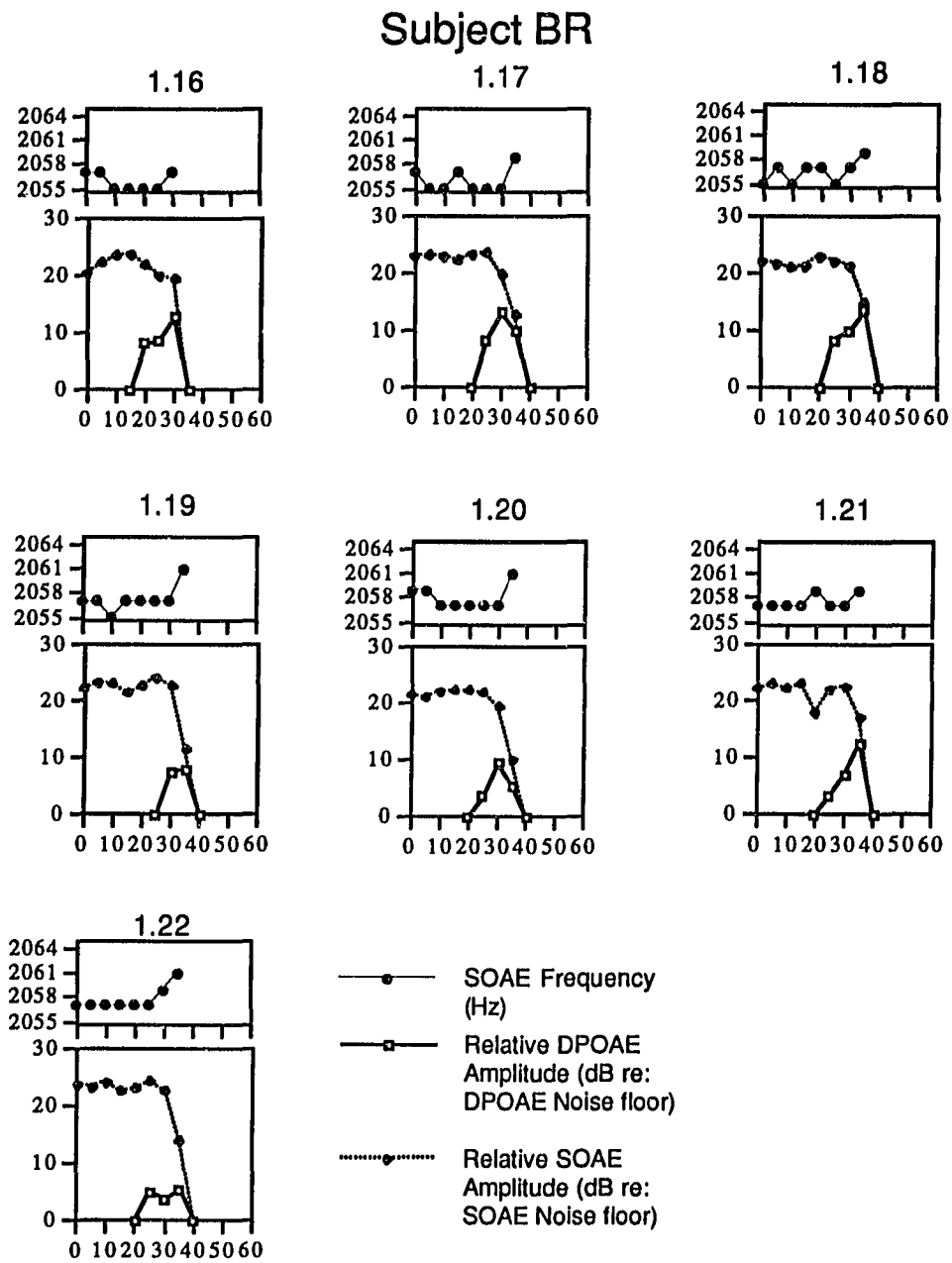

Figure 26B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

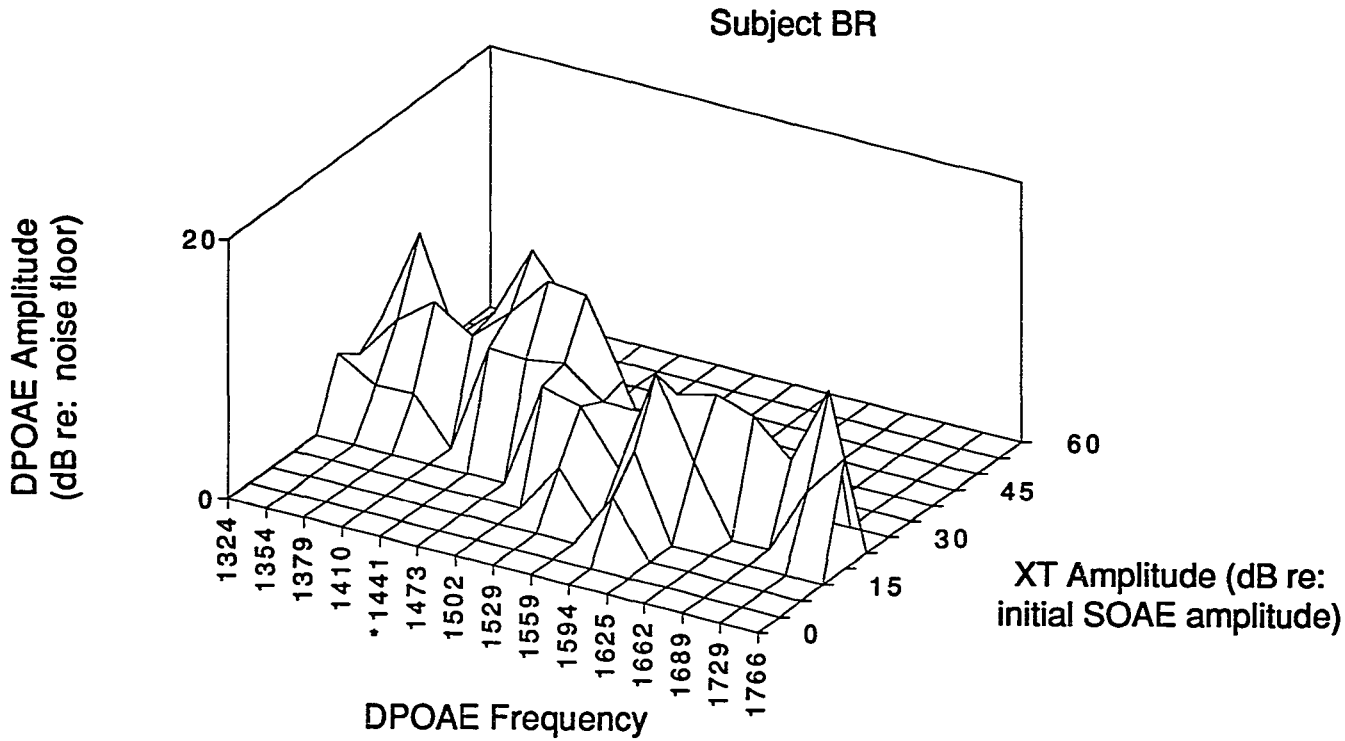

Figure 27. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

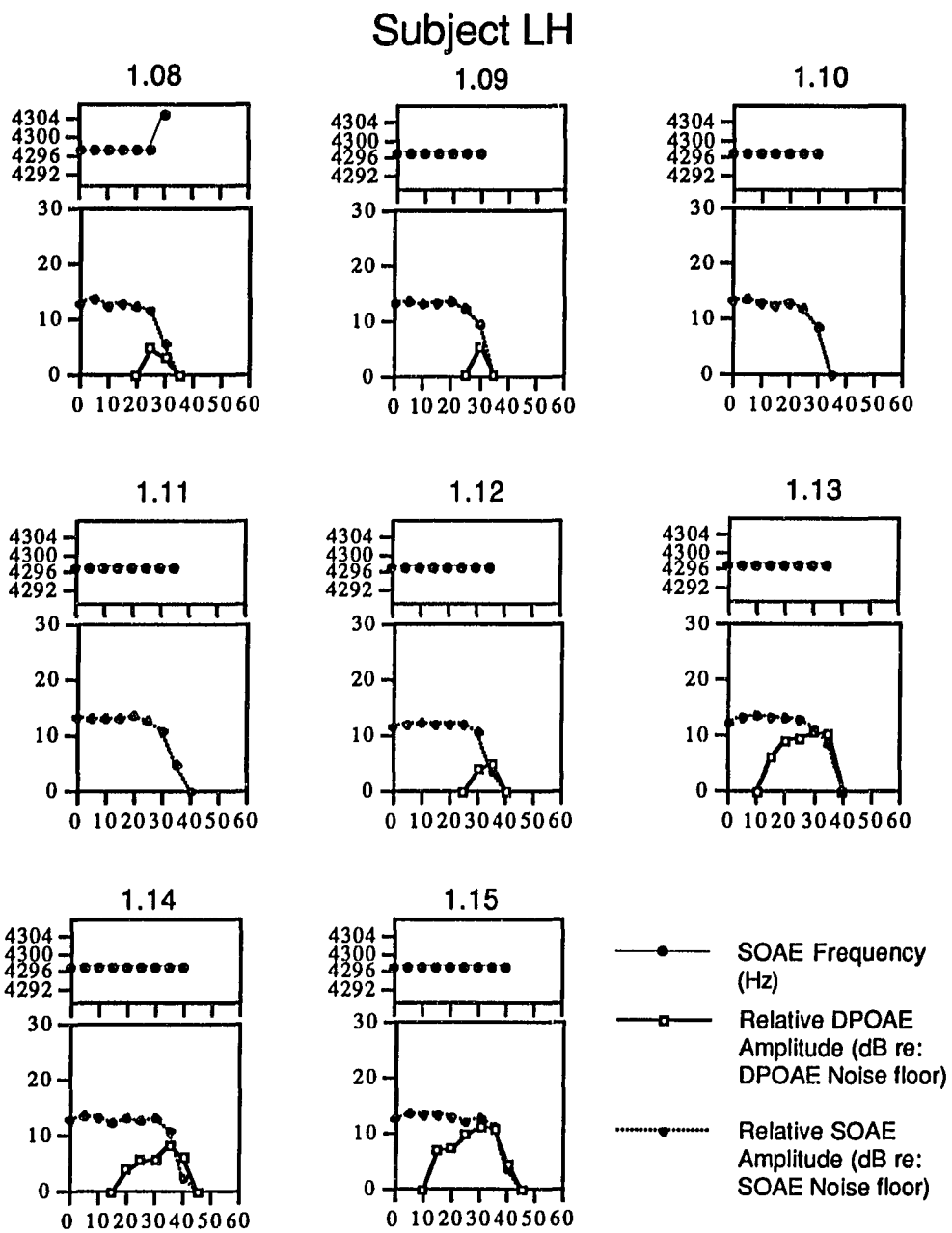

Figure 28A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

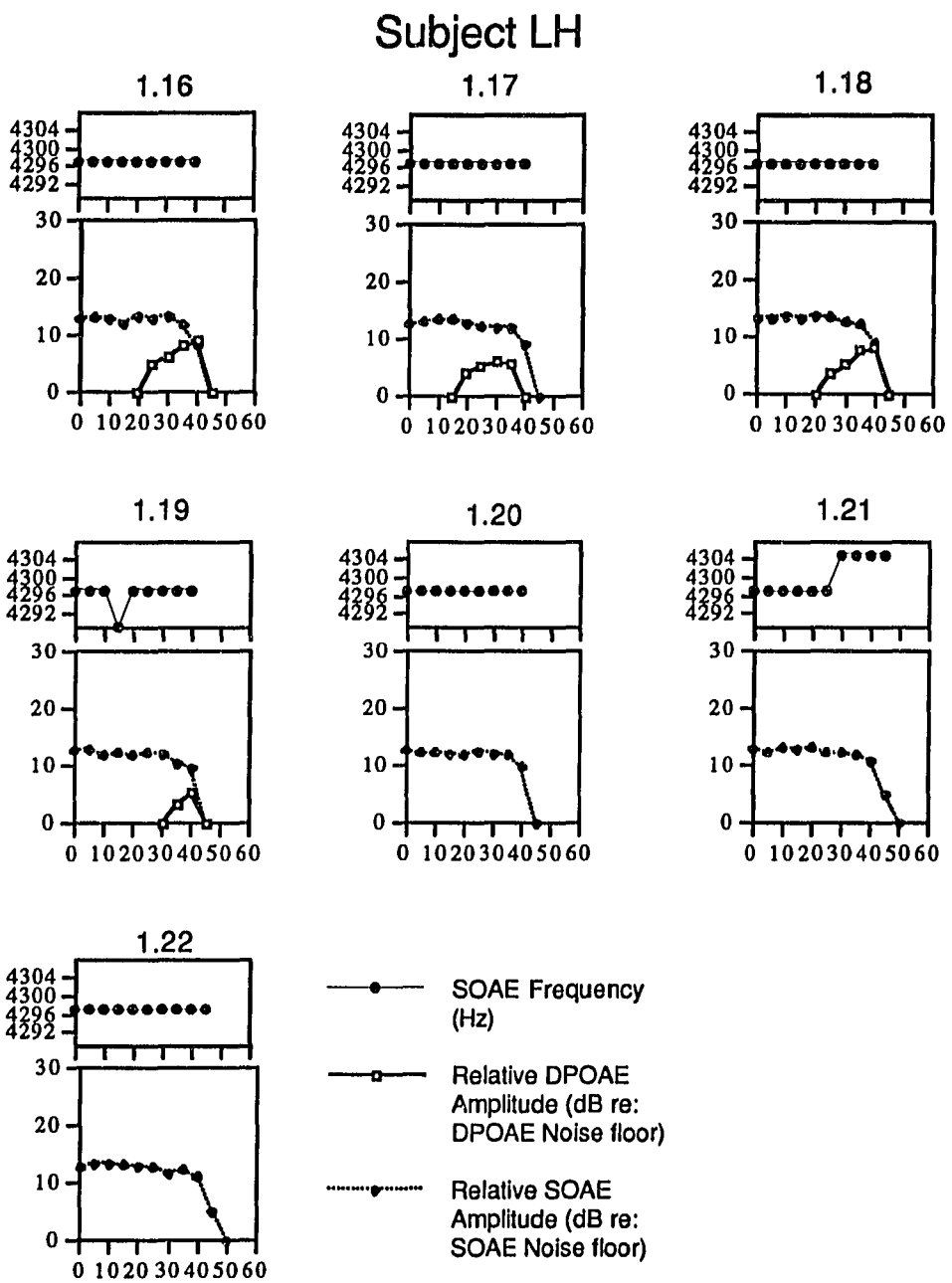

Figure 28B. Individual ratio graphs for SOAE/I1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

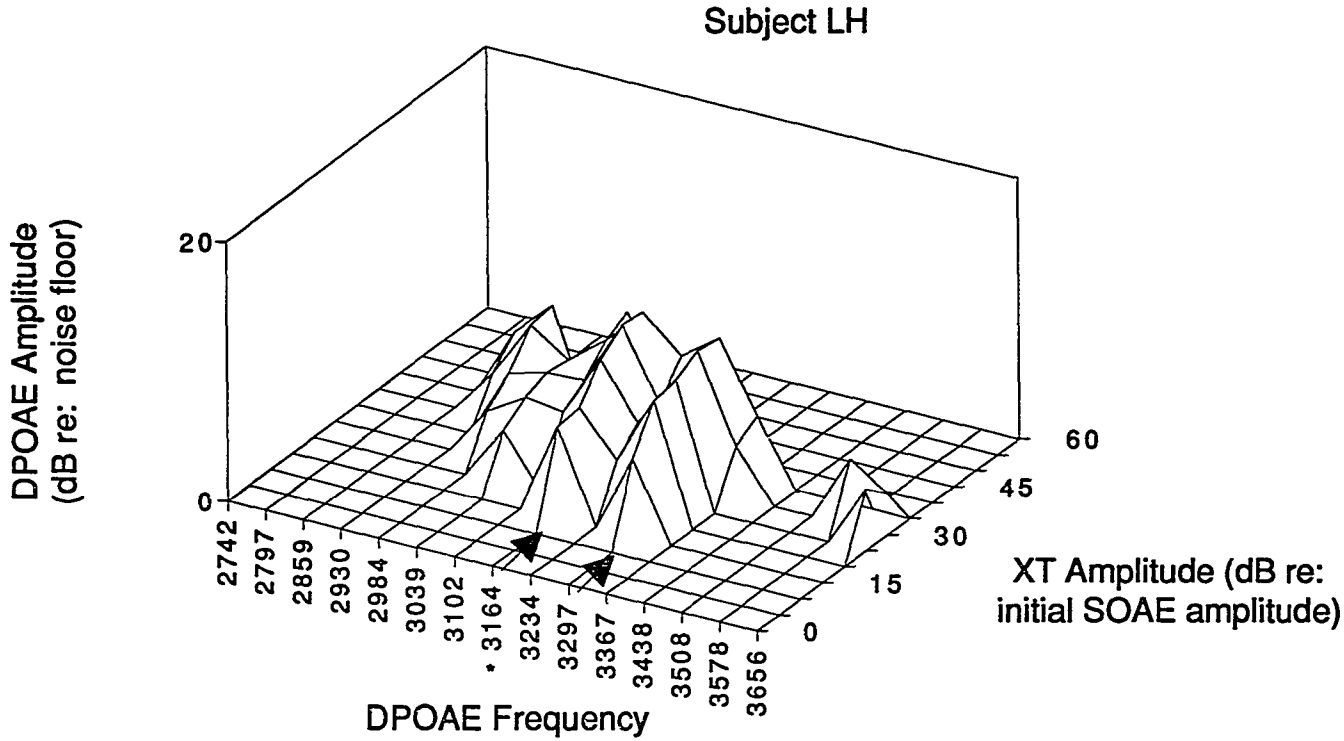

Figure 29. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE. An arrow indicates the frequency of a new SOAE.

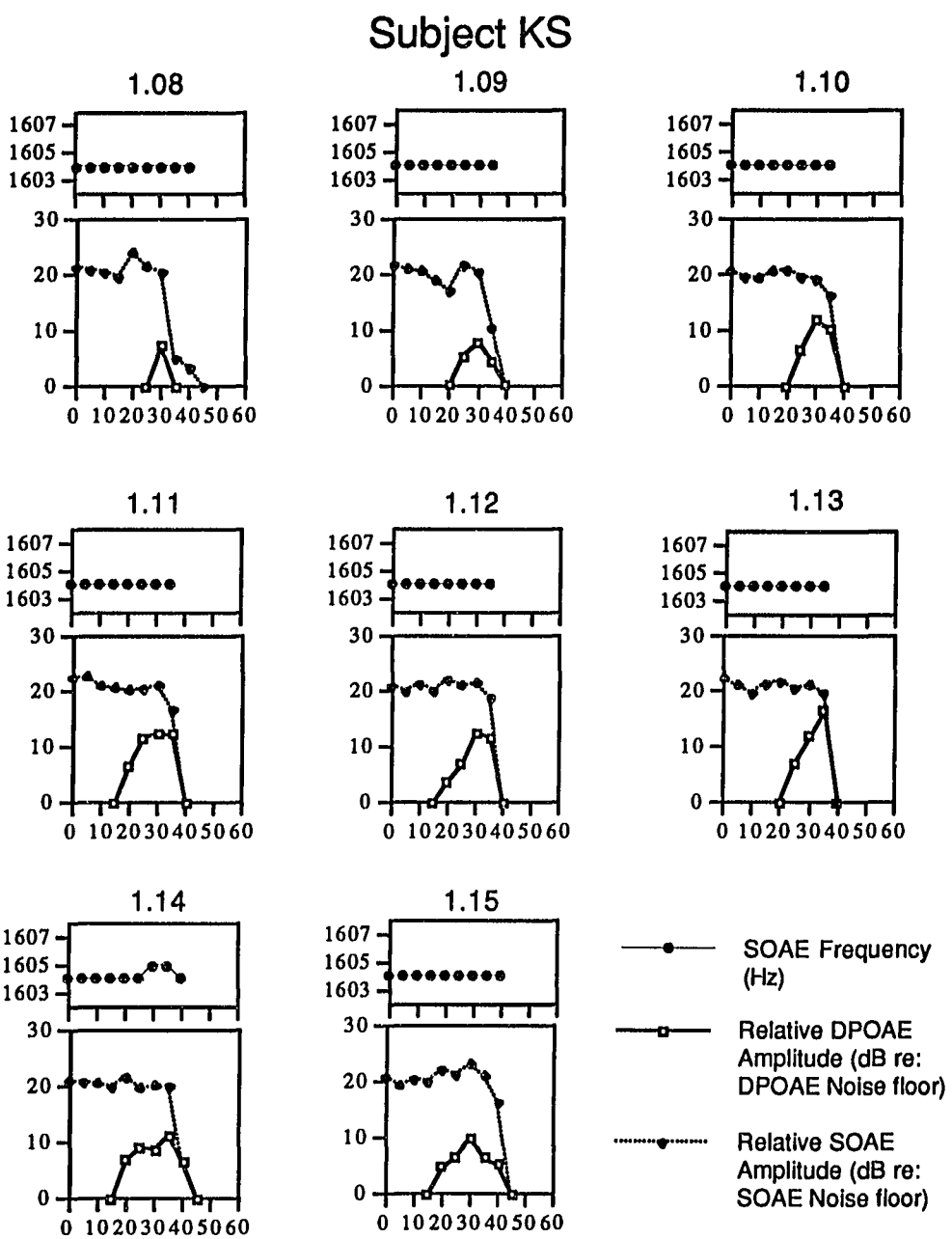

Figure 30A. Individual ratio graphs for SOAE/f1 ratios 1.08 to 1.15. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

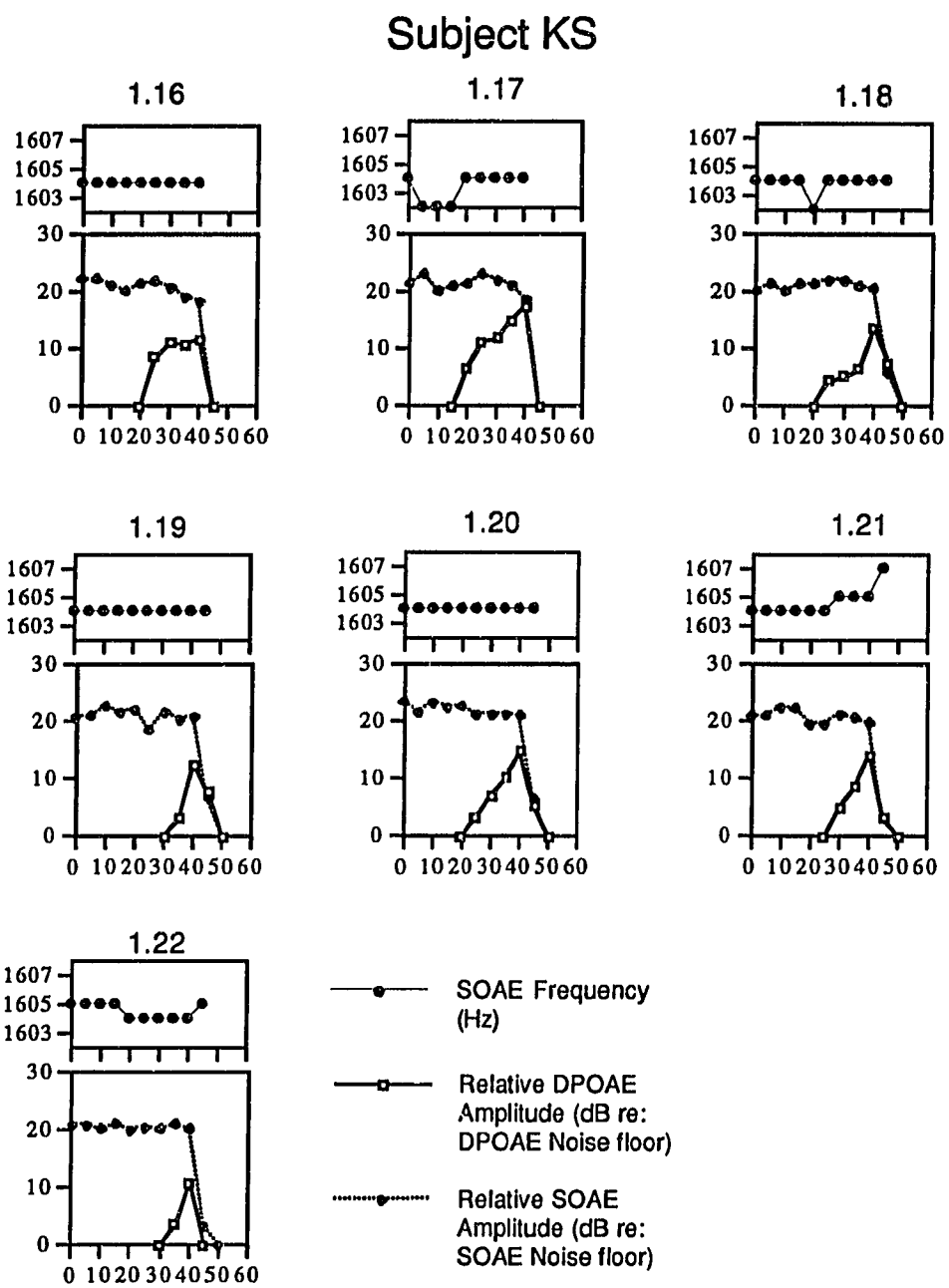

Figure 30B. Individual ratio graphs for SOAE/f1 ratios 1.16 to 1.22. X-axis for all panels is XT level (dB re: initial SOAE amplitude). Upper panels: SOAE frequency as a function of relative XT level. Lower panels: relative DPOAE and SOAE amplitudes as a function of relative XT level.

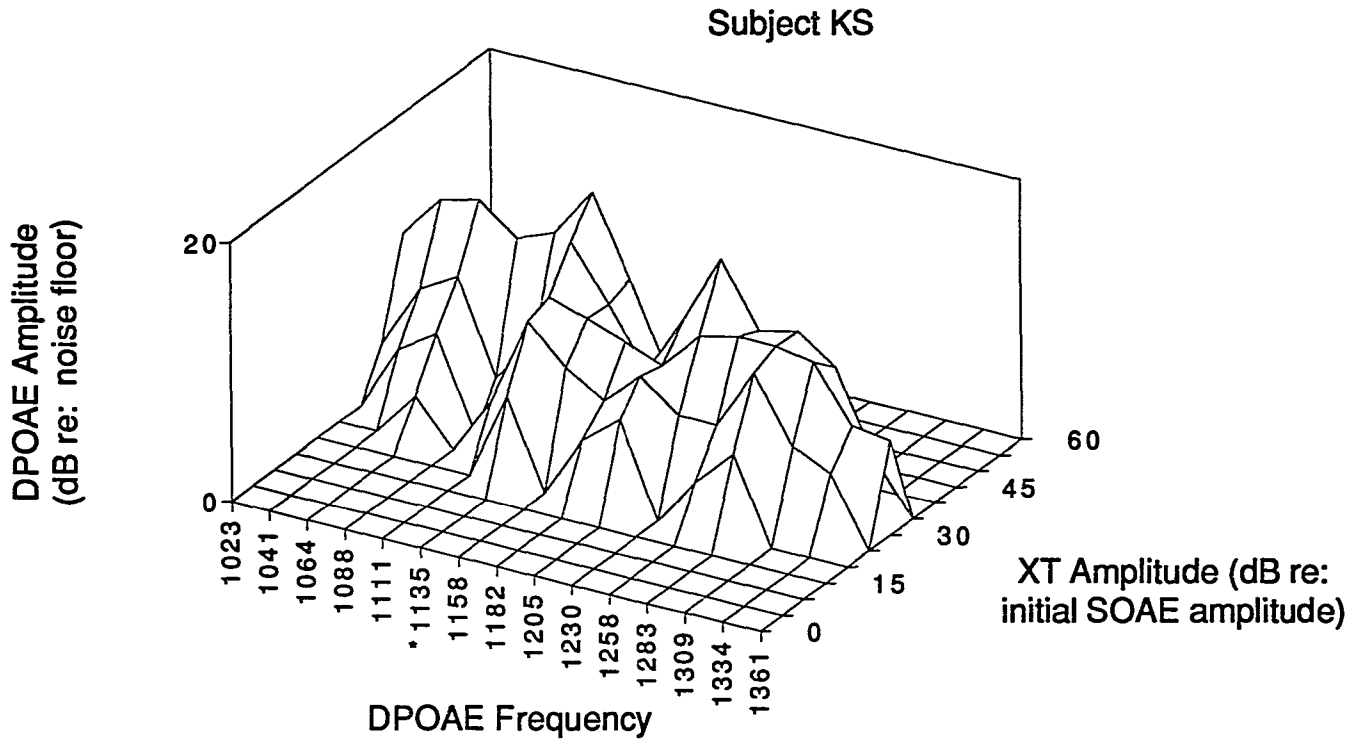

Figure 31. DPOAE amplitudes as a function of DPOAE frequency and XT amplitude. An asterisk indicates the frequency of the maximum DPOAE.

Summary

All 10 subjects exhibited DPOAEs created by a SOAE acting as f_2 and an XT as f_1 with the maximum amplitude located at a frequency approximately one-half octave lower than the frequency of the SOAE. Although the form of the SOAE and DPOAE input/output functions were similar across subjects and ratios, details of the functions were idiosyncratic. For example, DPOAEs were not observed at every ratio in every subject. Furthermore, the ratios that failed to produce DPOAEs were variable across subjects. Variability was also observed in the maxima and minima of the 3-D plots of DPOAE amplitude as a function of its own frequency and in the relative XT levels associated with DPOAE threshold and maximum. This variability was not simply related to the initial amplitude or frequency of the SOAE.

DISCUSSION

The purpose of this study was to: 1) describe distortion product otoacoustic emission (DPOAE) characteristics created via the interaction of external tones (XTs) and a spontaneous otoacoustic emission (SOAE); and 2) explore the relation of SOAE suppression to DPOAE generation. One hundred twenty-two women, between the ages of 18 and 40 years, were screened for the presence of SOAEs to obtain 10 women who qualified as subjects for this study (see selection criteria in Method section).

Prevalence of SOAEs

Prevalence estimates from this study (54% of women screened exhibited SOAEs) are in agreement with the female data reported by Bilger, Matthies, Hammel and DeMorest (1990). These authors pooled data from two SOAE surveys of adults and one survey of children ages 5 to 12 years to obtain a sample size of 75 female subjects. The prevalence estimate for the pooled data was 53%. However, the prevalence estimate from this study is lower than that provided in other studies (Burns et al., 1992; Whitehead, Kamal, Lonsbury-Martin, & Martin, 1993; Talmadge et al., 1993). These studies had sample sizes of 36 females or less and all reported prevalence estimates around 80%. Differences between studies can likely be attributed to one or more of the following: size of the sample, instrumentation used to record SOAEs, range of SOAE frequencies screened, and subject selection criteria.

As reported in previous investigations (Bilger et al., 1990; Burns et al., 1992) right ears were more likely to exhibit SOAEs than were left ears. This ear difference is not reported in all surveys (see Talmadge et al., 1993).

Frequency characteristics of the SOAEs were similar to those reported in the literature (Bright & Glattke, 1986; Burns et al., 1992; Lonsbury-Martin, Harris, Hawkings, Stagner, & Martin, 1990; Talmadge et al. 1993; Zurek, 1981; Wier, Norton & Kincaid, 1984). Most SOAEs were observed between the frequencies of 1 and 2 kHz. A likely explanation for this finding, as noted in the Introduction, is the reverse middle ear transfer function. The amplitude distributions of the SOAEs were also similar to those reported in other investigations (Burns et al., 1992; Lonsbury-Martin et al., 1990; Whitehead et al., 1993).

The Generation of DPOAEs

DPOAEs were recorded in 10 women who met the subject selection criteria. In three women, not included in this study, very few DPOAEs could be measured even though they also met the selection criteria. Differences between ears in terms of the generation of DPOAEs using two XTs have been attributed to whether or not the ear is able to produce stimulus frequency otoacoustic emissions (SFOAEs) in the areas of the DPOAE or the primary tones (Furst et al., 1988). Similarly, Burns, Strickland, Tubis, & Jones, (1984) and Jones et al. (1986) found DPOAEs, generated by two SOAEs, primarily when the DPOAE frequency corresponded to a strong SFOAE frequency or cochlear resonant enhancement. Although SFOAEs were not recorded in this study, further exploration of the impact of SFOAEs on DPOAEs generated using a SOAE and an XT is warranted and may help to explain differences between subjects.

DPOAE characteristics were highly variable across subjects and were not simply related to the initial amplitude or frequency of the SOAE. There were, however, certain trends common to all subjects. These trends consisted

of patterns of DPOAE growth, peak, rollover and disappearance into the noise floor with increasing levels of the XT. The fine structure of the DPOAE amplitude as a function of frequency, consisted of peaks and troughs with the largest amplitude DPOAEs occurring at a frequency approximately 1/2 octave lower in frequency than the SOAE. These trends will be discussed in light of what is known about DPOAEs created using two XTs.

Optimal Ratio and DPOAE Generation

The optimal SOAE/f_1 ratio (ratio associated with the largest DPOAE) varied across subjects. The mean optimal ratio, 1.16 (SD = .03), is similar to that reported by Brown & Gaskill (1990) using two XTs and a constant level of f_2 (L2) less than 40 dB SPL. These authors reported that DPOAEs were optimal at a frequency 0.552 octave below f_2, an approximate f_2/f_1 ratio of 1.19. It is not clear from their study what primary levels contributed to the optimal ratio. An optimal ratio of 1.16 is smaller than that reported for DPOAEs created by the interaction of two XTs using moderate- or high-level primaries (Bonfils et al., 1991; Gaskill & Brown, 1990; Harris et al., 1989; Lizerbram, 1993). For example, Gaskill and Brown (1990) used moderate level stimuli (50-65 dB SPL) and estimated the mean optimal ratio, calculated from 11 ears, to be 1.225 (SD = .03). In the current study the upper ratio tested was limited to 1.22 and it was the optimal ratio for only one subject. The prevalence of small optimal ratios observed in this study and in the Brown and Gaskill (1990) study, may be due to the intensity of the stimuli that were employed. Low-level tones, in contrast to high-level tones, are associated with narrower traveling wave envelopes on the basilar membrane (Johnstone, Patuzzi, & Yates, 1986). It follows that low-level primaries must be near each other in frequency to produce the interaction that

results in DPOAEs. This is in contrast to two medium- or high-level primaries that have broader traveling waves and can interact over a wider range of frequencies.

A small SOAE/f_1 ratio also could be related to processes associated with the expression of the DPOAE. A mean optimal ratio of 1.16 is compatible with theories regarding basilar membrane tuning because a ratio of 1.16 would place the DPOAE approximately one-half octave below the frequency of f_2. Allen (1990) suggested that the tectorial membrane has a low impedance at a frequency that is half an octave below f_2. If this is true, vibrational patterns (DPOAEs) could be enhanced at this frequency. Alternatively Brown and Gaskill (1990) suggested that while the level of f_2 does not exceed 40 dB SPL, the f_2 site is tuned to a frequency that is one-half octave lower than itself. That is, the amplified vibration for a particular frequency at f_2 is located in the region of a passive traveling wave peak for a frequency that is one-half octave below f_2. Either of these tuning explanations could account for a mean optimal ratio of 1.16. Further examination of a broad range of SOAE/f_1 ratios could determine the bandwidth characteristics of basilar membrane tuning at low stimulus levels.

DPOAEs were not present at every ratio for every subject. Additionally, peaks and troughs were noted in the amplitude functions as the f_1 frequency was changed. The results from each subject were peculiar to that subject. A similar fine structure of peaks and troughs was reported for DPOAEs created using two XTs (Gaskill & Brown, 1990; He & Schmiedt, 1993) and was explained by the presence of strong active stimulus frequency or spontaneous emission regions along the basilar membrane (Gaskill & Brown, 1990) or

nonuniformity in the damping factors of the DPOAE generators or OHCs (Sun, Schmiedt, He, & Lam, 1994a).

Growth and Decline of the DPOAE

DPOAE input/output functions have been found to vary depending on which primary is held constant and which one is incremented (Gaskill & Brown, 1990). In this study, the level of f_1 was incremented in 5 dB steps from an amplitude approximately equal to the initial SOAE amplitude until the SOAE and DPOAE disappeared into the noise floor. Two previous studies have used a paradigm in which f_1 is incremented and f_2 held constant (Gaskill & Brown, 1990; Brown & Gaskill, 1990). In the present study, DPOAEs appeared when the XT (f_1) was less than 40 dB SPL. Gaskill and Brown (1990) used an initial f_1 of 40 dB SPL: a level that may have been above DPOAE threshold.

The maximum DPOAEs were produced for mean XT levels between 30 and 43 dB SPL. Larger SOAE/f_1 frequency separations required higher levels of XT stimulation to reach a DPOAE maximum. The XT levels at the larger ratios are similar to what Gaskill and Brown (1990) reported using an f_2/f_1 ratio of 1.225 and a constant L2 of 25-30 dB SPL. They found maximum DPOAEs when f_1 was approximately 48 dB SPL. The maximum DPOAE amplitudes in the current study ranged from -2.8 to 5.9 dB SPL. Gaskill and Brown did not report the mean DPOAE amplitudes recorded using their lowest constant L2 stimuli; however, data from one subject plotted in their Figure 5a reveals a maximum DPOAE amplitude of approximately 7 dB SPL. They also found that the maximum DPOAE depended on the level of f_2 and frequency of the DPOAE. As was true in the present study, Gaskill and Brown noted large intersubject variability.

The mean slope of the DPOAE growth function in this study (M = .75 dB/dB, SD =.18) is very similar to that reported by Gaskill and Brown (1990) for DPOAEs created using two XTs with a stationary L2 stimulus less than 36 dB SPL (M = .88 dB/dB, SD = .13). In the present study, the decline in DPOAE level was approximately -2 dB/dB. Comparing this decline rate with data obtained from the Gaskill and Brown (1990) study suggests that two XTs can interact on the basilar membrane for a greater range of XT levels than can one XT and a SOAE. Although Gaskill and Brown (1990) did not calculate decline rates in their study, an examination of their Figure 5a reveals a decline rate of approximately -0.8 dB/dB for a constant f_2 level of 27 dB SPL (the lowest f_2 level reported in their study). One hypothesis for the differences in decline rates is that in the present study the oscillation due to the CA at f_2 ceased abruptly once the XT reached a critical level. Thus, the SOAE was suppressed into the noise floor and there was no longer a vibrational pattern at f_2 that could interact with the pattern due to f_1. In the case of two XTs, the oscillation due to the CA at f_2 may be dampened more gradually or passive basilar membrane mechanics may contribute to DPOAEs with increasing XT levels.

Optimal level differences (dB SPL difference between the SOAE and f_1 when the largest DPOAE was observed) varied with frequency ratio and ranged from a mean of 27 dB (SD = 6) at a ratio of 1.08 to 40 dB (SD = 7) at a ratio of 1.19. These level differences are difficult to interpret. Gaskill and Brown (1990) using two XTs found that the mean optimal level separation, for a ratio of 1.225, varied depending on the stationary L2 level. Their lowest (25 dB SPL) and highest (55-60 dB SPL) stationary f_2 levels were associated with an optimal level difference of 23.2 (SD = 2.0) and 5.2 dB SPL (SD = 2.7), respectively.

In the present study it is difficult to determine the internal level of the SOAE or f_2 (the stationary stimulus). It is probable that the subjects in the present investigation had SOAEs of dissimilar internal levels. This might help to explain some of the between subject variability observed in the XT levels associated with the maximum DPOAEs. SOAEs with higher internal levels would require a more intense f_1 stimulus than SOAEs with lower internal levels. Kemp (1980) and Shera and Zweig (1992) have discussed and developed models which take into account the middle ear system on reverse transmission for evoked otoacoustic emissions. Shera and Zweig assume the reverse transfer function to be the reciprocal of the forward transfer function while Kemp uses Zwislocki's (1962) acoustic analogue model of the middle ear to calculate both backward and forward middle ear transmission factors. According to Kemp's model, the backward middle ear transfer loss is 10 to 15 dB greater than that associated with forward transmission. The transfer loss for both forward and backward pressures is greater for the high frequencies than for the mid-frequencies. Further experiments are needed to determine whether or not these models can accurately predict the intracochlear energy of SOAEs at various frequencies.

For two subjects, DPOAE growth with increasing XT levels exhibited a nonmonotonicity (see ratio 1.22 of Figure 18B for JS and ratio 1.19 of Figure 20B for MK.). These nonmonotonicities occurred at XT levels of 49 dB SPL and 39 dB SPL, respectively. Patterns such as these have been reported for DPOAE growth functions using two XTs (Brown, 1987; Lonsbury-Martin et al., 1987; Popelka et al., 1993; Whitehead et al., 1992). For example, Popelka et al. (1993) reported irregularities in five out of 56 DPOAE growth functions in human subjects. The discontinuities occurred at XT levels of 40 to 60 dB SPL and were

associated with a rapid phase lag of the DPOAE. Many researchers attribute these patterns to phase cancellation between two DPOAE sources (Brown, 1987; Rosowski et al., 1984; Whitehead et al., 1992). Alternatively, they have been explained by a shift in the DPOAE fine structure peaks and troughs toward lower frequencies with increasing stimulus levels (Sun, Schmiedt, He, & Lam, 1994b). Further exploration of the origin of the notches reported in the present investigation is needed. Such explorations might include phase recordings of the DPOAE to determine if rapid phase changes are associated with the DPOAE growth function notches (Popelka et al., 1993; Whitehead et al., 1992).

There are limited data available describing DPOAEs created using low-level XTs with L2 constant and less than 40 dB SPL, and L1 varying. From the data available, similarities and differences are noted when compared to the generation of DPOAEs using a SOAE and an XT. The two paradigms are similar in that the DPOAE amplitude as a function of frequency exhibits a fine structure of peaks and troughs with the largest DPOAE amplitude occurring at a frequency approximately one-half octave lower than f_2. When plotted as a function of L1, the DPOAE amplitude function is nonmonotonic. The slope of the initial growth is less than 1.0 and the XT level associated with the maximum DPOAE (for ratios around 1.22) is approximately 45 dB SPL. Occasional irregularities have been noted in these growth functions.

Differences in DPOAE characteristics comparing the two paradigms is related to the DPOAE decline rate. The decline rate appears steeper and the DPOAE occurs over a smaller range of f_1 intensity levels in the SOAE/f_1 paradigm. These differences may be related to the constant level of the f_2 stimulus, which is difficult to equate across paradigms.

Suppression and DPOAE Characteristics

The second purpose of this study was to explore the relation of SOAE suppression to DPOAE generation. Although the XT levels associated with suppression of the SOAE varied across subjects, the following trends were observed: 1) the larger the frequency ratio the greater the XT level needed to suppress the SOAE; 2) the threshold of the DPOAE occurred with very little SOAE suppression whereas the maximum DPOAE was associated with a greater amount of SOAE suppression; and 3) the disappearance of the SOAE into the noise floor was most often accompanied by the disappearance of the DPOAE into the noise floor. These trends are unique to the case where DPOAEs are created using a SOAE and one XT and cannot be compared to those generated using two XTs.

DPOAE Generation

Suppression of an SOAE appeared to play an important role in DPOAE generation. In general, the SOAE amplitude was slightly reduced when the DPOAE first appeared while it was significantly reduced when the DPOAE reached its maximum value. Suppression of the SOAE was necessary but not sufficient for the generation of observable DPOAEs. DPOAEs were not observed at all ratios even though the suppression of the SOAE appeared similar to the suppression at ratios that were associated with DPOAEs. As previously mentioned, it is important to consider not only the characteristics of the DPOAE generating mechanism (e.g. the amount of f_1 and SOAE overlap on the basilar membrane, or suppression of the f_2 CA) but also the characteristics of the basilar membrane that influence expression of the DPOAE. For example,

a weak or absent SFOAE at a particular frequency could result in absent DPOAEs at that frequency.

SOAE Frequency and Amplitude Shifts

Initial frequencies and amplitudes of SOAEs shifted between test sessions. SOAE frequencies and amplitudes, without XT stimulation, also shifted during a test session. In addition, the frequencies often shifted in the presence of XT stimulation. Previous studies have reported on SOAE frequency shifts (Bright, 1985; Burns et al., 1984; Frick & Matthies, 1988; Kohler, Fredriksen, & Fritze, 1986; Rabinowitz & Widin, 1984; Whitehead, 1991; Wier et al., 1984; Zurek, 1981) and amplitude variability (Bright & Glattke, 1984; Long et al., 1991; Long, Tubis, & Jones, 1993; Rabinowitz & Widin, 1984; Whitehead, 1991). Zurek (1981) reported that his SOAE at 1910 Hz decreased to 1900 Hz over the course of 20-30 minutes of recording. He explored two potential causes of this shift: a slow buildup of static pressure with the probe inserted in the ear canal, and a warm-up effect or change in the characteristics of the microphone. Zurek concluded that neither of these could adequately account for the frequency shift of his SOAE.

Previous investigations have found that changing middle ear stiffness can influence SOAE frequency and amplitude (Mott, Norton, Neely, & Warr, 1989; Rabinowitz & Widin, 1984; Schloth & Zwicker, 1983). Schloth and Zwicker (1983) showed that activation of the acoustic reflex or a change in ear canal air pressure increases SOAE frequency and decreases its amplitude. However, in this study frequency and amplitude shifts occurred in both directions. Furthermore, two subjects demonstrated a systematic increase in the amplitude of their SOAE from the beginning to the end of the test session

without a corresponding shift in frequency. It is therefore unlikely that increases in middle ear stiffness could account for all of these shifts.

Frequency shifts have been noted during experiments that suppress an SOAE with an XT (Long et al., 1991; Long et al., 1993; Rabinowitz & Widin, 1984; Zurek, 1981). Observations have been described in terms of frequency "pulling" and "pushing" (see Introduction). In the present investigation, an upward shift in the frequency of the SOAE was often noted just prior to its disappearance into the noise floor (frequency pushing). Other frequency shifts, both upward and downward appeared small and unsystematic. Although Rabinowitz and Widin (1984) noted that suppressors more than 20 Hz lower in frequency than the SOAE were associated with small frequency changes in the SOAE, they did not report any systematic changes for this condition. A change in SOAE frequency and amplitude may be due to a shift in the outer hair cells that predominantly contribute to the SOAE or re-tuning of the same hair cells via a local change in the stiffness of the cochlear partition (McFadden & Pasanen, 1994). Middle ear mechanisms could also contribute to SOAE shifts. Several studies have explored the origin of these shifts using models of the cochlea. Modelling SOAEs as noise-perturbed, limit-cycle oscillators with active, nonlinear stiffness components has recently been demonstrated to account for outcomes that include positive, negative and no correlations between amplitude and frequency fluctuations of SOAEs (van Dijk, Wit, Tubis, Talmadge, & Long, 1994). Alternatively, a third-order oscillator model with a time delay of the feedback force can also account for these correlations (Wit, 1992). Whether or not these models can account for systematic frequency "pulling" or "pushing" in the presence of XT stimulation awaits further research.

Whitehead (1991) explored the origin of SOAE frequency and amplitude variations using human subjects. His study involved various conditions that included recording SOAEs 1) in the presence of pure tone stimulation less than 75 dB SPL, 2) in quiet with the probe in the ear canal, 3) after placing the probe in the non-SOAE ear for a period of 20-30 minutes, and 4) after subjects sat quietly in a sound-proofed room for 30 minutes without the probe in the ear canal. The last condition was the only one that did not result in systematic frequency and amplitude shifts. Whitehead concluded that a central nervous system influence on the emission generator produced these shifts. He postulated that an increase in arousal or of auditory selective attention when the probe is in either ear canal could account for systematic frequency shifts.

The medial olivocochlear system (MOC) is one mechanism which could mediate a central involvement on SOAE shifts. The MOC sends efferent projections primarily and directly to the OHCs (Warr & Guinan, 1979). Acoustical stimulation of the MOC has been shown to be associated with an increase in SOAE frequency and variability in its amplitude (Harrison & Burns, 1993; Long, 1989; Mott et al., 1989).

Appearance of New SOAEs

In four of the subjects in this study, new SOAEs appeared during the course of the recording session while in three subjects and one woman who was rejected from the study, new SOAEs appeared at the beginning of the test session that were not apparent during the screening session. Other investigators have also noted the appearance of new SOAEs across test sessions (Kohler & Fritze, 1992) and during recording sessions (Frick & Matthies, 1988; Whitehead, 1991; Long et al., 1993). A tradeoff of amplitude

among SOAEs (Burns et al., 1984; McFadden & Pasanen, 1994) has also been reported. To explain findings such as these, McFadden & Pasanen suggested that there is a "fixed, common pool of energy against which the various SOAEs in a particular frequency region draw, and any agent that acts to diminish one SOAE provides others with the opportunity to draw more energy and increase in level" (p. 3471). The authors also suggested that new SOAEs appear once established SOAEs are no longer able to draw upon the fixed energy pool.

SOAEs, located near the frequency of an expected DPOAE and created using two XTs, can have an impact on DPOAE characteristics such as amplitude and threshold (Cianfrone et al., 1990; Furst et al., 1988; Kemp, 1979; Moulin, Collet & Morgon, 1992; Wier et al., 1988; Wilson, 1980). Cianfrone et al. (1990) studied one ear with a SOAE of 2288 Hz and 6 to 7 dB SPL. These authors found that lower level primaries were needed to generate DPOAEs if they were 28 or 78 Hz away from a SOAE. Wier et al. (1988) found in four subjects that a DPOAE was 10-25 dB larger if it was within 100 Hz of a SOAE. SOAEs ranged in amplitude from 7 to 15 dB SPL. They also found that the DPOAE enhancement was present even when the SOAE was reduced into the noise floor by aspirin ingestion. They suggested that aspects of the mechanism that generate SOAEs remains active in the absence of an SOAE. In the present study the new SOAEs appeared to have little or no impact on the DPOAE characteristics. Three of the new SOAEs were higher in frequency than the target SOAE (see subjects KM, AR, and JV). Two of the new SOAEs appeared to have no impact on the DPOAEs when they were within 72 Hz of it (see ratio 1.10 for subject JV and 1.08 for subject BR). Four new SOAEs were associated with DPOAE thresholds approximately 5 dB lower than the surrounding ratios

(see ratio 1.12 for subject JS, 1.13 for subject IR, 1.13 and 1.15 for subject LH). In addition, subject LH's optimal ratio was 1.15, a ratio at which the DPOAE frequency (3164 Hz) was within 23 Hz of a new low-level SOAE. However, the maximum DPOAE amplitude at this ratio was only approximately 2.5 dB higher than that obtained at a ratio of 1.18 (the DPOAE associated with a ratio of 1.18 was not within 100 Hz of a new SOAE). It is difficult to determine from the present study whether these slightly lower thresholds and LH's optimal ratio at 1.15 are due to the presence of new low-level SOAEs. Further exploration regarding the impact of low-level SOAEs on DPOAE characteristics is needed.

A Nonquantitative Cochlear Model

Zwicker (1986) proposed a cochlear model in which suppression of the primaries was important in generating DPOAEs. According to Zwicker (1986), there are two methods of reducing the gain in the feedback loop of a cochlear amplifier. One method is increasing the energy of a particular tone (self-suppression). As the energy increases, the CA saturates and behaves like a passive linear system. The other method is to apply energy to a tone of a different frequency. This can also saturate the feedback amplifier. This latter method was used in the present study to saturate the CAs associated with SOAEs.

A schematic of a positive feedback amplifier, modified from Yates (1990) (Figure 32) serves to explain the present data. The model shows two inputs to the feedback amplifier: the output of an oscillator (an SOAE) and an external tone (f_1). The feedback loop consists of a nonlinear term most likely associated with the OHCs and a frequency selective gain term that depends on the coupling between the organ of Corti and the tectorial membrane. There is an

inverse relationship between the nonlinear term and the frequency selective gain term (the greater the input to the nonlinear term the lower the gain and vice versa). Only those frequencies that are passed by the frequency selective gain term are added to the input at the summing junction. In the example of Figure 32, the SOAE is generated by the oscillator. Its frequency is similar to the CF of the filter (2 kHz) and the SOAE is therefore amplified. In this study, the SOAE was suppressed by adding an XT (f_1) to the ear at various frequencies and levels. The frequency of f_1 remained outside the passband of the frequency selective gain term (e.g. 1.6 kHz) and therefore, was not amplified. F_1 did, however, interact nonlinearly with the SOAE. This interaction resulted in DPOAEs and a larger input to the nonlinear term. A larger input to the nonlinear term reduced the gain term and consequently the amplitude of the SOAE was also reduced. Total saturation of the nonlinear term opened the feedback loop and caused total suppression of the SOAE. Without an SOAE, the XT no longer had a vibrational pattern to interact with and the DPOAEs disappeared.

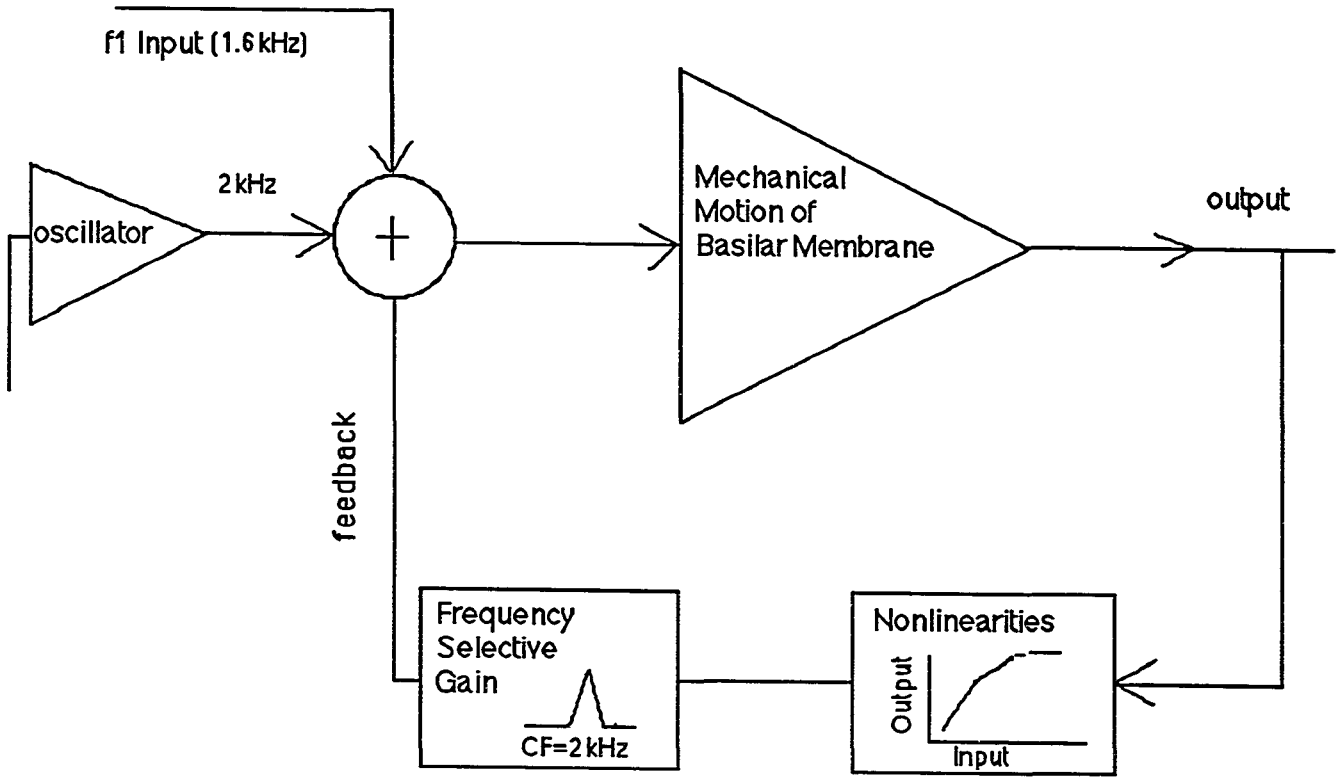

Figure 32. Schematic of electrical circuit model of a cochlear amplifier with inputs from an oscillator (SOAE) and an XT (f1), (modified from Yates, 1990). The circuit includes an amplifier and a feedback path consisting of nonlinear and frequency selective gain terms. See text for more details.

Pictorial representations of the SOAE, XT, and DPOAE vibrational patterns on the basilar membrane are provided in Figure 33 (modified from Yates, Geisler, Patuzzi, & Johnstone, 1989). This figure is intended to provide insight into possible micromechanical processes within the cochlea. The first DPOAEs appeared at low XT levels and were a result of initial saturation of some of the OHCs comprising the feedback loop of the CA (Figure 33A). Saturation of some of the OHCs reduced the amplifier gain by eliminating some of the active mechanisms that are thought to increase basilar membrane displacement. The maximum amplitude DPOAEs appeared when XT levels were approximately 30 to 40 dB SPL. These XT levels, in contrast to lower levels depicted in Figure 33A, were associated with greater overlap of the two tones on the basilar membrane. Therefore, a larger number of OHCs were saturated causing the gain of the amplifier to decrease even further (Figure 33B). Further increases in XT level saturated all OHCs of the SOAE, the feedback loop of the CA opened and the SOAE, along with the DPOAE, disappeared rapidly into the noise floor (Figure 33C).

These representations may also explain some of the SOAE shifts to higher frequencies observed prior to total suppression of the SOAE into the noise floor. The last of the OHCs to be suppressed were the basal OHCs that contributed to the SOAE (Figure 33B).

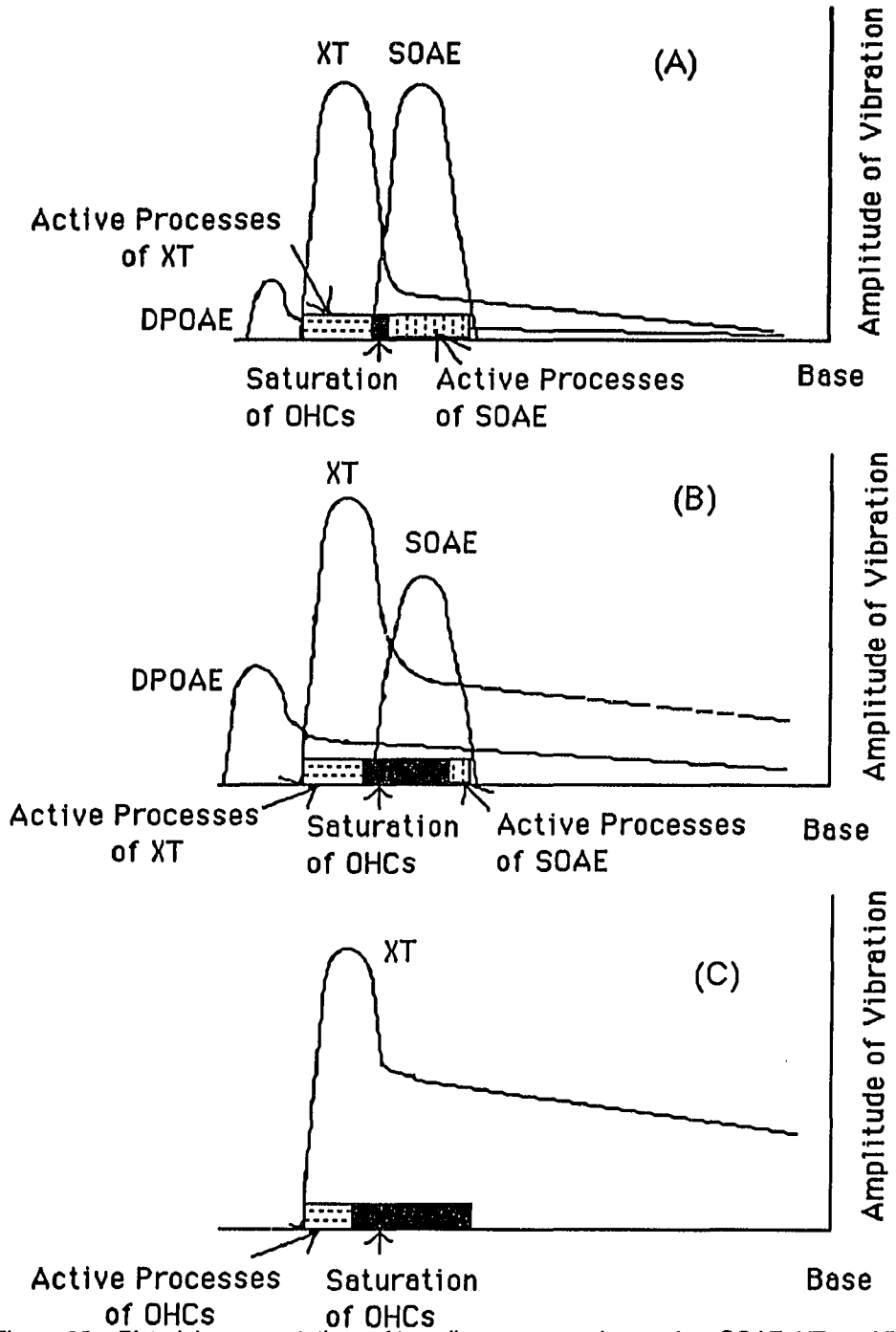

Figure 33. Pictorial representations of traveling wave envelopes of an SOAE, XT, and DPOAE on the basilar membrane (modified from Yates et al., 1989). See text for explanation.

Individual variability can be attributed to different gain settings of the CA associated with each subject's SOAE or by differences in the nonlinear term. Also, some SOAEs may have reflected CA operation nearer to saturation than others. Those SOAEs close to complete saturation required very little XT stimulation before DPOAEs were observed. The differences in saturation points might be influenced by the MOC efferent system which has direct connections to the OHCs.

Analysis of the data revealed that suppression of the SOAE (saturation of the feedback loop of the CA by inference) was not always sufficient in the generation of observable DPOAEs. This suggests that the filter characteristics of the basilar membrane (see Brown & Gaskill, 1990) and/or separate SFOAEs associated with each DPOAE (see Kemp & Brown, 1983) should be considered as potential contributor(s) to the observed amplitudes of DPOAEs.

Concluding Remarks

Until recently, the micromechanics of the cochlea have been difficult to study noninvasively. A noninvasive technique is critical because of the cochlea's vulnerability to mechanical insults. The discovery of otoacoustic emissions has made it possible to explore the micromechanics in living and healthy cochleae. Available evidence suggests that the micromechanics of the cochlea behave differently at high and low stimulus levels. Active and passive elements are believed to contribute to basilar membrane motion patterns at low and high stimulus levels, respectively. This study measured DPOAEs created by the interaction of a SOAE and an XT less than 60 dB SPL. Because low to medium stimulus levels were employed, the DPOAE characteristics primarily reflect active cochlear micromechanics.

The results indicated intersubject variability and complicated patterns of DPOAE growth and SOAE suppression with increasing levels of the XT. Individual variability in these patterns is likely a result of unique active elements that contribute to each CA or variability in the efferent control system.

Despite the variability between subjects, certain trends were noted. For example, the optimal ratio in all subjects was approximately one-half octave lower in frequency than the SOAE. This is consistent with suggestions that the micromechanical elements of the cochlea filter the DPOAEs when low to medium stimulus levels are employed (Allen & Neely, 1992). This finding suggests the need for further exploration of the bandwidth characteristics of this filter.

With increasing levels of the XT, a steep DPOAE decline, that often paralleled the decline of the SOAE, was observed. Such a steep decline has not been reported in studies that have employed two low-level XTs as primaries. Although further exploration of the DPOAE decline associated with two XTs is needed, the observations suggest that there are differences in the mechanisms that generate DPOAEs when two XTs are used as primaries compared to one XT and one SOAE.

Many questions remain unanswered and make it difficult to interpret similarities and differences between the two paradigms (two XTs versus one XT and one SOAE). For example, 1) What is the internal energy of a SOAE? 2) Is the Bekesy traveling wave envelope for an XT similar to that for a SOAE? 3) What is the source of the intersubject variability? These questions and others regarding the nature of SOAE and DPOAE generation await further acoustic exploration and cochlear modeling.

APPENDIX A

Human Subjects Consent Form

Subject's Consent

The creation of distortion products through the interaction of spontaneous otoacoustic emissions and externally generated tones

I AM BEING ASKED TO READ THE FOLLOWING MATERIAL TO ENSURE THAT I AM INFORMED OF THE NATURE OF THIS RESEARCH STUDY AND OF HOW I WILL PARTICIPATE IN IT, IF I CONSENT TO DO SO. SIGNING THIS FORM WILL INDICATE THAT I HAVE BEEN SO INFORMED AND THAT I GIVE MY CONSENT. FEDERAL REGULATIONS REQUIRE WRITTEN INFORMED CONSENT PRIOR TO PARTICIPATION IN THIS RESEARCH STUDY SO THAT I CAN KNOW THE NATURE AND THE RISKS OF MY PARTICIPATION AND CAN DECIDE TO PARTICIPATE OR NOT PARTICIPATE IN A FREE AND INFORMED MANNER.

PURPOSE

It is known that we can detect very soft sounds in your outer ear canal by placing a miniature microphone near the opening of the ear. The purpose of this study is to obtain records of sounds that are produced when no sound is being presented to your ear and when one or more sounds are led to the same ear. These low level sounds are produced within your inner ear and are called otoacoustic emissions.

SELECTION CRITERIA

I am being invited to participate because:
1. I have normal hearing sensitivity
2. I do not have a family history of hearing loss
3. I have not been exposed to loud noises for prolonged periods of time
4. I have not taken drugs which are ototoxic
5. I do not have a history of middle ear infections.

PROCEDURE

If I agree to participate, I will be asked to agree to the following:

I will be given a brief hearing test. Following the test, I will be asked to recline in a comfortable chair and a microphone assembly with a soft rubber tip will be placed in one of my ear canals. Next, a variety of sounds of various frequencies will be presented to my ear. None of the sounds are intense enough to cause harm to my ear.

PARTICIPATION COSTS AND SUBJECT COMPENSATION

The entire procedure will require about 8 hours of my time (four separate sessions, 2 hours per session). Other than my time, there are no costs to me for my participation. I will be paid $5 dollars per hour for participating in this study.

RISKS

Although there is no known danger associated with this study, there are no funds available to compensate me for lost time or wages or pay for any medical or hospital services that I might require as a result of my participation in this study.

CONFIDENTIALITY

All records will be kept strictly confidential. My name will be known only to the investigator who signs this form. My medical information, hearing test results, and results of this study will be stored only with a numerical code that cannot be traced to me personally.

I am free to ask questions at any time. The investigator who signs this form may be reached at 621-7065 (office). If I have questions concerning my rights as a research subject, I may call the Human Subjects Committee office at 626-6721.

BEFORE GIVING MY CONSENT BY SIGNING THIS FORM, THE METHODS, INCONVENIENCES, RISKS, AND BENEFITS HAVE BEEN EXPLAINED TO ME AND MY QUESTIONS HAVE BEEN ANSWERED. I UNDERSTAND THAT I MAY ASK QUESTIONS AT ANY TIME AND THAT I AM FREE TO WITHDRAW FROM THE PROJECT AT ANY TIME WITHOUT CAUSING BAD FEELINGS. MY PARTICIPATION IN THIS PROJECT MAY BE ENDED BY THE INVESTIGATOR FOR REASONS THAT WOULD BE EXPLAINED. NEW INFORMATION DEVELOPED DURING THE COURSE OF THIS STUDY WHICH MAY AFFECT MY WILLINGNESS TO CONTINUE IN THIS RESEARCH PROJECT WILL BE GIVEN TO ME AS IT BECOMES AVAILABLE. I UNDERSTAND THAT THIS CONSENT FORM WILL BE FILED IN AN AREA DESIGNATED BY THE HUMAN SUBJECTS COMMITTEE WITH ACCESS RESTRICTED TO THE PRINCIPAL INVESTIGATOR, LINDA E. WEILAND, OR AUTHORIZED REPRESENTATIVE OF THE SPEECH & HEARING SCIENCES DEPARTMENT. I UNDERSTAND THAT I DO NOT GIVE UP ANY OF MY LEGAL RIGHTS BY SIGNING THIS FORM. A COPY OF THIS SIGNED CONSENT FORM WILL BE GIVEN TO ME.

Subject's signature	Date
Parent/Legal Guardian (If necessary)	Date

INVESTIGATOR'S AFFIDAVIT

I have carefully explained to the subject the nature of the above project. I hereby certify that to the best of my knowledge the person who is signing this consent form understands clearly the nature, demands, benefits, and risks involved in his/her participation and his/her signature is legally valid. A medical problem or language or educational barrier has not precluded this understanding.

Signature of Investigator	Date

APPENDIX B

Case History Information

Case History Information

Date _____

Identifying Information

Name _____ D.O.B. _____ M _____F_____

Phone # _____

PART I

PLEASE CHECK IF YOU HAVE HAD OR PRESENTLY HAVE ANY OF THE FOLLOWING MEDICAL PROBLEMS:

_____Meningitis
_____Muscle Disorder
_____Temporal Bone Fracture
_____Measles
_____Stroke
_____Autoimmune Disease
_____Meniere's Disease
_____Diabetes Mellitus
_____Otosclerosis
_____Thyroid Problems
_____Kidney Disease
_____Multiple Sclerosis
_____Hypertension
_____Syphilis
_____Sudden Hearing Loss
_____Mastoiditis

PART II

PLEASE CHECK IF YOU HAVE HAD OR PRESENTLY HAVE ANY OF THE FOLLOWING MEDICAL PROBLEMS. IF YOU HAVE OR HAVE HAD ANY OF THE FOLLOWING, PLEASE CHECK THE "YES" OR "NO" ANSWER FOR EACH ITEM.

_____Mumps; Did mumps result in hearing loss? _____yes _____no

_____Cancer; Were you treated with radiation to the ear? _____yes _____no

Did you receive Cis-Platinum? _____yes _____no

_____Influenza; Did influenza result in hearing loss? _____yes _____no

_____Heart Disease; Have you had open heart surgery? _____yes _____no

Have you ever taken loop diuretics such as furosemide or ethacrnynic acid?

_____yes _____no

_____Have you ever been treated with Quinine? _____yes _____no

Have you ever experienced acoustic trauma, e.g., from an explosion or gunshot? Symptoms would include rupture or laceration of the middle ear and/or inner ear structures? _____yes _____no

Have you had an ear infection in the last three months? _____yes _____no

Have you ever been treated with ototoxic drugs such as kanamycin, gentamycin, or streptomycin? _____yes _____no

PART III

OTHER CASE HISTORY INFORMATION

PLEASE CHECK IF YOU HAVE HAD OR PRESENTLY HAVE ANY OF THE FOLLOWING:

_____Chronic Headaches
_____Dizziness
_____Noises in your head or ears
_____Family History of ear disease
_____Noise exposure. Please describe situations in which you were exposed to loud noises (e.g. gunfire, concerts, loud machinery)

_____

_____

_____

_____Ear Infections. Please list # of infections and your approximate age;

_____

_____

_____Ear surgery (including pressure equalization tubes). Please list the # and types of surgeries and your approximate age; _____

_____

_____

_____ Current medications; please list_____

_____

_____

_____

_____ Use of aspirin over the last month; please indicate the average # of aspirin per day and the # of days of use_____

_____

_____

_____

Criteria to be used for subject selection purposes:

1. PART I. If any condition is checked, the subject will be excluded from the study.

2. PART II. If any question is answered with a "yes", the subject will be excluded from the study.

3. PART III. This information will be used to obtain additional subject information but will not be part of the exculsionary criteria.

APPENDIX C

Questionnaire Used to Obtain Subject Information

Questionnaire Used to Obtain Subject Information

Name _____D.O.B._____ Age_____

DATE OF SESSION 1 _____

Over the past week I have had, been, or have taken: If yes, please explain

_____middle ear infections. _____

_____ear surgery. _____

_____exposure to loud music. _____

_____exposure to gunfire. _____

_____exposure to other loud noises. _____

_____aspirin. _____

_____ hospitalized. _____

_____none of the above.

DATE OF SESSION 2 _____

Since Session 1 testing, conducted on _____, I have had, been, or have taken:

_____middle ear infections. _____

_____ear surgery. _____

_____exposure to loud music. _____

_____exposure to gunfire. _____

_____exposure to other loud noises. _____

_____aspirin. _____

_____ hospitalized. _____

_____none of the above.

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Human Subjects Committee

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November 9, 1993

Linda E. Weiland, M.A.
Theodore Glattke, Ph.D.
Department of Speech/Hearing Sciences
Old BPA Building, Room 226
Main Campus

RE: **HSC A93.100** THE CREATION OF DISTORTION PRODUCTS THROUGH THE INTERACTION OF SPONTANEOUS OTOACOUSTIC EMISSIONS AND EXTERNALLY GENERATED TONES

Dear Investigators:

We received your above-cited research proposal. The procedures to be followed in this study pose no more than minimal risk to participating subjects. Regulations issued by the U.S. Department of Health and Human Services [45 CFR Part 46.110(b)] authorize approval of this type project through the expedited review procedures, with the condition(s) that subjects' anonymity be maintained. Although full Committee review is not required, a brief summary of the project procedures is submitted to the Committee for their endorsement and/or comment, if any, after administrative approval is granted. This project is approved effective 9 November 1993 for a period of one year.

The Human Subjects Committee (Institutional Review Board) of the University of Arizona has a current assurance of compliance, number M-1233, which is on file with the Department of Health and Human Services and covers this activity.

Approval is granted with the understanding that no further changes or additions will be made either to the procedures followed or to the consent form(s) used (copies of which we have on file) without the knowledge and approval of the Human Subjects Committee and your College or Departmental Review Committee. Any research related physical or psychological harm to any subject must also be reported to each committee.

A university policy requires that all signed subject consent forms be kept in a permanent file in an area designated for that purpose by the Department Head or comparable authority. This will assure their accessibility in the event that university officials require the information and the principal investigator is unavailable for some reason.

Sincerely yours,

William F. Denny, M.D.
Chairman, Human Subjects Committee

cc: Departmental/College Review Committee