Chapter 2
Morphological and Functional Ear Development

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The aim of argument, or of discussion, should not be victory, but progress.
—Joseph Joubert

1 Introduction

The development of peripheral auditory function in humans has been observed and documented using a variety of investigative tools. Because these tools must all be noninvasive in nature, they are indirect and, therefore, somewhat imprecise probes of function. Measured function at one level of the peripheral auditory system is undoubtedly influenced by the functional status of other parts of the system. Thus, the most effective way to define and document the physiology and developmental course of the human auditory system is to consider and integrate findings, with an acute awareness of the limitations of each assay and the relationship among results. To make this treatise a reasonable endeavor, only human auditory developmental data are presented and discussed, although when it elucidates a pattern common to humans, mammalian development in general may be considered.

Section 2 of the chapter deals with the most peripheral segments of the auditory system, the outer and middle ear, and with acoustical measurements of the functioning...
of the ear canal and middle ear in adults and infants. Section 3 examines what is known about the development of human cochlear function focusing on otoacoustic emission (OAE) measurements in human adults and infants. Following these descriptions of functional development, Sect. 4 attempts to interpret the findings and consider their possible sources, speculating, when possible, about the implications for development of hearing. The authors of this chapter sometimes have very different views on the sources of maturational differences in the described data. To the extent that readers become informed on diverse viewpoints concerning current issues in the field, this may be of some benefit. The diverse viewpoints presented here should encourage readers to access additional literature en route to formulating their own well-reasoned opinions.

2 Development of External- and Middle-Ear Responses

2.1 Maturational Changes in Anatomy

The anatomy of the human ear canal continues to mature after full-term birth. The canal is straighter and shorter in infants than in adults (Northern and Downs 1984), for example, a length of 1.68 cm in newborns, which is roughly two thirds of that in adults (Crelin 1973). The canal wall of the newborn has no bony portion and consists of a thin, compliant layer of cartilage (Anson and Donaldson 1981), and the tympanic ring does not completely develop until age 2 years (Saunders et al. 1983). The adult canal has a bony wall in its inner two thirds and soft-tissue wall in the outer one third, whereas the canal in newborns is almost completely surrounded by soft tissue (McLellan and Webb 1957). The ear-canal diameter and length each increases from birth through the oldest test age of 24 months (Keefe et al. 1993). Human temporal bone data show maturational changes in the middle ear during childhood. The tympanic-membrane plane relative to the central axis of the ear canal is more horizontal in the newborn, with a more adult-like orientation by age 3 years (Ikui et al. 1997).

The tympanic membrane develops embryologically as a structure composed of ectodermal, mesodermal, and endodermal layers. The outer layer is similar to the epidermis of skin; the lamina propria is composed of a matrix and two layers of type II collagen fibers, and the thin inner lamina mucosal layer with columnar cells forms a boundary of the tympanic cavity (Lim 1970). The collagen fibers of the mucosal layer provide mechanical stiffness to the tympanic membrane (Qi et al. 2008). The thickness of the adult tympanic membrane has broad variability across its surface as well as across subjects; it ranges from 0.04 to 0.12 mm in a central region of the pars tensa (Kuypers et al. 2006). The tympanic membrane in newborns is thicker than in adults, with a thickness ranging from 0.4 to 0.7 mm in the posterior–superior region, 0.7–1.5 mm in the umbo region, and 0.1–0.25 mm in the posterior–inferior, anterior–superior, and anterior–inferior regions (Ruah et al. 1991). Qi et al. (2008) confirmed that the measured thickness of the tympanic membrane in the temporal bone of a 22-day-old infant was in the range of the Ruah et al. data.
The volume of the middle-ear cavity increases postnatally until the late teenage years (Eby and Nadol 1986); this growth may change the ossicular orientation and influence the mechanical functioning of the middle ear. A smaller middle-ear cavity volume also increases middle-ear stiffness at an ear-canal location just in front of the tympanic membrane, because the membrane motion drives against the volume compliance. The volume of the middle-ear cavity includes contributions from the tympanic cavity, the aditus ad antrum, the mastoid antrum, and mastoid air cells. The tympanic-cavity volume is approximately 640 mm$^3$ in adults and 452 mm$^3$ in 3-month-old infants (Ikui et al. 2000), and approximately 330 mm$^3$ in a 22-day-old. The mastoid process begins to develop approximately 1 year after birth. The ossicles of the middle ear are completely formed around the sixth month of fetal life (Crelin 1973) and the middle-ear muscles are then fully developed (Saunders et al. 1983). The distance between the stapes footplate and the tympanic membrane in adults is larger than that of infants through 6 postnatal months (Eby and Nadol 1986). Dimensions of ear-canal and tympanic-membrane components in the adult and newborn ear (with the latter based on temporal-bone data from a 22-day-old) are tabulated in Qi et al. (2006).

### 2.2 Maturational Changes in Function

#### 2.2.1 Diffuse-Field Effects

Under free-field listening conditions in the adult human ear, the transfer-function level between sound pressure at the ear drum relative to a location near the opening of the ear canal is close to 0 dB at low frequencies, and has a resonant boost in pressure near 2.7 kHz. This boost is accompanied by a more broadly tuned concha resonance near 4.5–5 kHz that leads to an overall gain between 2 and 7 kHz in the adult ear (Shaw and Teranishi 1968). The ear-canal resonance frequency near 3.0 kHz in children ages 2–12 years remains slightly higher than adult values (Dempster and Mackenzie 1990). Thus, maturation of ear-canal function extends at least to the onset of puberty.

The diffuse-field absorption cross-section $A_D$ quantifies the ability of the external ear to collect sound power from free-field sources and of the middle ear to absorb this power (Rosowski et al. 1988; Shaw 1988). For a sound source sufficiently far from a listener, the sound field in a room approximates a diffuse field averaging out the directional properties of sound. Neglecting power loss at the canal walls, $A_D$ is the ratio of the sound power absorbed by the middle ear relative to the diffuse-field power density in the room. It has units of area; its level is defined as $10 \log_{10} A_D$ with 0 dB at 1 cm$^2$.

In measurements on adults and in infants of age 1–24 months, this transfer function increased with increasing frequency at approximately 6 dB per octave at low frequencies up to a maximum level at a resonance frequency that varied with age (see Fig. 2.1). The frequency of maximum response increased with decreasing age from 2.5 kHz in adults to 5 kHz in 1-month-olds. $A_D$ was larger in adults than in
infants, and decreased by approximately 10 dB with increasing age (Keefe et al. 1994). Thus, external and middle ears are more efficient at collecting sound energy in adults compared to infants. In relative terms across frequency, the efficiency of infant ears is maximal at a higher frequency than in adults. This is partially explained by maturational growth in the cross-sectional area of the ear canal area. A larger area leads to more efficiency in collecting and absorbing sound power, and thus a larger $A_d$. Because ear-canal area increases with age, the ability of the ear to absorb sound power improves with increasing age, whether sound is presented in the free field or via some type of coupler measurement.

### 2.2.2 Maturational Factors of Ear-Canal Acoustics

Many hearing experiments and clinical measurements are performed using a probe microphone and sound source in a probe inserted in a leak-free manner into the ear canal. Such a procedure removes acoustic influences of the outer ear and torso, and thus all directional hearing cues. The only remaining outer-ear effect is ear-canal acoustics, which vary with age.

Using median measurements of the ear canal area for infants ($A_i$) and for adults ($A_o=58 \text{ mm}^2$) (Keefe and Abdala 2007), the distributions of the area measured in groups of infants were normalized to the median adult area in terms of the relative area level (in dB) by $\Delta L_a = 20 \log_{10} (A_i/A)$. 

**Fig. 2.1** The mean diffuse-field absorption cross-section $A_d$ is plotted as a function of frequency for adults and infants in the listed age groups [This figure was originally published in Keefe et al. (1994)]
Morphological and Functional Ear Development

These are plotted in the top panel of Fig. 2.2, with outlier responses shown by asterisks. The median of the relative area level was 17 dB larger for term infants than for adults, and decreased to 5 dB at 6 months.

The median ear-canal length based on Keefe and Abdala (2007) increased with increasing age from newborns to adults (bottom panel of Fig. 2.2). Negative length values in the tails of the infant distributions result from small errors in the estimates. A regression line for ear-canal length across the infant age groups shows that length increased from 0.3 cm to 1.1 cm with increasing age to 6 months. This contrasts with the 2-cm length in adult canals between the probe and the tympanic membrane. These length estimates correspond to the geometric length minus the insertion depth of the probe assembly.

The relatively large interquartile ranges of ear-canal length and area in Fig. 2.2 show the critical importance of individual variability at each age. Measurements at ages up to 24 months show that neither the ear-canal length nor the area is mature at 24 months (Keefe et al. 1993), and it is likely ear-canal growth continues at a slower rate until the onset of puberty. From the standpoint of air-conducted sound, the ear canal acts as a filter whose magnitude and phase characteristics change ever more...
slowly from infancy through adulthood. The frequency response of the filter influences the representation of sound encoded neurally, which would affect subjective properties such as loudness and timbre.

2.2.3 Absorbance in the Ear Canal

Aural absorbance is a dimensionless acoustic transfer function that assesses how efficiently the middle ear, and the ear canal to the extent that any wall loss is present, absorbs power (Feeney and Keefe 2010). It has particular relevance for hearing experiments and clinical measurements performed using a sound source within the ear canal. Absorbance varies between 0 and 1, with a value of 1 when the ear absorbs all the power from the forward-traveling sound in the ear canal, and a value of 0 when the ear absorbs no power (so that all the power is reflected at the tympanic membrane). The mean absorbance has a maximum in the range from 2 to 4 kHz for adults and infants of age 1.5 months and older. In term newborns, the peak occurs slightly below 2 kHz. This contrasts with the peak frequency in the diffuse-field absorption cross section (Fig. 2.1), which varied with frequency and occurred at higher frequencies for younger infants. Thus, maturational differences in ear-canal and middle-ear function vary depending on whether the sound source is in a free field or delivered into the ear-canal using a small insert probe.

Because the wave nature of sound produces significant effects above 1 kHz in the adult ear canal, it is not simple to decompose ear-canal and middle-ear function into independent “black boxes” as the location of the sound source is varied. Models incorporating wave effects are constructed to perform such a decomposition using measured data. This is why absorbance is so useful, because its value does not vary with probe location within the canal. In contrast, acoustic admittance, which is another aural acoustic transfer function that is widely used in clinical tympanometry, varies with probe location within the ear canal. The ability to decompose an admittance tympanogram into an ear-canal volumetric component and a middle-ear component is only possible at frequencies near 226 Hz (in the absence of a model that takes into account the wave nature of sound and the motion of the ear-canal walls). Switching from admittance to absorbance allows a study of middle-ear function over a much broader frequency range (i.e., up to 8 kHz). This has clinical translational significance as further described in Feeney and Keefe.

2.2.4 Forward Ear-Canal Transfer Function

A forward ear-canal transfer function level $L_{FE}$ describes the forward transmission of sound from the ear-canal probe to the tympanic membrane. $L_{FE}$ is defined as the magnitude (level in dB) of the ratio of the forward ear-canal pressure to the total pressure at the probe (Keefe and Abdala 2007). $L_{FE}$ is relevant to the interpretation of any hearing experiment in which sound is presented within the ear canal. It describes how differences in ear-canal length and cross-sectional area influence the
forward-traveling pressure wave that is directed toward the eardrum in terms of the total pressure measured by the probe microphone, and includes standing-wave effects in the ear canal. It also depends on the processes of sound absorption and reflection at the tympanic membrane. \( L_{FE} \) varies systematically with age as a result of the maturation of the acoustical functioning of the ear canal and middle ear.

Wideband measurements of \( L_{FE} \) in infants and adults show a prominent peak in the adult ear close to 3.4 kHz, but no major peak in infants (see top panel of Fig. 2.3). This emphasizes the importance of standing waves in the adult ear canal between 3 and 4 kHz, and confirms the relatively flat response in the shorter ear canals of infants. The fact that the ear-canal length is about twice as long in the adult ear as in the 6-month-old helps explain differences in \( L_{FE} \) between adults and infants in Fig. 2.3. The pronounced maximum \( L_{FE} \) in adults is a standing-wave effect in the ear canal that is much reduced in infants.

The general pattern suggested by Fig. 2.3 is that wideband acoustic transfer functions of the ear canal and middle ear are immature in infants. These functions remain non-adult-like until at least 11 years of age (Okabe et al. 1988). This long period of maturation throughout childhood is consistent with the slow maturation of some components of ear-canal and middle-ear anatomy described in Sect. 2.1. Maturation differences in these transfer functions affect stimulus levels used in hearing development experiments.

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**Fig. 2.3** The mean \( L_{FE} \) (top) and \( L_{RE} \) (bottom) are plotted versus frequency for adult and infant age groups.

![Absolute Forward Ear–Canal Transfer Function](image)

![Absolute Reverse Ear–Canal Transfer Function](image)
2.2.5 Reverse Ear-Canal Transfer Function

An evoked OAE measurement presents forward-directed sound stimuli, which elicit a cochlear-generated signal that is reverse-transmitted back to the ear canal. Unlike other acoustic variables used in hearing experiments, OAEs depend not only on $L_{FE}$ but also on a reverse ear-canal transfer function level ($L_{RE}$). $L_{RE}$ is defined in terms of the magnitude of the ratio of the total pressure measured at the ear-canal probe microphone to the reverse-transmitted pressure in the canal adjacent to the tympanic membrane. This transfer-function level is plotted for adult and infant age groups in the bottom panel of Fig. 2.3 based on results in Keefe and Abdala (2007). Aside from excess variability across age below approximately 0.5 kHz, where OAE responses are rarely measured, $L_{RE}$ in infants varies within a few decibels relative to adult levels between 0.5 and 6 kHz. This contrasts with the large difference in $L_{FE}$ between 2.8 and 4 kHz that was observed between infants and adults. Above 6 kHz, $L_{RE}$ is much larger in adult than infant ears, which acts to attenuate the level of high-frequency OAEs in infants relative to adults.

2.2.6 Forward Middle-Ear Transfer Function

At any particular frequency, a forward middle-ear transfer function level $L_{FM}$ describes the forward transmission of sound through the middle ear from the tympanic membrane to the cochlear vestibule. It is defined in terms of the ratio of the forward-directed pressure difference at the basal end of the cochlea to the ear-canal forward pressure just in front of the tympanic membrane. The total forward transfer function from the ear canal to the cochlear base is $L_f = L_{FE} + L_{FM}$. For any hearing experiment, knowledge of $L_f$ would describe the transformation of stimulus level associated with ear-canal and middle-ear function. Although $L_{FE}$ was straightforward to evaluate based on measurements of aural acoustic transfer functions (see top panel of Fig. 2.3), a measurement of $L_{FM}$, and thus $L_f$, would require highly invasive procedures that are impossible to perform in human subjects for ethical reasons.

From the standpoint of understanding hearing development, it is important to understand maturational differences in how sound is conducted through the ear canal and middle ear, or, in the general case of free-field listening, through the outer ear and middle ear. Although this conductive pathway functions as a linear system (in the absence of middle-ear muscle reflex effects that occur at relatively high sound levels), it, nonetheless, may have a profound effect on cochlear nonlinearity, and thus on the neural encoding of sound. As considered in detail later in this chapter, cochlear function has a compressive nonlinearity. For example, this means that a 1-dB increase in the sound pressure level in the ear canal produces an increase of less than 1 dB in the amplitude of vibration of the basilar membrane. If the forward conduction of sound through the outer and middle ear would differ between infant and adult ears, then the effective input to the cochlea would also differ. Any such change in input level would modify the “set point” on the compressive nonlinearity of cochlear mechanics. Thus, any maturational effects of linear middle-ear functioning become intermixed with effects of cochlear nonlinearity.
An OAE experiment differs from other hearing experiments in that the OAE is measured in the ear canal. As further described in Sect. 4.2, a combination of non-invasive measurements can reveal how $L_{FM}$ differs between infant and adult ears.

### 2.2.7 Newborn Ear-Canal Wall Motion Effects

The increased compliance of the ear-canal wall in infants compared to older children and adults has functional consequences. Video-otoscopy results show that air pressures close to 300 daPa produced changes (with significant intersubject variability) of up to 70% in ear-canal diameter in some 1- to 5-day-olds, 10% changes or less in infants between 31 and 56 days, and no detectable change beyond 56 days (Holte et al. 1991). It is reasonable that variations in acoustic pressure within the canal might also produce variations in wall displacement. A model of ear-canal wall motion in infants was developed using a viscoelastic model of the mechanics of how the wall moves in response to pressure changes (Keefe et al. 1993). Properties of aural acoustic transfer functions below 1.2 kHz, which were observed in 1- to 3-month-old infant responses but not in responses from older children and adults, were predicted by this model. Whereas sound introduced into the ear canal of an older child or adult acts on the air enclosed within the ear canal and the tympanic membrane, sound in the neonatal ear canal also acts “in parallel” to drive ear-canal wall motion. Finite-element modeling of the newborn ear canal at frequencies between 226 Hz and 1 kHz confirms that sound presented in the ear canal produces a sinusoidal volume change of the ear canal (Qi et al. 2006). A finite-element model of a 22-day-old newborn middle ear (Qi et al. 2006) predicts that the ear-canal wall volume displacement may be larger than the volume displacement of the tympanic membrane. This is a confounding factor in interpreting standard 226-Hz probe tone tympanograms in full-term infant ears in the first 2 or 3 months of development.

### 2.2.8 Acoustic Reflex Effects

High-level sound presented in the ear canal can elicit a contraction of the stapedius muscle of the middle ear to produce a middle-ear muscle reflex (MEMR). This change can be detected in terms of a measured change in level or phase of a probe sound in the ear canal. An acoustic reflex test is influenced by ear-canal, middle-ear, cochlear, and afferent and efferent neural function. A wideband test of the acoustic reflex was used to measure thresholds in 80 ears of normal-hearing adult and 375 ears of newborn infants with normal hearing based on having distortion product OAE (DPOAE) responses within a normal range (Keefe et al. 2010). Although the MEMR is typically assumed to extend to frequencies below 2.8 kHz, reflex shifts were observed at frequencies up to 8 kHz. These observed shifts may also include contributions from the medial olivocochlear efferent system in addition to the MEMR.

MEMR thresholds were measured using a broadband noise activator to elicit the reflex. Thresholds were quantified in terms of the minimum activator SPL at which a reflex shift was observed. Infant thresholds based on in-the-ear SPL were only
2 dB higher than adult thresholds. Inasmuch as this 2-dB difference was small compared to variability in thresholds within each age group, the MEMR thresholds using broadband noise did not appear to differ between newborn infants and adults. Interpreting this outcome is complicated by procedural differences between infant and adult ears in how a reflex shift was classified as present or absent, and by the fact that the forward transmission of sound in the broadband noise activator and probe signal varied across frequency as well as across age.

3 Development of Human Cochlear Function

3.1 Developmental Changes in Anatomy

General mammalian cochlear development proceeds from base to apex (Bredberg 1968; Pujol and Hilding 1973). This gradient applies both to general architecture as well as cellular and subcellular mechanisms reviewed below (Pujol et al. 1991). Around the fifth embryological week in humans, the otocyst is formed by a cartilaginous matrix (Sanchez-Fernandez et al. 1983). At 9 weeks, three cochlear coils are apparent with a well-differentiated otic capsule and a septum separating the coils. Around this time, the process of hair cell differentiation begins. Afferent fibers, and possibly efferent, are entering the otocyst and forming what appears to be targeted, specific synapses well before hair cells are fully differentiated. At this point in development, transmission electron microscopy shows presumptive hair cells in the epithelium that can be distinguished from supporting cells by cytoplasmic content and afferent fibers at their base (Pujol and Lavigne-Rebillard 1985).

By weeks 10 and 11, a more definitive differentiation of inner (IHCs) and outer hair cells (OHCs) begins with IHC preceding OHC development. Stereocilia starts to form on IHCs and about a week later, on OHCs. This sequence exemplifies the second developmental gradient noted in the cochlea: IHC differentiation, morphological development and innervation all precede comparable processes in the OHC. Once stereocilia begin to form, microvilli disappear quickly from the cuticular plate and the remaining cilia are organized in three or four parallel rows, graded in length. Lateral and tip links are observed as soon as stereociliary organization is established (Lavigne-Rebillard and Pujol 1986; Lim and Rudea 1992). Hair cell length is changing at this time, along with stereocilia length. IHC length is similar throughout cochlea; however, OHC length depends on the cell’s position along the basilar membrane and increases from base to apex (Pujol et al. 1992). By weeks 20 and 21, there are adult-like stereocilia bundles on both types of hair cells (Lavigne-Rebillard and Pujol 1986; 1987). The general shape of the OHC remains immature much longer than IHC shape (Pujol and Hilding 1973). Both types of cells are organized in rows but OHCs are less regimented and more chaotic in their organization compared to highly organized IHCs. At 20 fetal weeks, OHCs in the apical cochlea are sparse and their organization is particularly chaotic, with irregular spacing and
uneven development of ciliary bundles (Tanaka et al. 1979). At some point in
development, supernumerary sensory cells that are generally not seen in the mature
system are present in the developing cochlea (Bredberg 1968; Igarishi 1980).
The functional significance of these extra sensory cells is not well understood.

The protracted course for OHC development in mammals is loosely correlated
with changes in functional properties of the cochlea such as increasing sensitivity,
frequency tuning, and shifts in the place code (Pujol et al. 1980; Romand 1987). For
example, there is an accumulation of actin in the cuticular plate (Raphael et al.
1987), redistribution of mitochondrial and endoplasmic reticulum, and the forma-
tion of intricate subsurface cisternae preceding OHC motility observed in vitro.
OHC somatic motility is thought to power the cochlear amplifier, which augments
tuning and auditory sensitivity. The human OHC appears morphologically mature,
including postsynaptic specialization, some time in the third trimester, though this
final stage has not been well delineated. It is not clear whether subtle morphological
immaturities remain after term birth (Pujol et al. 1998).

The physical features of the basilar membrane have not been characterized
during fetal and early postnatal life. Imaging of delicate membranes in postmortem
temporal bones may not provide an effective means of characterizing features such
as basilar membrane mass and/or its stiffness gradient. Supernumerary hair cells
have been identified in fetal and neonatal tissue and could affect mass of the mem-
brane, as could differences in its dimensions. At present, the maturational time
course for the dimensions and material properties of the basilar membrane in the
human auditory system is not known.

Afferent VIIIth nerve fibers stop spreading and focus on the base of the existing
sensory cells once the hair cells are fully differentiated. They innervate IHCs around
the 11th or 12th fetal week (Lavigne-Rebillard and Pujol 1986). Vesiculated efferent
endings are observed in the inner spiral sulcus below IHCs around the 14th week
(Pujol and Lavigne-Rebillard 1985). The subsequent axosomatic synapses that form
between large medial efferent fibers and IHCs are transient and may represent a
transitional period (Simmons 2002). The formation of more permanent, classic
efferent axosomatic synapses with OHCs occurs sometime between 20 and 30 weeks
(Lavigne-Rebillard and Pujol 1990). These synapses are most scarce at the apex of
the cochlea and continue to show immaturities, such as incompletely developed
postsynaptic features and small presynaptic varicosities into the third trimester and
possibly beyond.

The morphological and functional development of the cochlea does not determine
the onset of hearing. Auditory nerve fibers must be able to conduct sound-evoked
impulses to produce hearing. This milestone occurs sometime around 25–27 weeks
as defined by in utero studies of fetal movement or heart rate (Birnholz and
Benacerraf 1983) and 27–28 weeks as defined by onset of auditory brain stem
responses (ABRs) that reflect averaged, synchronous nerve and brain stem poten-
tials (Galambos and Hecox 1978). Although present by 27 fetal weeks, the ABR is
not mature when initially observed and requires approximately the first year of
postnatal life to become adult-like in morphology and speed of transmission
(i.e., latency) as detailed in Eggermont and Moore, Chap. 3.
3.2 Maturational Changes in Function

3.2.1 Basic OAE Characteristics

Otoacoustic emissions provide a unique, noninvasive window into human cochlear function through which auditory peripheral maturation can be studied. OAEs are preneural cochlear responses that can be easily and noninvasively recorded with a sensitive microphone and earphone assembly placed at the entrance of the ear canal, making them ideal measures to study human cochlear function.

Early studies reported spontaneous OAEs (SOAEs) to be present in infants and adults with equal prevalence, around 65% (Strickland et al. 1985; Burns et al. 1992). However, later reports found higher prevalence in premature and term newborns, ranging from 82% to 90% (Morlet et al. 1995; Abdala 1996). By all accounts, newborns have higher level SOAEs than adults (Burns et al. 1992). It is not unusual to observe a newborn SOAE in the range of 20–25 dB SPL compared to a more modest 5–10 dB SPL signal in a young adult. In addition, newborns also have more numerous SOAEs per ear than adults, although they show adult-like sex and ear trends: females and right ears have more SOAEs.

Approximately 90–100% of normal-hearing adults and newborns exhibit click-evoked OAEs (CEOAEs) (Bonfils et al. 1989). Prematurely born neonates in the NICU are reported to have slightly lower prevalence rates of approximately 80–90%, especially if they are tested within the first 3 days of birth, most likely due to debris and fluid that drains from the external auditory canal and middle-ear space in the first 72 h (Stevens et al. 1990). Newborns have higher level CEOAEs in general than adults and adolescents; response levels decrease with increasing age (Norton and Widen 1990; Prieve 1992).

Distortion product OAEs (DPOAEs) are also present in 90–100% of normal-hearing adults and newborns (Lonsbury-Martin et al. 1990; Bonfils et al. 1992). Neonates tend to have slightly higher DPOAE levels (~2–5 dB) than adults, generally in the low- and mid-frequency range (Lasky et al. 1992; Smurzynski et al. 1993). Prematurely born infants tested at 33–36 weeks postconceptional age have mildly reduced DPOAE levels compared to their term-born counterparts; however, levels increase with age through 40 weeks, at which time they are comparable to those observed in term neonates (Smurzynski 1994; Abdala et al. 2008). Between birth and 6 months of age, there is little change in OAE level, though OAEs continue to show greater magnitude than adult emissions well into childhood (Prieve et al. 1997a, b).

Spontaneous OAEs are centered near 2.3 kHz for adults and 3.5 kHz for newborns (Strickland et al. 1985; Burns et al. 1992; Abdala 1996). Some studies have indicated a consistent upward shift of SOAE frequency (in absence of amplitude shift) as a function of postconceptional age in prematurely born infants (Brienesse et al. 1997). Other studies have reported stable SOAE frequency through 24 months of age in human infants (Burns et al. 1994). It appears that the bias toward high frequencies disappears sometime between birth and preadolescence (Strickland et al. 1985). Evoked OAEs from neonates generally show an extended high-frequency...
spectral content compared to those from adults. Both CEOAE and DPOAE spectra are flatter and exhibit a wider bandwidth than the adult response (Lasky et al. 1992; Abdala et al. 2008).

### 3.2.2 OAE Generation Mechanisms

**Background**

Otoacoustic emissions are generally present in the normal mammalian cochlea if the cochlear amplifier, driven by OHC somatic motility, is intact and functional. Thus, OAEs reflect normal OHC physiology and cochlear amplifier function. With little exception, when OAEs are absent (as long as the conductive system is normal), cochlear function can be judged to be abnormal. In the last 10 years, an updated taxonomy has been developed that differentiates among OAE types by their source region on the basilar membrane (Knight and Kemp 2001) and by their distinct generation mechanisms (Shera and Guinan 1999). Investigating these generation mechanisms in the developing human auditory system may provide a more targeted study of how and when distinct aspects of human cochlear function mature.

It is hypothesized that different OAE types reflect at least two cochlear processes (Shera and Guinan 1999). DPOAEs are a “mixed” type emission, encompassing both cochlear processes, and thus best elucidating this framework of OAE generation. In addition, DPOAEs are typically recorded with moderate level stimulus tones in newborns, making it possible to achieve adequate signal-to-noise ratio (SNR). Other OAEs, such as stimulus frequency OAEs (SFOAEs), may be less confounded by mixed sources but require a low-level stimulus. SFOAE measurements have only recently been reported in human newborns (Kalluri et al. 2011). Recent theory, supported by strong experimental evidence, contends that DPOAEs comprise two fundamentally different mechanisms: (1) OHC-based intermodulation distortion, which is an intrinsically nonlinear process (generated at the overlap region between primary tones) and (2) linear coherent reflection initiated near the DPOAE frequency by place-fixed irregularities or “roughness” along the length of the basilar membrane (Zweig and Shera 1995; Shera and Guinan 1999).

The phase behavior of each of the two DPOAE components differs markedly (Talmadge et al. 1998; Shera and Guinan 1999). For distortion or “wave-fixed” components generated at the overlap of the $f_1, f_2$ primary tones (and recorded with a fixed $f_2/f_1$ ratio), phase is relatively invariant over most of the frequency range. The distortion source is induced by the wave (in this case, the interaction of the two primary tones) thus, the source moves with the wave as stimuli are swept in frequency. Since DPOAE phase is referenced to the phases of the primary tones, as long as the $f_2/f_1$ ratio is kept constant, DPOAE phase should be constant. In contrast, the reflection or “place-fixed” component generated at $2f_1-f_2$ is linked to irregularities along the membrane; hence, the corresponding reflection phase shifts with these irregularities because they are fixed in position. This produces rapidly rotating phase, and consequently a steep OAE phase slope (Shera and Guinan 1999).
Output from an inverse fast Fourier transform (IFFT) conducted to separate the two components is shown in Fig. 2.4. The upper panel of Fig. 2.4 shows the phase behavior from the “mixed” ear canal DPOAE as well as each of the two DPOAE components after separation by a time windowing technique. The DPOAE measured at the microphone in the ear canal represents the vector sum of these components, which combine constructively and destructively. It is the difference in phase rotation of the components that produces DPOAE fine structure shown in Fig. 2.5.
When they are similar in magnitude and 180° out-of-phase, components cancel, producing dips or minima in fine structure; when they add while in-phase, DPOAE level is augmented as noted by peaks or maxima in fine structure.

Overall, at moderate and high stimulus levels, the nonlinear distortion component of the DPOAE is dominant in normal ears across most of the frequency range, as noted in the lower panel of Fig. 2.4. CEOAEs, SFOAEs, and SOAEs exhibit phase features consistent with linear reflection linked to fixed-position perturbations along the basilar membrane. There are several reviews written to describe this theoretical framework of OAE generation in detail and the reader is referred to these for additional information (Shera and Guinan 1999; Shera 2004; Shera and Abdala 2010).

Development

How can this emerging theoretical framework of OAE generation contribute to the study of peripheral auditory system development? If OAE components reflect distinct cochlear properties, do these distinct properties mature at different rates and does the relative contribution of each source-type to the OAE measured at the microphone change during infancy or childhood? Does OAE phase and its rotation as a function of frequency offer a clue about basilar membrane motion during development? To what extent do immaturities in outer- and middle-ear function contribute to the observed immaturities in OAE responses? These are intriguing questions that

**Fig. 2.5** When recorded with high resolution, DPOAE level exhibits alternating peaks (maxima) and valleys (minima) or fine structure, reflecting interference between two DPOAE sources.
are now being explored to better understand the maturation of cochlear function. As of yet, few studies have separated and individually analyzed DPOAE components in human infants to scrutinize targeted cochlear properties.

A recent series of experiments (Abdala and Dhar 2010; Abdala et al. 2011b) has begun to describe DPOAE fine structure, phase, and individual DPOAE components in human newborns. These investigations found more prevalent DPOAE fine structure and narrower spacing between oscillations in newborns compared to adults. An IFFT and time-windowing technique was applied to separate DPOAE components (see Fig. 2.4) and found that, although the distortion-source component of the DPOAE was similar in magnitude between adults and newborns, the reflection component was significantly larger in the infants. Enhanced newborn reflection, coexisting with adult-like distortion levels, prompts speculation that the peripheral properties underlying each component may have distinct maturational time courses. The implications of this finding are considered in Sect. 4. The ability to study two unique OAE components, each purported to represent distinct physiological processes within the cochlea, may allow for more targeted study of peripheral maturation. The need for further study notwithstanding, it may be possible to move beyond simple categorical distinctions of mature versus immature to more precisely characterize age-related (or with a bit of optimism, pathology-related) changes in targeted cochlear properties such as OHC nonlinearity, cochlear tuning and cochlear amplification.

3.2.3 DPOAE Phase and Basilar Membrane Motion

OAE phase may offer a unique glimpse into aspects of cochlear function. DPOAE phase is influenced by the material properties of the basilar membrane such as the spatially distributed stiffness, mass and damping. In gerbil, active processes (cochlear amplifier gain and sharp tuning) in the cochlear base are eliminated after death, whereas basilar membrane phase is minimally altered (Ren and Nuttall 2001). These findings suggest that active processes can be impacted independently from the more gross aspects of passive basilar membrane motion. Likewise, it may be possible to examine their maturational time courses somewhat independently. OAE phase may provide a tool for this purpose.

Some relatively early studies measured CEOAE and/or DPOAE onset latency and phase in an attempt to estimate cochlear travel time in human infants. However, these produced widely disparate results, which are now understood to be primarily due to methodological differences (Brown et al. 1994, 1995; Eggermont et al. 1996). As new theories of OAE generation have emerged, DPOAE phase has taken on renewed importance and more attention has been paid to its precise quantification and interpretation (Shera et al. 2000; Tubis et al. 2000). Two recent observations have reported DPOAE phase from human infants (Abdala and Dhar 2010; Abdala et al. 2011b). In the first of these studies, DPOAE phase was analyzed as group delay in adults and term newborns. Although both age groups showed a prolongation of group delay in the low frequencies (consistent with a break from cochlear
scaling in the apical half of the mammalian cochlea), the newborn delay was significantly more prolonged than the adult delay (upper panel of Fig. 2.6). A longer group delay represents a steeper DPOAE phase gradient.

A subsequent experiment extended these initial findings using a targeted, low-frequency protocol with extensive averaging to ensure adequate SNR and to further scrutinize apical cochlear function in newborns. Initially, a suppressor tone was
presented near the DPOAE frequency \((2f_0 - f_2)\) to ensure that the DPOAE was dominated by the distortion component (not the reflection source). The lower panel of Fig. 2.6 shows the resulting phase versus frequency functions for infant and adult subjects. The “break” frequency (elucidating the transition from invariant to steeply sloping phase) as well as the phase slope of the segment above and below this transition frequency were calculated. The break, thought to reflect the apical–basal demarcation, was centered near 1.4 kHz for both adults and newborns. However, consistent with the previous report, newborns exhibited significantly steeper slope of phase than adults in apical regions of the cochlea. The implications of this finding with respect to cochlear scaling are considered in Sect. 4.1.

### 3.2.4 Cochlear Response Growth

The relationship between a signal presented to the cochlea and the resulting cochlear output can be represented with an input/output (I/O) function. Although influenced by many complex factors, the OAE I/O function may provide insight into cochlear compressive nonlinearity. The typical adult OAE I/O function shows a relatively linear increase in amplitude with low stimulus levels and response saturation at mid- to high levels. In adults, click-evoked OAEs clearly show this pattern of nonlinear, compressive growth (mean slope = 0.34) (Prieve 1992). There are no detailed data on CEOAE I/O functions in newborns and sparse data describing pediatric CEOAE I/O functions in general. The most comprehensive study included subjects aged 6 months through 17 years and reported that CEOAE I/O functions were roughly similar in shape and slope across all age groups (Prieve et al. 1997a).

DPOAE response growth has been fairly well characterized during development. Early reports described neonatal DPOAE I/O functions as nonmonotonic, with slope values ranging from 0.5 to 0.6 depending on frequency (Popelka et al. 1995). Others described I/O slope values that ranged from 0.25 at the lowest frequencies to 0.9 at the highest \((f_2 = 10\, \text{kHz})\) (Lasky et al. 1992; Lasky 1998). More recent studies (Abdala and Keefe 2006; Abdala et al. 2007) reported that infant DPOAE I/O functions were displaced upward, consistent with higher DPOAE amplitude, and were more linear compared to adult functions. Up to 37% of I/O functions from premature newborns did not plateau, even at maximum stimulus levels of 85 dB SPL (Abdala 2000), whereas adults rarely showed functions without a saturating segment. Overall, adult I/O functions saturated around 65–70 dB SPL, whereas the infant functions that showed saturation plateaued at stimulus levels of 75–80 dB SPL. The DPOAE saturation threshold moved downward toward adult values throughout the premature period (33 weeks PCA) and during the first 6 postnatal months (Abdala 2003; Abdala et al. 2007). Figure 2.7 shows mean DPOAE I/O functions from infants tested at birth and again at 3, 4, 5, and 6 months of age. The source of age effects on the DPOAE I/O function appear to be related, not to cochlear immaturity as initially hypothesized, but rather to middle and ear canal immaturities (considered further in Sect. 4.1).
3.2.5 Development of Cochlear Frequency Selectivity

OAE ipsilateral suppression has been applied to the study of cochlear tuning in human infants. OAE amplitude is measured in the test ear at a fixed primary tone level and \( f_2/f_1 \) while ipsilateral tones are presented across a range of frequencies and levels to suppress the emission. Iso-response OAE suppression tuning curves (STCs) are generated by plotting the suppressor level required to produce a fixed-criterion amplitude reduction as a function of suppressor frequency. In the normal ear, STCs show morphology that is similar to auditory nerve frequency threshold curves and psychoacoustic tuning curves: a narrow tip centered slightly higher than the \( f_2 \) frequency, a steep high-frequency flank, and a shallower low-frequency flank with a tail-like segment for lower-frequency primary tones (Brown and Kemp 1984; Martin et al. 1987).

Early studies of DPOAE STCs generated with primary tones of \( f_2 = 3 \) kHz and 6 kHz (and a 6 dB suppression criteria) found roughly comparable tuning curve shape in term-born neonates and adults (Abdala et al. 1996). However, subsequent studies including larger groups of prematurely born as well as term-born infants, and more detailed quantification of STC features, found clear age differences in DPOAE suppression tuning between newborns and adults (Abdala 1998, 2001, 2003). Newborns showed better, narrower DPOAE suppression tuning with a steeper slope.

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**Fig. 2.7** Mean DPOAE input/output functions recorded in a group of adults and infants tested at birth and then at four additional postnatal ages as denoted in the key [This figure was originally published in Abdala and Keefe (2006)].
on the low-frequency flank and a deeper, sharper tip than adults at $f_2 = 6$ kHz (see Fig. 2.8). At $f_2 = 1.5$ kHz, more subtle age differences were noted (e.g., the tuning curve width was narrower in newborns) but they were not as consistent, possibly because of elevated noise floors in the low-frequency range.

In lieu of generating iso-response STCs, plotting DPOAE level as a function of increasing suppressor level characterizes the growth of suppression across frequency. Tones on the low-frequency side of the probe ($< f_2$) produce linear or expansive suppression growth whereas high-frequency side suppressors ($> f_2$) produce suppression that is compressive and shallow (Abdala and Chatterjee 2003), as shown in Fig. 2.9. This pattern is consistent with both mechanical and neural responses observed in laboratory animals and closely mimics the two-tone suppression patterns recorded at various levels of the auditory system (Abbas and Sachs 1976; Delgutte 1990; Ruggero et al. 1992). It suggests that suppressors around or higher than $f_2$ are impacted by the expected compressive nonlinearity (just basal to the peak of the $f_2$ traveling wave), and thus are not effective suppressors as their level is increased. Conversely, suppressor tones lower in frequency than $f_2$ produce approximately linear reduction of the DPOAE with increases in level as the “tail” of the traveling wave effectively suppresses the more basal $f_2$ site. As noted in Fig. 2.9,
newborns show elevated suppression thresholds and non-adultlike (more compressive) growth of suppression for suppressors $<f_2$. This pattern of suppression growth explains the source of the steep low-frequency flank in newborn STCs.

A longitudinal study tracking DPOAE suppression tuning and suppression growth in a group of nine premature neonates over a 2-month period (33 weeks PCA through 38–40 weeks PCA) confirmed that the narrow tuning, steeper low-frequency flank and the sharper, deeper tip of the DPOAE STC at 6 kHz does not become adult-like by the equivalent of term birth (Abdala 2003). A second study (Abdala et al. 2007) extended this finding to the postnatal period and showed that STC width ($Q_{10}$), tip-to-tail measures, STC low-frequency slope and tip level remain immature, consistent with narrower and sharper tuning through at least 6 months of age, as noted in Fig. 2.10.

### 3.2.6 The Medial Olivocochlear (MOC) Reflex

No discussion of peripheral auditory development is complete without considering maturation of the descending, inhibitory efferent fibers that modulate cochlear function.
The MOC system is thought to enhance auditory perception in difficult listening conditions and provide a measure of protection from noise damage (Micheyl and Collet 1996; Maison and Liberman 2000). In laboratory animals, both visual and auditory inhibitory synapses have critical periods during development that influence later perception if disrupted (Walsh et al. 1998; Morales et al. 2002). In addition, as described in the previous section on maturation of cochlear anatomy, the medial efferent innervation of OHCs is one of the later developing processes in cochlear development, occurring sometime late in the third trimester and possibly into the perinatal or early postnatal period.

Early work with laboratory animals (Mountain 1980; Siegel and Kim 1982) confirmed that electrically stimulating the olivocochlear (OC) bundle in the brain stem alters cochlear output. Later work in humans showed that acoustic stimulation can evoke similar activation of OC fibers, producing reduction in OAE magnitude, presumably by hyperpolarizing OHCs (Puel and Rebillard 1990; Collet 1993). The medial portion of the OC tract (MOC) is strongly cholinergic and predominantly innervates OHCs through both crossed and uncrossed pathways (Fex 1962). A reduction in OAE amplitude when the MOC system is activated reflects MOC-induced inhibition (Guinan 2006). One typical paradigm used to probe MOC-mediated

Fig. 2.10 Mean DPOAE suppression tuning curves ($f_2 = 6,000$ Hz, $L_1-L_2 = 65-55$ dB SPL) recorded from young adults and a group of infants tested initially at birth and then again at 3, 4, 5, and 6 months of age [This figure was originally published in Abdala et al. (2007)]
regulation of cochlear function includes the presentation of broadband noise (BBN) in the contralateral ear while OAE level is monitored in the ipsilateral or test ear.

The contralateral MOC reflex has been studied in human newborns, though methodology varied significantly among studies. Given updated theories of OAE generation, it is clear that appropriate controls may not have been included and parameters were likely not optimized in these early studies (Morlet et al. 1993; Ryan and Piron 1994; Abdala et al. 1999). In general, CEOAE-based measures of the MOC reflex in newborns were reported to be immature in prematurely born infants, but adult-like by term birth (Morlet et al. 1993; Ryan and Piron 1994). DPOAE-based measures of the MOC reflex, likewise, found that adults and term-born infants exhibit comparable MOC effects at low and mid frequencies (Abdala et al. 1999). The magnitude of the reflex on average was 1.2 dB at $f_2 = 1.5$ kHz. Premature newborns, in contrast, did not show a significant MOC reflex; however, nearly half of their data included episodes of increasing DPOAE level (i.e., enhancement) produced by contralateral BBN. When enhancement values were eliminated, the magnitude of the MOC reflex overlapped for the three age groups suggesting that the strength of the reflex may be adult-like even during the premature period. Nevertheless, the increased prevalence of DPOAE level enhancement in prematurely born infants is noteworthy and may be related to interference between dual DPOAE sources.

The most current DPOAE-based protocols to measure the MOC reflex have taken the dual-mechanism model into account and calculate the reflex at fine structure peaks only or for the distortion- and reflection-source components separately (Abdala et al. 2009; Deeter et al. 2009). Consider the following sequence of events: (1) The DPOAE is recorded at a frequency where distortion and reflection components sum in the ear canal while 180° out-of-phase, producing cancellation (i.e., at a minimum frequency in fine structure); (2) the MOC reflex is activated by BBN and alters the reflection-source more than the distortion-source component, consequently shifting the phase relationship between them; (3) the shifted phase relationship releases phase cancellation, thus producing an abrupt and marked increase in DPOAE level. This DPOAE enhancement appears to be largely due to component interference. To address component interference (and its effects on measures of the MOC reflex), a recent investigation recorded the MOC reflex at DPOAE fine structure peak frequencies. This ensures that DP components were in-phase (Abdala et al. 2009). More than 90% of resulting MOC reflex values showed inhibition with this technique and overall mean MOC reflex values were in the range of 1.5–2.0 dB in young adult ears.

DPOAE-based MOC reflex protocols, with additional controls for component interference, phase- and magnitude-based reflex metrics and MEMR measures, are currently being applied to prematurely born and term newborns to assess the maturation of the MOC system (Abdala et al. 2011c). Preliminary data from 23 term-born neonates shows an overall mean MOC inhibitory reflex of 1.3 dB in newborns compared to 1.1 dB in adults. When measuring the MOC reflex as a vector difference (i.e. considering BBN-induced phase changes as well as magnitude effects) term newborns and adults showed mean MOC reflexes (expressed as a fraction of baseline DPOAE amplitude) of 0.21 and 0.16, respectively. These
preliminary results suggest robust, adult-like MOC reflexes at term birth; however, reflex strength may not be the only metric of maturity. It will be informative to assess the efficiency of the MOC reflex in providing enhanced signal in noise detection. These efforts are currently ongoing. The development of careful OAE-based measurement and analysis protocols for the MOC reflex may help disentangle the hypothesized sources of auditory peripheral immaturity in human newborns.

4 Interpretation of Findings

In the preceding section, non-adultlike OAE results from human infants were reported. The interpretation of these findings is not straightforward because the OAE is generated in the cochlea (and thus, sensitive to cochlear function) but is influenced by ear-canal and middle-ear function, as well as by descending medial efferent system function. The complexity of the task notwithstanding, the overarching objective is to explore what an infant hears. Thus, the following section attempts to place the reported findings into a cohesive, or at least interesting, explanatory framework for readers. Section 4.1 explains the OAE immaturities based on the possibility of cochlear immaturity (or non-adultlike function) during the perinatal and/or early postnatal period. Section 4.2 explains these immaturities in OAE responses based on the theory that cochlear function is mature at birth.

4.1 Cochlear Explanations

4.1.1 Active Processes

Adult–newborn differences in the prevalence, level, and number of SOAEs per ear have been observed. The global standing wave theory (Kemp 1979; Zweig 1991; Zweig and Shera 1995) predicts that SOAEs are standing wave resonances produced by multiple internal reflections, initiated by external sound or intrinsic physiological noise. Reflections occur off of irregularities on the basilar membrane and are “…self-sustaining when the total round trip power gain matches the energy loss (i.e., viscous damping and acoustic radiation in the ear canal) experienced en route” (Shera 2003, p. 245). Consistent with numerous and robust SOAEs in newborns, when the DPOAE is separated into its two components (distortion and reflection), the relative contribution of the reflection source is enhanced in newborns compared to adults. Thus, both SOAE and component-specific DPOAE findings are indicative of stronger cochlear reflection at birth.

The enhanced newborn OAE reflection component could be interpreted in several ways. It might indicate that the infant basilar membrane has more irregular or “rough” architecture and less regimented cellular organization, producing more robust back-scattering of energy; however, more roughness alone would not ensure
coherence of reflected wavelets. More likely, it indicates stronger cochlear amplification in newborns relative to adults. Reflection emissions are a low-level source, thought to mirror efficiency of the cochlear amplifier as they arise from the peak of the traveling wave. The coherent reflection model explains this type of OAE as reflection of incident sound from distributed inhomogeneities on the basilar membrane (i.e., cochlear roughness). In this model, the amplitude of the OAE is increased with increased height and breadth of the traveling wave. A more active newborn amplifier would produce enhanced gain, a taller and broader wave, and consequently an increased coherence of the backscattered wavelets. If the mechanisms of cochlear reflection are strongly functional at birth, other reflection emissions (CEOAEs, SFOAEs, and SOAEs) should also be more robust in infants. This, indeed, is a common finding (Norton and Widen 1990; Prieve 1992; Kalluri et al. 2011).

What process might provide stronger cochlear amplification in the neonatal cochlea? The idea of a functional overshoot period is plausible. The cochlear amplifier gain and tuning may be excessive during a brief developmental interlude, similar to what had been described in neonatal gerbils from 23 to 29 days after birth (Mills and Rubel 1996). The source of this overshoot in gerbils appears related to changes in the endocochlear potential (EP). Mills and Rubel hypothesized an adaptation mechanism that adjusts the “set-point” of the gain for optimal processing while reserving excess power. It is not known when the EP becomes adult-like in human infants because this requires invasive methodology. However, a transient overshoot period of similar origin, characterized by exuberant cochlear amplifier activity, is possible. A second possibility is that the pristine neonatal cochlea, as of yet untarnished by noise and ototoxins is at the pinnacle of performance, manifesting optimally functional cochlear amplification at birth, in contrast to an adult cochlea already impacted by natural aging, common ototoxins and noise. In this scenario, the newborn cochlea is non-adultlike but not immature since a subtle, subclinical degradation of the adult cochlear amplifier would be the source of age effects. Third, it is also possible that the efficiency of the medial efferent-OHC synapses may not be fully developed, and thus efferent-mediated modulation of OHC motility may not be effective, leading to somewhat unregulated gain and sharper tuning for a brief developmental period.

Another cluster of findings consistent with a strong neonatal cochlear amplifier is excessively narrow DPOAE suppression tuning observed in infants. Active processes are thought to underlie exquisite frequency tuning in the human cochlea. Recall that when the slope of suppression growth was examined in adults and infants for $f_2=6$ kHz, low-frequency side suppressors (< $f_2$) showed elevated suppression thresholds and compressive growth of suppression, consistent with narrower DPOAE suppression tuning (see Figs. 2.9 and 2.10). Because suppression growth is measured as a slope value derived re: each subject’s own suppression threshold, it is difficult to explain these results with simple attenuation of sound pressure level through an immature newborn middle ear. One possibility is that the elevated suppression thresholds for suppressors $<$ $f_2$ (i.e., newborn functions shifted rightward on abscissa of Fig. 2.9; upper left panel) produced abbreviated growth functions, making it difficult to obtain a comprehensive picture of suppression growth.
Elevated suppression thresholds could be explained by lower levels reaching the infant cochlea, thereby leaving the middle ear as a possible contributor to these findings.

To scrutinize the impact of middle-ear development on DPOAE STCs, Abdala et al. (2007) examined correlations between measurements of DPOAE suppression tuning at \( f_2 = 6 \text{ kHz} \) and selected acoustic transfer functions measured in the ear canal (admittance and energy reflectance at various frequencies). All measurements were performed longitudinally in the same group of infants from birth through 6 months of age. A relatively weak correspondence between cochlear and middle-ear indices was observed over the first half year of life. Of 75 correlations generated (5 DPOAE variables, 5 admittance variables, 3 frequencies), the admittance phase correlated most strongly with the tip-to-tail ratio of infant DPOAE STCs and could account for a maximum 25% of variance in DPOAE suppression tuning with age. Susceptance at 5.7 kHz could account for 18% of the variance with age. These weak associations might be due to relatively small numbers of infant subjects (\( n = 20 \)) or noise intrinsic to each data set, but they do not suggest that changes in acoustic transfer functions measured in the ear canal can fully account for the developmental changes noted in DPOAE suppression tuning during the first months of life. As can be appreciated in any longitudinal study, a limitation was that the oldest infant age group was only 6 months with no other age groups tested between this age and adulthood.

Non-adultlike DPOAE suppression growth and narrower suppression tuning may be consistent with a vigorous cochlear amplifier in newborns. However, it is not likely that active processes underlying frequency selectivity, such as OHC somatic motility, remain immature into the sixth month of life. Morphological data do not support a prolonged postnatal time course for maturation of human OHC function. As speculated earlier, it is feasible that a transient overshoot period occurs as part of an EP-related adaptation mechanism, or that the age differences noted are explained by an optimally functional newborn sensory organ contrasted with a “well-worn” adult cochlea. As presented in Sect. 4.2, compelling arguments and models also exist that explain much of the infant DPOAE suppression tuning based solely on immaturity of ear-canal and middle-ear factors.

4.1.2 Cochlear Apex

Readers will recall that the slope of DPOAE phase was steeper in newborns than in adults for low-frequency signals coded in the apical half of the cochlea (see Fig. 2.6). Morphologically, the apex of the cochlea develops last and the apical region retains some immature-like (re: the base) features into adulthood (Pujol et al. 1998). In adult guinea pigs, among other small laboratory animals, the apex does not manifest fully nonlinear behavior or provide effective cochlear amplification (Cooper and Rhode 1995). In light of this relatively prolonged apical maturation, what might a prolonged DPOAE group delay and steeper slope of phase in the low frequencies indicate for newborn cochlear function? Because the middle ear impacts stimulus levels during forward transmission, it is first important to quantify the level
dependence of DPOAE phase, in particular for frequencies below 1.5 kHz where age effects have been observed. Toward this aim, DPOAE phase and individual component features were characterized in normal-hearing young adults at four stimulus levels (Abdala et al. 2011a). The DPOAE phase slope measured in the low-frequency segment (0.5–1.4 kHz) of phase-frequency functions did not show significant level dependence. In addition, once DPOAE components were separated, the distortion-component phase (which dominates the phase behavior shown in Fig. 2.6 and hence drives the phase trend in these data) showed no level dependence either. This result suggests that lower levels driving the infant cochlea (due to inefficient forward transmission through the newborn middle ear) do not easily explain steeper DPOAE phase observed in the apical half of the neonatal cochlea.

The fact that a steep phase gradient in newborns is observed in the apical region of the cochlea is intriguing and may be suggestive of immaturities in cochlear scaling. To understand cochlear scaling symmetry, it is necessary to refer back to tonotopic representation on the basilar membrane. Signals of different frequencies have to “travel” different distances along the cochlear partition to their characteristic frequency (CF) place. Scaling symmetry implies that the number of cycles to the CF place is relatively independent of frequency, and signals of all frequencies accumulate approximately the same amount of total phase (cycles) at their unique CF site. Because the cochlear frequency-place map is exponential, the traveling wave envelope is simply transposed along the basilar membrane for different CFs, producing shift similarity (Zweig 1976). The property of scaling can be observed and gauged with DPOAE phase. In a scaled system, when a fixed $f_2/f_1$ ratio is presented, the relative phases of the primary tones are constant throughout the frequency range; consequently DPOAE phase (which is calculated re: stimulus tone phase) is constant as well (Shera and Guinan 1999; Shera et al. 2000). This DPOAE phase invariance is thought to reflect local cochlear scaling symmetry.

In the mammalian auditory system, a breakdown in scaling in the apical half of the cochlea has been described (Zweig 1976; Shera et al. 2000, 2010). Such a break can be noted in the DPOAE data presented in lower panel of Fig. 2.6, where DPOAE phase transitions from flat in the mid- and high-frequencies to more rapidly cycling in the low-frequencies. Physiological evidence supports the hypothesis that the cochlear apex functions differently than the base (Cooper and Rhode 1995; Nowotny and Gummer 2006; Shera et al. 2010). The apical-basal transition derived from DPOAE phase versus frequency functions is centered between 1 and 1.4 Hz in adults and infants (depending on how it is measured) as shown in Fig. 2.6. Of interest here is that newborns exhibit a more marked break from scale invariance than adults suggesting a relatively prolonged course for the maturation of apical basilar membrane motion. The increased steepness of phase below this apical–basal transition frequency could imply a more marked deviation in the exponential relationship between frequency and place in the newborn apex. It might also indicate an age difference in the broadening of filters in the apical half of the cochlea. These interpretations, though intriguing, are speculative and warrant further consideration and experimentation.

DPOAE phase believed to mirror basilar membrane phase and motion, shows age differences for low-frequency signals, whereas cochlear amplification in the
infant cochlea appears to be mature at birth. The developmental sequence observed in most mammals is that passive motion of the basilar membrane develops before micromechanical aspects of cochlear function. Some relatively recent findings in mouse support this sequence (Song et al. 2008), though exceptions in gerbil have been reported (Overstreet et al. 2002). If DPOAE phase is determined by the material properties of the basilar membrane, does its immaturity in newborns suggest immature traveling wave properties in the apical half of the cochlea? There is a dearth of information about mass and stiffness characteristics of the basilar membrane in human fetal tissue. Therefore this issue cannot be adequately addressed with available literature.

Theories of cochlear development (and models of cochlear mechanics in general) have almost completely been formulated from observations in the mid- and high-frequency regions of the cochlea, yet the frequency range below 2 kHz is important to human communication and includes frequencies salient to speech intelligibility. Recent reports of DPOAE phase differences between newborns and adults at low frequencies suggest that studying the apical half of the human cochlea around the time of birth may prove fruitful in elucidating immaturities in cochlear mechanics. The difficult task will be to develop paradigms that effectively target and assay low-frequency features of cochlear function in the challenging newborn population.

4.2 Ear-Canal and Middle-Ear Explanations

Possible sources of OAE immaturity may be better understood through considering how immaturities in ear-canal and middle-ear functioning affect OAEs. Elements of a model were introduced in Sects. 2.2.4–2.2.6, in which the goal for a general hearing experiment was to describe forward transmission of sound from the canal to the cochlea. Measurements of the forward ear-canal transfer function level $L_{FE}$ and reverse ear-canal transfer function level $L_{RE}$ were described. A total forward transfer function level $L_F$ was defined as the sum of $L_{FE}$ and a forward middle-ear transfer function level $L_{FM}$. The following description of bidirectional OAE transmission also introduces a total reverse transfer function level $L_R$ as the sum of $L_{RE}$ and a reverse middle-ear transfer function level $L_{RM}$.

4.2.1 Modeling DPOAE Response Growth

As described in Sect. 3, the largest differences in DPOAE response growth between measurements in young adult and infant ears occurred at a stimulus frequency $f_2$ of 6 kHz. This response growth is shown in Fig. 2.7 as a DPOAE I/O function, in which $f_2/f_1$ was fixed at 1.2, and $L_2$ was always 10 dB below $L_1$. Here, a model is adopted that cochlear mechanics are mature at birth, so that all age differences in DPOAE I/O functions can be accounted for by ear-canal and middle-ear immaturities. The goal was to describe maturational changes in these DPOAE data at 6 kHz as a function of maturational changes in $L_F, L_{FM}, L_R$, and $L_{RM}$, in which maturational
changes in $L_{FE}$ and $L_{RE}$ were independently calculated (see Fig. 2.3). The model calculated relative levels $\Delta L_F$, $\Delta L_{FM}$, $\Delta L_R$, and $\Delta L_{RM}$, respectively, in which the each relative level was defined as the level difference in an infant group minus the adult group. For example, $\Delta L_F$ was the difference in $L_F$ for that infant group minus $L_F$ for the adult group. Even though $L_F$ and other transfer functions could not be directly measured for any age group (because an invasive measurement would be needed; see Sect. 2.2.6), it was possible to calculate the relative total forward transfer function level $\Delta L_F$ based on DPOAE response growth data.

In this approach, horizontal translation of a DPOAE I/O function toward higher stimulus levels corresponded to reduced forward transmission of the stimuli eliciting the DPOAE. Vertical translation of the I/O function toward higher DPOAE levels corresponded to increased reverse transmission of the DPOAE. The amounts of horizontal and vertical translations needed to best align the I/O function at each infant age group (full term, 3, 4, 5, and 6 months) with the “reference” adult I/O function provided $\Delta F_L$ at the $f_1$ stimulus frequency and $\Delta R_L$ at the DPOAE frequency ($2f_1-f_2$) (Abdala and Keefe 2006). The fact that adequate fits were obtained for each infant age group confirmed the hypothesis of cochlear maturity.

Maturational transmission differences were then calculated using data in Fig. 2.3. The relative forward ear-canal transfer function level $\Delta L_{FE}$ was calculated from 0.25 to 8 kHz as the difference in $FE_L$ for each infant group relative to adults; the relative reverse ear-canal transfer function level $\Delta L_{RE}$ was similarly calculated. Only $\Delta L_{FE}$ at $f_1=6$ kHz and $\Delta L_{RE}$ at the DP frequency were used in the model.

Inasmuch as $L_F = L_{FE} + L_{FM}$, then $\Delta L_F = \Delta L_{FE} + \Delta L_{FM}$, so that the relative forward middle-ear transfer function level was calculated by $\Delta L_{FM} = \Delta L_F - \Delta L_{FE}$. These relative forward levels are plotted in the top panel of Fig. 2.11 for each age group according to the mean level and ±1 standard error (SE) of the mean. The ear-canal component $\Delta L_{FE}$ was close to 0 dB for term infants but increased with increasing age. This is a clear sign that immaturities related to ear-canal function and to the absorption and reflection properties of the tympanic membrane have not diminished by age 6 months. Conversely, the middle-ear component $\Delta L_{FM}$ showed 16 dB of attenuation in the term infant that was reduced to 8 dB of attenuation by age 6 months. Almost accidentally, the sum of these two processes represented by $\Delta L_F$ is within ±1 SE of 0 dB, which would appear to suggest maturity in forward transmission at 6 kHz in the absence of this decomposition. In fact, $\Delta L_{FE}$ varied rapidly with frequency between 2 and 8 kHz (because of the rapid fluctuation of $L_{FE}$ for the adult ear response in the top panel of Fig. 2.3), so that the near cancellation at 6 kHz would be unlikely to occur at other frequencies.

By an analogous argument, the relative reverse middle-ear transfer function levels were calculated by $\Delta L_{RM} = \Delta L_R - \Delta L_{RE}$. Each is plotted (mean and SE) in the bottom panel of Fig. 2.11. In contrast to forward transmission, each relative reverse level was largest and positive for term infants, and each tended to decrease with increasing age out to 6 months. A positive $\Delta L_R$ signifies that OAE levels were boosted in infants compared to adults. Most of this effect comes from an increase in $\Delta L_{RM}$, because, as is evident from comparing infant and adult values of $L_{RE}$ in the bottom
panel of Fig. 2.3, $\Delta L_{RE}$ remained close to 0 dB. $\Delta L_{RM}$ was immature in all age groups, decreasing from 9 dB in term infants to 7 dB at age 6 months (Fig. 2.11).

An interesting finding was that the infant middle ear attenuated the stimulus in the forward direction ($\Delta L_{FM} < 0$) but boosted the DPOAE in the reverse direction ($\Delta L_{RM} > 0$). This appears counterintuitive inasmuch as the middle ear might be expected to have an attenuation in both directions or neither. This sign difference is explained in the model by its prediction that $\Delta L_{RM}$ is related to $\Delta L_{FM}$ via the relative

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**Fig. 2.11** Relative ear-canal and middle-ear transfer-function levels corresponding to DPOAEs at $f_2=6$ kHz as functions of infant age. **Top**: Relative forward transfer-function levels. **Bottom**: Relative reverse transfer-function levels [Parts of the figure were originally published in Keefe and Abdala (2007)]
area level (see Fig. 2.2), that is, $\Delta L_{RM} = \Delta L_{FM} - \Delta L_a$. $\Delta L_a$ was always positive and as large as 17 dB in full-term infants, which is the factor producing an overall positive $\Delta L_{RM}$. The small ear-canal area in infants accounts for this boost in reverse transmission. From the perspective of a general hearing experiment (i.e., not concerned with reverse transmission), the relevant finding is that forward transmission was attenuated in the infant ear compared to the adult ear (at least at 6 kHz).

Immaturities in ear-canal and middle-ear function would also affect other OAE types. For example, it would be possible model immaturities in CEOAE and SFOAE responses using a similar approach to that used for DPOAEs. CEOAE levels are larger in infants than in adults, consistent with reverse-transmission effects described above for DPOAEs. However, estimating the relative forward and reverse levels (i.e., $\Delta L_F$ and $\Delta L_R$) from a CEOAE I/O function is more difficult. DPOAE I/O functions are nonmonotonic so that it was possible to translate an infant DPOAE I/O function to best match an adult DPOAE I/O function. CEOAE I/O functions are much more nearly monotonic functions of stimulus level, which would complicate the procedure used for DPOAEs.

### 4.2.2 DPOAE Suppression

A measurement of a DPOAE I/O function implicates ear-canal and middle-ear functioning via forward transmission of stimulus energy at $f_1$ and $f_2$, and reverse transmission at the DP frequency. Measuring the suppression of a DPOAE further implicates ear-canal and middle-ear functioning via forward transmission level differences of each suppressor tone at its suppressor frequency. Given the ability to explain immaturities in DPOAE I/O functions at $f_2 = 6$ kHz in terms of immaturities in the conductive pathway, several studies (Abdala and Keefe 2006; Abdala et al. 2007; Keefe and Abdala 2007) explored whether conductive immaturities also explain immaturities in DPOAE suppression tuning.

As described in Sect.3.6 and shown in Figs. 2.8 and 2.10, DPOAE STCs in infants were most different from STCs in adults at $f_2 = 6$ kHz. For example, the tip-to-tail level reported by Abdala and Keefe (2006) was 27 dB in infants compared to only 15 dB in adults at reference L1–L2 levels of 65–55 dB SPL. As shown in the top panel of Fig. 2.11, the attenuation in forward transmission level ($-\Delta L_F$) was 15 dB in full-term neonates. DPOAE STCs in infants were compared to STCs in adults at probe levels that were reduced by 15 dB, that is, at L1–L2 of 50–40 dB SPL, to calibrate equal cochlear levels based on $\Delta L_F$. The DPOAE STC in adults at this reduced probe level had a tip-to-tail level of 26 dB, nearly equal to that for the infant STC at the reference level.

With immaturities in forward transmission of suppressor tones accounted for, it remained to correct for immaturities in the magnitude of the DPOAE that was being suppressed. The tip of the adult STC at the reduced probe level was about 15 dB below the tip of the infant STC at the reference probe level. The “best fit” in the tip region was obtained by adding 15 dB to the adult STC. This amount was within a
couple dB of the boost in reverse transmission of $\Delta L_{RL} = 13$ in full-term infants (bottom panel of Fig. 2.11). Clearly, at $f_2 = 6$ kHz, immaturities in ear-canal and middle-ear function explained much of the difference in the DPOAE STCs of term infants and adults. This result is consistent with the theory that cochlear function is mature, and ear-canal and middle-ear functions are immature, in the human neonate.

As noted in Sect. 4.1, several frequency-specific components of admittance and reflectance did not correlate strongly with DPOAE STC indices in the first 6 months of life (Abdala et al. 2007). This is not surprising inasmuch as these components did not directly measure the total forward or reverse transmission through the ear canal and middle ear (such a measurement would require invasive techniques as explained in Sect. 2.2). Each component is a measure of conductive function as viewed in a mid-canal location that may not highly correlate with transmission to or from the cochlea.

Even though DPOAE suppression in infants would be influenced by forward transmission at each suppressor frequency, a single measurement of forward transmission at the probe frequency ($f_2$) would not specify the transmission over the range of suppressor frequencies. Suppressor levels are assessed using pressure measurements at the probe, but acoustic standing-wave distributions of ear-canal pressure differ substantially between infants and adults over the frequency range of the DPOAE STC. It would be preferable to measure STCs using a variable that is not influenced by standing waves in the ear canal.

As shown in Fig. 2.11, even though forward transmission ($\Delta L_{FL}$) at 6 kHz was estimated to be nearly adult-like in 6-month-olds, the low-frequency flank of the mean DPOAE STC in Fig. 2.10 remained immature in 6-month-olds. This immaturity has been cited as possible evidence for residual immaturities in cochlear mechanics (Dhar and Abdala 2007; Abdala and Dhar 2010). Maturational differences in forward transmission at any suppressor frequency might produce differences in the resulting DPOAE STC, but data at the suppressor frequencies were not included in the model that predicted $\Delta L_{FL}$ (DPOAE I/O function data would be required at each suppressor frequency). One problem is the presence of age-related differences in ear-canal standing waves at each suppressor frequency, so that the use of sound pressure level can be misleading. It is possible to control partially for these differences by measuring the DPOAE STC using a different acoustic ear-canal variable—absorbed sound power rather than sound pressure.

Keefe and Schairer (2011) described differences between measuring a SFOAE STC as a function of absorbed sound power level compared to SPL. The shape of the SFOAE STC recorded in adult ears at a stimulus frequency of 8 kHz was significantly changed when absorbed power level rather than SPL was used to specify the level of the stimuli generating and suppressing the SFOAE. The underlying causes were the presence of acoustic standing waves in the adult ear canal that produced maximal effects around 4 kHz, and the increased efficiency of absorbing sound energy by the middle ear at frequencies between 2 and 4 kHz compared to lower and higher frequencies up to 8 kHz. The effects of these standing waves in adults are shown for $L_{FL}$ near 4 kHz (see top panel, Fig. 2.3).
Keefe and Abdala (2011) described maturational differences between DPOAE STC measurements as a function of absorbed sound power level compared to measurements based on SPL. These were based on DPOAEs recorded at the same set of stimulus SPLs in full-term infants, infants at age 6 months and adults. The DPOAE STC was first transformed from a criterion SPL at each suppressor frequency to its absorbed power level. Then, it was translated based on the conversion from the stimulus SPLs to the stimulus absorbed power level averaged over the stimulus frequencies \( f_1 \) and \( f_2 \). This STC conversion controlled for maturational differences in the unsuppressed DPOAE generated using fixed stimulus levels across the age groups (stimulus frequencies and levels were the same as for the STC results shown in Fig. 2.10). The resulting suppressor power level difference was defined as the difference between the absorbed-power DPOAE STC (at \( f_2 = 6 \) kHz) producing the criterion decrement of the DPOAE, and this stimulus absorbed power level.

The mean and SE of the suppressor power level differences are plotted in Fig. 2.12. DPOAE STCs were similar across age at nearly all suppressor frequencies, once the SPLs of the stimulus and suppressor tones were converted to absorbed power level (i.e., compare Figs. 2.10 and 2.12). Normalizing the mean suppressor power level difference using the stimulus absorbed power level matched the STC tip levels across age in Fig. 2.12. These results suggest that the cochlear mechanics that underlie DPOAE suppression are substantially mature in full-term infants.
Thus, it appears possible to explain the differences between infants and adults in DPOAE STCs at a probe frequency of 6 kHz in terms of ear-canal and middle-ear immaturities. A particularly important factor is that the $L_{FE}$ has a strong frequency resonance in adults in the octave range below 6 kHz, whereas it is slowly varying in infants (top panel, Fig. 2.3). This is an instructive example of why it might be helpful in future research involving measurements in infant and adult ears to calibrate ear-canal stimuli in terms of absorbed power level rather than SPL. This would also apply in experiments measuring electrophysiological or behavioral responses.

4.2.3 Immaturities in SOAEs

Age differences in prevalence, level, and spectra of SOAEs may also be explained in terms of immaturities in ear-canal and middle-ear function. In terms of the standing wave theory of SOAE generation (see Sect. 4.1.1), SOAEs are influenced in a similar manner by the relative forward and reverse ear-canal transfer function levels $L_{FE}$ and $L_{RE}$ in infants relative to adults. SOAE age differences may be consistent with increased reverse transmission of energy from the cochlea to the ear canal in infants. Increased reverse transmission would boost the SOAE level, thus increasing the likelihood of detection for a fixed noise level. This increased SOAE level would correctly predict an increased prevalence of SOAEs in infants relative to adults. While no forward-transmitted stimulus elicits the SOAE, multiple internal reflections of the SOAE would create a cascade of forward-transmitted signals. Similarly, SOAEs are also influenced by the relative forward and reverse middle-ear transfer-function levels $\Delta L_{FM}$ and $\Delta L_{RM}$. As with other OAE types, SOAE levels are boosted by the smaller ear-canal area and shorter length in infants relative to adults.

4.2.4 Discussion

The findings that immaturities in ear-canal and middle-ear function are sufficient to adequately explain immaturities in DPOAE response growth and DPOAE suppression suggest that they may help explain immaturities in other types of DPOAE responses. The underlying idea as regards forward transmission is that changes in cochlear input level, which are produced by functional immaturities in more peripheral parts of the ear, lead to changes in cochlear output level resulting from the compressively nonlinear response on the basilar membrane. More peripheral immaturities in reverse transmission filter the OAE signal measured in the ear canal and change the strength of any multiple internal reflections.

Whether residual immaturities in the cochlear structures of infants lead to detectable functional differences in OAE responses is a challenging and interesting scientific problem. It will be important in such future experiments to consider the
impact of immaturities in ear-canal and middle-ear function that will otherwise tend to dominate the immaturities observed in OAE responses.

5 Summary

The maturational processes underlying the acoustical and mechanical functioning of the human ear and the structures serving those functions were described in the first part of this chapter. The development of ear-canal and middle-ear structures is not yet complete in the full-term infant, but continues at least up to the onset of puberty. While postnatal growth of these peripheral structures is rapid during the first year of life, more study is needed concerning the postnatal maturation of soft tissue such as the tympanic membrane. The substantial postnatal maturation of the ear canal and middle ear produces functional immaturities in how the ear canal and middle ear receive, absorb, and transmit sound energy to the cochlea. A description of the acoustical–mechanical coupling of power flow through the ear can account for maturational differences in how sound presented in the ear canal is transmitted to the inner ear, based on a detailed examination of experimental data collected in adult ears and in the ears of infants throughout the age span. Understanding the maturational processes in terms of power flow through the peripheral auditory system is important to understanding maturation processes in hearing and in interpreting measurements of physiological responses such as OAEs and auditory brain stem responses.

The second part of this chapter reviewed maturation of cochlear structure and function as explained by measures of otoacoustic emissions. In contrast to outer- and middle-ear anatomy, cochlear structures appear to be substantially completed sometime in the third trimester but the final stages are poorly delineated and little is known about changes in the physical properties of the basilar membrane during the early postnatal period. This section provided a careful examination and prudent interpretation of OAEs recorded in human infants to better understand underlying cochlear mechanics during maturation. All indications from these findings suggest the cochlea of newborns produces robust cochlear amplification, yet age differences were identified in the relative magnitude of DPOAE components and in the DPOAE phase gradient, particularly in the apical half of the newborn cochlea. These results warrant further consideration and study. It is clear from the information presented in this chapter that any such endeavor must take into account the relatively long time course for the maturation of outer- and middle-ear properties and to consider how these factors impact OAE recordings from infants and children.

Though it would have been convenient (for readers, perhaps) to outline a more definitive timeline for the development of human cochlear function and provide more firm interpretations, the complexity of this maturational process and the influence of noncochlear factors such as the conductive system and the medial efferent system make this a formidable task. OAE-based measures provide a glimpse into the human cochlea during the earliest segments of perinatal and postnatal life though
it is clear that they provide this glimpse through a two-way mirror of ear-canal and middle-ear function.

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