

CHAPTER

9

Tympanometry in Clinical Practice

Janet Shanks and Jack Shoet

HISTORY AND DEVELOPMENT OF TYMPANOMETRY

Two “must read” articles on the development of clinical tympanometry are Terkildsen and Thomsen (1959) and Terkildsen and Scott-Nielsen (1960). Their interest in estimating middle-ear pressure and in measuring recruitment with the acoustic reflex had a profound effect on the development of clinical instruments. Each time I read these articles, I am struck first by the incredible amount of information in the articles, and second, by the lack of scientific data to support their conclusions. Most amazing of all, however, is that the principles presented in these two articles have stood up for nearly 50 years, and provide the basis for commercial instruments and tympanometry procedures still in use today.

These articles laid the foundation for the use of hard-walled calibration cavities and the term “equivalent volume of air”, compensation of ear-canal volume, and the selection of a single low-frequency probe tone. You should be surprised to read that the probe tone “frequency of 220 cps was chosen partly at random” (Terkildsen and Scott-Nielsen, 1960, p. 341). A low-frequency probe tone was preferred because the current day microphones were nonlinear at high frequencies and the probe tone level could be increased without eliciting an acoustic reflex. In other words, the selection of a 220 Hz probe tone was made without any consideration of its diagnostic value in evaluating middle-ear function. Finally, these articles also set the precedent for measuring only the magnitude of complex acoustic immittance rather than both magnitude and phase angle. Phase angle measurements were abandoned because the middle ear is so stiffness controlled at 220 Hz that phase angle did not vary considerably in either normal or pathological middle ears.

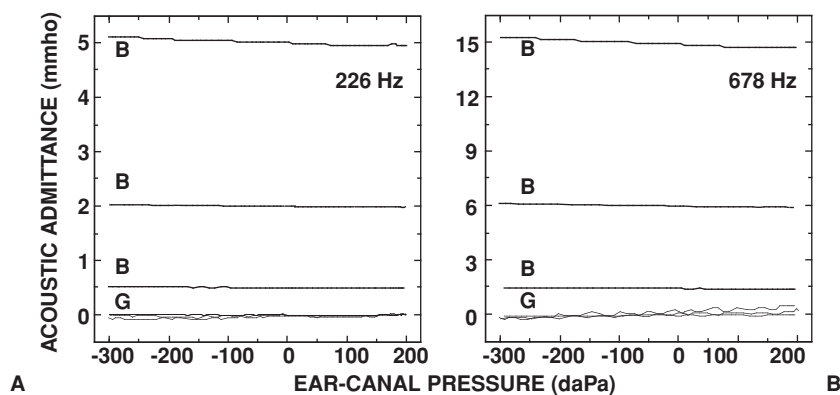
The focus of Terkildsen and Thomsen (1959) was on estimating middle-ear pressure. They adapted an electroacous-

tic impedance instrument to allow for variation in ear-canal pressure over a range of ± 300 mm H₂O and described the first “tympanogram” as a uniform pattern “. . . with an almost symmetrical rise and fall, attaining a maximum at pressures equaling middle ear pressures” (p. 413). They further noted that, “. . . the smallest impedance always corresponded exactly to the zone of maximal subjective perception of the test tone” (p. 413). In other words, the probe tone was the most audible and the tympanogram peaked when the pressure was equal on both sides of the eardrum.

In addition, these authors recognized that although the measure of interest was the acoustic immittance in the plane of the eardrum, for obvious reasons, the measurements had to be made in the ear canal. They introduced the clinical procedure used to compensate for the volume of air enclosed between the probe tip and the eardrum. “Under conditions where the ear drum is under considerable tension, the impedance volume measured is dominated by the volume of the ear canal space itself. With decreasing tension of the ear drum the influence of the middle ear space gradually increases, attaining a maximum under conditions where the pressures in both spaces are identical”. “. . . the difference between highest and lowest impedance volumes as obtained by variations of the pressure, is thought to some extent to indicate the vibrating characteristics of the individual ear drum” (p. 414). The difference between the highest and lowest impedances, therefore, was attributed to the middle-ear system, absent the effects of the ear canal. Here, they laid the basis for estimating ear-canal volume and calculating peak compensated static acoustic admittance.

Instead of reporting tympanograms in acoustic ohms, Terkildsen and Thomsen (1959) reported them in terms of an equivalent volume of air with units in milliliters (ml) or cubic centimeters (cc or cm³). This precedent was followed because at low frequencies such as 220 Hz, the ear functions

FIGURE 9.1 Uncompensated acoustic susceptance (B) and conductance (G) tympanograms recorded at 226 Hz (A) and 678 Hz (B) in calibration cavities with volumes of 0.5, 2.0, and 5.0 cm³.



like a hard-walled cavity of air. Although the use of equivalent volumes is acceptable at low frequencies, its use is not appropriate at high frequencies where the middle ear is no longer stiffness dominated. Instead of simplifying measurements, the term equivalent volume of air continues to be a source of confusion.

Based largely on these early studies, the electroacoustic immittance systems developed in the early 1960's measured only the magnitude of acoustic impedance for a single, low-frequency probe tone of 220 Hz. The first generation of instruments, like the popular Madsen ZO70, did not incorporate an automatic gain control (AGC) circuit to maintain the sound pressure level of the probe tone at a constant level. As a result, the amplitude of the tympanogram, reported in arbitrary units from 1 to 10, was partially dependent on ear-canal volume. The sound pressure level of the probe tone and the height of the tympanogram were higher in small ear canals than in large ear canals *even when the acoustic immittances of both middle ears were identical* (see Shanks, 1984: Figure 4). Not surprisingly, quantifying the height of tympanograms in arbitrary units was not useful clinically.

To circumvent this problem, the next generation of instruments incorporated AGC circuits to maintain a constant probe tone level in ear canals of all sizes. Measurements no longer were reported in arbitrary units but rather in absolute physical units, the acoustic mmho. In 1970, Grason Stadler introduced a new instrument (Model 1720) with two probe-tone frequencies, 220 and 660 Hz, and two admittance components, acoustic susceptance (B_a) and conductance (G_a). Feldman (1976b) used this instrument to collect 220 and 660 Hz tympanograms from ears with a variety of middle-ear pathologies, and clearly demonstrated the advantage of a high-frequency probe tone in evaluating mass related pathologies of the middle ear. Despite findings such as these, high-frequency tympanometry has never become routine.

The latest generation of computer-based instruments (e.g., Virtual, Model 310; Grason Stadler TympStar, Version 2¹; Madsen OTOflex 100) continued to offer multiple frequency probe tones (e.g., 226, 678, and/or 1000 Hz) and

multiple admittance components [e.g., acoustic admittance magnitude (Y_a), phase angle (φ), B_a , and G_a]. These instruments are extremely versatile and have the added convenience of being able to store commonly used tympanometry protocols and several records of patient data. By the end of this chapter, you will hopefully recognize several applications for the under utilized high frequency options on these instruments.

CALIBRATION

ANSI S3.39 requires that three calibration cavities (0.5, 2.0, and 5.0 cm³) be provided with each instrument. Measuring the acoustic admittance of these calibration cavities provides a convenient introduction to tympanometry estimates of ear-canal volume, compensated for ear-canal volume versus uncompensated tympanograms, and flat tympanograms. Figure 9.1 shows acoustic susceptance (B) and conductance (G) tympanograms in the three calibration cavities for 226 Hz (Panel A) and 678 Hz (Panel B) probe tones. For the smallest cavity at 226 Hz, the B and G tympanograms are straight lines at 0.5 and 0 acoustic mmho, respectively. Similarly, for the 2.0 and 5.0 cm³ cavities, G at 226 Hz remains at 0 acoustic mmho and B is equal to the volume of the cavities. Because $G=0$ in these calibration cavities, admittance magnitude (Y) also would be identical to B (see Eq. 8.5) and equal to the volumes of the calibration cavities. At 678 Hz, the results are the same except B is increased by a factor of 3 to 1.5, 6.0, and 15.0 acoustic mmho while G remains at 0 acoustic mmho in all three cavities. You should further notice that the B tympanograms slope slightly upward as pressure decreases. This is expected because the acoustic susceptance (B_a) of an enclosed volume of air increases slightly as the density of air decreases (Beranek, 1954; Lilly and Shanks, 1981).

Hard-walled cavities are used for calibration because they can be constructed as an ideal acoustic element. That is, at sea level, an enclosed cavity of air with certain constraints placed on the radius and length of the cavity can be modeled as pure acoustic compliance (or inversely, stiffness) with negligible mass and resistance up to approximately 1000 Hz (Beranek, 1954; Lilly and Shanks, 1981; Margolis and Smith, 1977; Moller, 1960, 1972; Rabinowitz, 1981; Shanks and Lilly, 1981; Shaw, 1974). The low-frequency probe tone was

¹ Grason Stadler, a division of VIASYS Healthcare, Inc. supported this endeavor by providing test equipment for research by the second author.

increased from 220 to 226 Hz in the 1987 standard because under standard atmospheric conditions, the acoustic admittance of a 1 cm³ cavity is equal to 1 acoustic mmho at 226 Hz (see Lilly and Shanks [1981] for further discussion). This “happy coincidence” discussed in Chapter 8 simplifies calibration.

The dimensions of calibration cavities are more constraining for high-frequency probe tones. The Grason Stadler TymStar, for example, uses all three cavities to calibrate at 226 Hz, uses the 0.5 and the 2.0 cm³ cavities to calibrate at 678 Hz, and uses only the 0.5 cm³ cavity to calibrate at 1000 Hz. A fourth, high-resistance cavity is provided for high-frequency calibration. Although this is an unsealed cavity that cannot hold pressure, measurements in this cavity also are instructive. With the probe inserted in this cavity and the starting pressure set to 0 daPa, high conductance (G_a) with minimal susceptance (B_a) is recorded briefly before the procedure is aborted due to an inadequate seal. This is the opposite B_a to G_a relationship recorded in the three sealed cavities.

The exercise depicted in Figure 9.1 should help to clarify the term acoustic immittance of an equivalent volume of air (in ml or cm³) that is shown on the y-axis of some low-frequency tympanograms. This convention developed because measurements made with early instruments showed that at 220 Hz, the admittance of the middle ear was primarily determined by its stiffness with comparatively low mass and resistance. In other words, the ear practically functioned like a hard-walled cavity, and therefore, the measurements were reported relative to the immittance of an enclosed volume of air. If both panels in Figure 9.1 were replotted in equivalent volumes, the B_a (and Y_a) tympanograms at both 226 and 678 Hz would completely overlap rather than being three times larger at 678 Hz.

As mentioned briefly, the density of air has an effect on the acoustic admittance of a volume of air. Density changes only slightly with changes in ear-canal pressure during tympanometry, but changes significantly with elevation. The tympanograms in Figure 9.1 were recorded at sea level, but these values would not be recorded at higher elevations. For example, at 5000 feet, B_a in the 0.5 cm³ cavity should read 0.61 acoustic mmho at 226 Hz and 1.83 acoustic mmho at 678 Hz (see Lilly and Shanks, 1981 for correction factors).

Although equipment calibration in most Audiology clinics is contracted, calibration should be checked periodically by recording tympanograms on the calibration cavities. Calibration, particularly at high frequencies, is sensitive to debris in the probe device. The probe should be cleaned regularly according to the procedure specified by the manufacturer and should be visually inspected before recording each tympanogram.

CONSTRAINTS OF TYMPANOMETRY

Prior to analyzing tympanograms, Zwislocki's (1976) simple block diagram of the middle ear depicting sound transmis-

sion from the ear canal to the cochlea reminds us of two important constraints. First, acoustic immittance measures the acoustic energy that flows *into* the middle ear, and not what flows *through* the middle ear to the cochlea. Some acoustic energy is lost or absorbed in the decoupled portion of the eardrum and in the ossicular joints, and is not passed on to the cochlea. Tympanometry, therefore, should not be used to make assumptions regarding hearing sensitivity. An ear with atrophic scarring of the eardrum is a prime example of a case where tympanometry can be grossly abnormal with minimal effect on hearing.

Second, when the immittance probe is sealed into the ear canal, measurements are made into a virtual “black box”. The measured input admittance cannot be used to determine which specific middle-ear structure/s in the ear contributed to a change in stiffness or a change in mass. The measurements simply reflect the total contribution from all of the individual elements. Just as a sum of 10 can be derived from adding 2+3+5 or from adding 1+9+0, the same input admittance can be derived from different pathologies. In other words, do not expect a 1:1 correspondence between a specific middle-ear abnormality and a specific tympanogram pattern. In reality, the same pathology can produce several different tympanogram patterns, and conversely, the same tympanogram pattern can arise from different middle-ear pathologies. Middle-ear disease changes along a continuum and not in identical discrete steps for every individual. In the case of otitis media, for example, the middle-ear mucosa can become inflamed and negative middle-ear pressure can develop with or without middle-ear fluid, or the middle-ear cavity can contain a small amount of thin serous fluid or can be completely filled with thick fluid. In all cases, the ear might be classified as having otitis media, but the tympanogram pattern will not be identical in all phases of the disease process. Similarly, a tympanogram can be flat when the eardrum is immobilized by fluid or by a fixed malleus. Expectations from tympanometry must be realistic and must be analyzed in conjunction with all available information including case history, otoscopy, and the magnitude and configuration of the hearing loss.

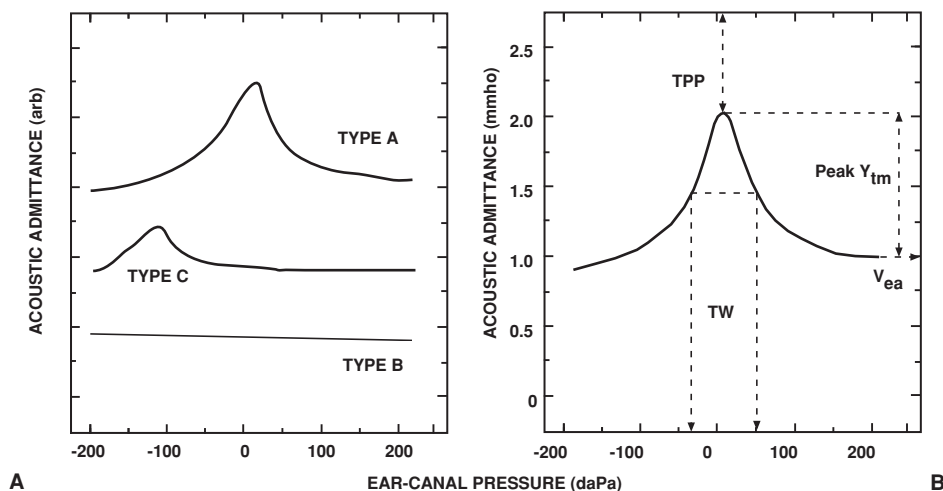
NORMAL 226 HZ ADMITTANCE TYMPANOGRAMS

This section discusses the most commonly used tympanometry procedure that has remained virtually unchanged since 1970. Figure 9.2 demonstrates two methods, one qualitative and one quantitative, for analyzing Y_a tympanograms at 226 Hz.

Qualitative Analysis

The qualitative typing procedure popularized by Jerger is shown in Figure 9.2A (Jerger, 1970; Liden, 1969; Liden et al., 1970, 1974). Notice that no measurement units are shown on the y-axis. Recall that the most commonly used instrument at

FIGURE 9.2 Two methods for analyzing 226 Hz Y_a tympanograms, a qualitative analysis (A) of tympanogram shape in arbitrary units (arb) designated as Type A, B, or C after Jerger (1970) and a quantitative analysis (B) of equivalent ear canal volume (V_{ea} in cm^3), peak static acoustic admittance (peak Y_{tm} in mmho), tympanogram peak pressure (TPP in daPa), and tympanogram width (TW in daPa).



the time, the Madsen ZO70, expressed tympanogram amplitude in arbitrary units (arb); the height of the tympanogram was partly dependent on the volume of the ear canal rather than solely on conditions encountered in the plane of the eardrum. Tympanogram amplitude, therefore, could not be quantified meaningfully, and instead tympanogram shape was categorized into one of three patterns based on simple visual inspection.

A normal tympanogram with a peak near 0 daPa was designated Type A. Subcategories of the Type A were Type A_S for a “shallow” peak (i.e., low admittance) and Type A_D for a “deep” peak (i.e., high admittance) (Jerger et al., 1972). Type A_S tympanograms were associated with otosclerosis and Type A_D tympanograms were associated with ossicular discontinuity or atrophic scarring of the eardrum. The Type C tympanogram was characterized by negative peak pressure, and the Type B tympanogram was flat. Flat tympanograms were recorded from ears with middle-ear effusion (MEE) and eardrum perforation. No amplitude norms were presented to differentiate among Type A, A_S , and A_D tympanograms recorded in arbitrary units, and no pressure norms were presented to differentiate between Type A and C tympanograms. Feldman (1976a) criticized this coding strategy because it implied that everyone was conversant with the typing procedure. To communicate the pertinent information without confusion, he recommended a descriptive analysis of tympanometry peak pressure (e.g., normal, absent, -100 daPa), amplitude (e.g., normal, flaccid, stiff), and shape (e.g., normal, peaked, flat, notched). The typing convention popularized by Jerger, however, is deeply engrained after 35+ years of use and is unlikely to change.

Quantitative Analysis

When AGC circuits were introduced to keep the level of the probe tone in the ear canal constant, tympanograms could be measured in absolute units and quantified. Figure 9.2B shows that tympanogram shape can be precisely described by four numbers.

EAR-CANAL VOLUME

The goal in tympanometry is to measure the acoustic admittance of the middle ear. The probe device, however, cannot be placed at the eardrum but instead must be sealed in the ear canal. The admittance measured at the probe tip (Y_a), then, is the sum of the admittance of the ear-canal volume (Y_{ec}) plus the admittance in the plane of the tympanic membrane (Y_{tm}). If Y_{ec} can be measured independently of the middle ear, then Y_{tm} can be calculated.

Recall from the previous discussion that Terkildsen and Thomsen (1959) proposed that an ear-canal pressure of 200 daPa places the eardrum and middle ear under “considerable tension”, and theoretically, all of the energy of the probe tone is reflected at the surface of the eardrum. In other words, Y_{tm} at 200 daPa is 0 acoustic mmho, and the admittance measured at the probe tip is attributed solely to the ear canal, i.e., $Y_a = Y_{ec}$. In Figure 9.2B, Y_a at 200 daPa is 1.0 acoustic mmho for an ear-canal volume (V_{ea}) of 1.0 cm^3 . As the volume of the ear canal changes, the Y_a tympanogram simply shifts higher or lower on the y-axis without altering the shape of the tympanogram. This is the primary advantage of measuring acoustic-admittance components rather than acoustic-impedance tympanograms. Acoustic-impedance tympanograms not only shift along the y-axis, but also change shape when ear-canal volume changes (Shanks and Lilly, 1981). In other words, the ear canal has a linear effect on admittance tympanograms but a non-linear effect on impedance tympanograms. The interested reader is referred to Lilly (1972a), Margolis (1981), and Van Camp et al. (1986) for a discussion of series versus parallel acoustic admittances and impedances that accounts for this difference.

Several observations reveal that tympanometry overestimates ear-canal volume by as much as 24% to 39% (Margolis and Smith, 1977; Moller, 1965; Rabinowitz, 1981; Shanks and Lilly, 1981; Vanpeperstraete et al., 1979). Listen to the level of the probe tone as you record a tympanogram from your own ear. Although the probe tone is noticeably softer at both extreme positive and negative pressures, the tone is always

audible. If an ear canal pressure of 200 daPa were enough to decrease Y_{tm} to 0 acoustic mmho, the probe tone would not be audible and Y_a at 200 daPa would be the lowest point on the tympanogram. V_{ea} calculated from the negative tail of a 226 Hz tympanogram, however, is up to 0.2 cm³ lower than the positive tail. The asymmetry has been attributed to eardrum movement, distension of the cartilaginous portion of the ear canal, movement of the probe tip, and residual middle-ear effects (Elner et al., 1971; Margolis and Popelka, 1975; Van Camp et al., 1986; Vanpeperstraete, et al., 1979).

Although 200 daPa does not produce the most accurate estimate of V_{ea} , it is historically the most commonly used pressure. Even more important than selecting the most precise procedure is utilizing a consistent procedure so that estimates of V_{ea} can be compared across clinics and patients. With the advent of computer-based instruments, estimating V_{ea} at 200 daPa has become more practical. The software automatically estimates V_{ea} at the selected starting pressure. Descending pressure changes (+/-) are preferred because they result in fewer seal problems in neonates (Holte et al., 1991; Sprague et al., 1985) and produce more consistent tym-

panogram morphology, particularly when high-frequency probe signals are used (Margolis et al., 1985; Wilson et al., 1984). If a descending pressure sweep is desired, then V_{ea} automatically will be estimated at a positive starting pressure.

The most important application of V_{ea} is to differentiate between intact and perforated eardrums or between blocked and functioning tympanostomy tubes (TT). Table 9.1 shows normal mean and 90% ranges for the measures depicted in Figure 9.2B. Unless otherwise indicated, the following recording procedures were used: V_{ea} estimated at 200 daPa from Y226 Hz tympanograms, +/- pressure direction, and a pump speed of 200 daPa/s near the peak. V_{ea} changes both with gender and age. V_{ea} is smaller in females than in males at all ages and averages a difference of 0.2 cm³ in adulthood (Haapaniemi, 1996; Margolis and Heller, 1987; Roup et al., 1998; Shahnaz and Davies, 2006; Shanks et al., 1992; Wan and Wong, 2002; Wiley et al., 1996). In view of the large range of normal variability, separate norms are not needed for males and females. Different norms, however, are required for children and adults. Table 9.1 shows that V_{ea} increases from an average of 0.6 cm³ for children less than

TABLE 9.1 226 Hz tympanometry norms from several large scale studies^a

Study	No. of		Age (years)	V_{ea} (cm ³)		Y_{tm} (mmho)		TW (daPa)	
	(years)	Sex		Mean	90% Range	Mean	90% Range	Mean	90% Range
Shanks et al., 1992	668	B	0.15–6.7	0.58	0.3–0.9				
Roush et al. 1995	1827	B	.5–2.5			0.45	0.2–0.7	148	102–204
Margolis & Heller 1987	47	M	2.8–5.8	0.8		0.49		105	
	42	F	2.8–5.8	0.7		0.52		95	
	92	B	2.8–5.8	0.75	0.42–0.97	0.5	0.22–0.81	100	59–151
Koebseil & Margolis 1986 ^b	88	B	3.7–5.8					133	80–200
Nozza 1992 ^c , 1994 ^{d,e}	130	B	3–16	0.9	0.6–1.35	0.78	0.40–1.39	104	60–168
Haapaniemi 1996	942	B	6–15	0.8	0.5–1.2	0.5	0.3–1.1		
Hanks & Rose 1993	316	B	6–15	1	0.6–1.5	0.7	0.3–1.5		
Shanks 1985	63	M	Adult	1.59	0.75–2.0				
Margolis & Heller 1987 ^e	49	M	19–61	1.14		0.77		79	
	38	F	19–61	0.93		0.65		74	
	87	B	19–61	1.05	0.63–1.46	0.72	0.27–1.38	77	51–114
Wiley et al. 1996	825	M	48–90	1.49	1.0–2.2	0.72	0.2–1.6	73	35–125
	1322	F	48–90	1.28	0.9–1.9	0.62	0.2–1.4	76	40–120
	2147	B	48–90	1.36	0.9–2.0	0.66	0.2–1.5	75	35–125
Holte 1996	136	B	20–90			0.84	0.3–1.8	84	38–141

V_{ea} , ear canal volume; Y_{tm} , admittance in the plane of the tympanic membrane; TW, tympanogram width; B, both male and female; M, male; F, female.

^a Unless indicated, V_{ea} was estimated at 200 daPa from tympanograms recorded using descending pressure changes at a rate of 200 daPa/s.

^b $\min V_{ea}$

^c 300 V_{ea}

^d 400 V_{ea}

^e Handheld probe

7 years (Shanks et al., 1992) to 0.8 cm^3 between 6 and 15 years (Haapaniemi, 1996) to 1.4 cm^3 in adulthood (Wiley et al., 1996).

When the eardrum is perforated, V_{ea} is comprised of the ear canal plus the middle-ear space, antrum, and the mastoid air cell system. Middle-ear volume estimates in patients undergoing stapes mobilization (Zwislocki, 1962) and in cadaver temporal bones (Molvaer et al., 1978) averaged 6.5 to 8.7 cm^3 . These large volumes are due predominantly to the mastoid air cell system, which is minimal in newborns and grows rapidly in 2 to 7 year olds (Bentler, 1989; Dolan, 1979; Eby and Nadol, 1986). The normal mastoid is not fully aerated in girls until 10 to 15 years and in boys until 11 to 19 years (Ars, 1989; Diamant, 1965; Dolan, 1979; Eby and Nadol, 1986; O'Donoghue et al., 1986; O'Tuama and Swanson, 1986; Rubensohn, 1965). The relevance of the size of the mastoid air cell system and Eustachian-tube function has been the subject of endless research. Debate continues on whether a small mastoid air cell system is the cause or the result of chronic middle-ear disease. Evidence from animal studies does suggest that chronic middle-ear disease inhibits pneumatization, particularly when it occurs at a very young age (Ikarashi and Nakano, 1988). Andreasson (1977) also showed that longstanding chronic otitis media (COM) with recurrent drainage reduced the volume by shutting off the mastoid air cell system. This is supported by a comparison of volume estimates from ears with perforated eardrums due to COM versus trauma. Lindeman and Holmquist (1982) and Andreasson (1977) reported volumes of 9.7 versus 3.3 cm^3 for perforated eardrums due to trauma versus COM, respectively.

Although several investigators have reported normal ranges for V_{ea} , few have reported V_{ea} in ears with known perforations or patent tympanostomy tubes (TT). Shanks et al. (1992) compared V_{ea} before and after placement of TT in 668 ears of children between 6 weeks and 6.7 years. Average V_{ea} increased from 0.6 cm^3 preoperatively to 2.3 cm^3 6 to 7 weeks after placement of the TT. In addition, post-operative V_{ea} was significantly larger in males than in females and increased as a function of age for both males and females, reflecting continued development of the mastoid air cell system in children under seven years old. The range in post-operative V_{ea} was large and often exceeded the 7 cm^3 recording limits of the equipment. Comparing V_{ea} in the two ears, particularly in cases where V_{ea} falls at the upper end of the normal range, also can be helpful in identifying eardrum perforation. For children under 7 years of age, Shanks et al. (1992) suggested criteria of $V_{\text{ea}} > 1.0 \text{ cm}^3$ or a pre/post difference $\geq 0.4 \text{ cm}^3$. Wilber and Feldman (1976) suggested V_{ea} exceeding 1.5 to 2.0 cm^3 in children or a difference between ears $> 0.5 \text{ cm}^3$ is consistent with eardrum perforation. In adult males, $V_{\text{ea}} > 2.5 \text{ cm}^3$ is consistent with a perforated eardrum (Shanks, 1985).

V_{ea} is an under-utilized measure. Although several applications have been suggested, many clinicians do not even obtain tympanograms in ears with eardrum perforation or

TT. Studies that have monitored V_{ea} following placement of TT have found a gradual increase in volume coincident with resolution of fluid and mucosal edema in the middle ear and with growth of the mastoid air cell system in young children (Sederberg-Olsen et al., 1983; Tashima et al., 1986). Other investigators have noted that recurrence of secretory otitis media following extrusion of TT and failed tympanoplasty are more likely to occur in ears with small mastoid air cell systems (Andreasson and Harris, 1979; Diamant, 1965; Holmquist, 1970; Holmquist and Bergstrom, 1977; Jackler and Schindler, 1984; Sederberg-Olsen et al., 1983). An inverse relationship between the size of the mastoid air cell system and risk of otitis media in adults or risk of barotrauma in scuba divers also has been reported (Holmquist, 1970; Sade and Fuchs, 1996; Uzun et al., 2002).

Shanks (1985) cautioned that although $V_{\text{ea}} > 2.5 \text{ cm}^3$ clearly is indicative of a perforated eardrum in adults, a volume $< 2.5 \text{ cm}^3$ does not necessarily rule out a perforated eardrum. V_{ea} was measured in 24 adult males scheduled for tympanoplasty to repair a perforated eardrum. Half of the patients had normal middle ears and half had cholesteatoma, granulation tissue, or contracted, sclerotic mastoid air cells. Only 1 of 12 patients with surgically confirmed normal middle ears had a $V_{\text{ea}} < 2.5 \text{ cm}^3$, whereas 6 of the 12 patients in the abnormal group had $V_{\text{ea}} < 2.5 \text{ cm}^3$. A small V_{ea} with a known eardrum perforation indicates the need for more frequent post-surgical follow up.

TYMPANOGRAM PEAK PRESSURE

As the ear-canal pressure is decreased from 200 daPa, more and more of the acoustic energy of the probe tone flows into the middle ear. Energy flow into the middle ear (and the loudness of the probe tone) reaches a maximum when the pressures on both sides of the eardrum are equal. The pressure that coincides with peak admittance, then, provides an estimate of middle-ear pressure. In the normal ear, atmospheric or ambient pressure (0 daPa) is maintained within the middle-ear space by the periodic opening of the Eustachian tube during swallowing. The tympanogram in Figure 9.2B has a normal tympanogram peak pressure (TPP) of 5 daPa and falls well within the normal range of ± 50 daPa. In contrast, when the Eustachian tube does not open properly, negative pressure builds up within the middle-ear space and MEE sometimes develops. In addition to this popular ex vacuo theory proposed by Politzer in the 19th century, several other mechanisms have been identified that contribute to pressure fluctuations in the middle ear. See Sade and Amos (1997) and Margolis and Hunter (1999) for a review of these mechanisms.

TPP can overestimate middle-ear pressure by 30 to 70 daPa, particularly in ears with small middle-ear volumes or highly compliant eardrums (Eliachar and Northern, 1974; Flisberg et al., 1963; Renvall and Holmquist, 1976). Middle-ear pressure also is slightly overestimated due to hysteresis or lag effects related both to the pressure transducer and to

the viscoelastic properties of the eardrum and middle ear (Decraemer et al., 1984; Van Camp et al., 1986). Both effects are on the order of 10 to 30 daPa and are in the direction of the pressure change. That is, TPP is shifted more positive for ascending (−/+) pressure changes and more negative for descending (+/−) pressure changes. Some of the new computer-based instruments employ an “x-axis offset” on the order of 25 daPa to correct for instrument hysteresis at fast pump speeds.

Aside from small inaccuracies in equating TPP with middle-ear pressure, extreme deviations in TPP from 0 daPa do reflect true changes in middle-ear pressure. The clinical relevance of negative middle-ear pressure, however, has changed over the years. The 1979 ASHA guideline for acoustic-immittance screening recommended medical referral for TPP in excess of −200 daPa. Many of the children referred for medical treatment, however, had no evidence of middle-ear pathology (Lous, 1982; Roush and Tait, 1985). Middle-ear pressure has been found to fluctuate greatly in children and is no longer considered to be an indication of significant disease (Fiellau-Nikolajsen, 1983; Haughton, 1977; Lildholdt, 1980; Margolis and Heller, 1987; Nozza et al., 1994; Paradise et al., 1976). In the absence of abnormal findings from other tympanometry measures, otoscopy, hearing test, or case history, medical referral on the basis of TPP alone has led to a high over-referral rate and is no longer recommended. Children with high negative pressure, however, are more likely than those with normal TPP to develop MEE and should be monitored closely (Antonio et al., 2002).

Positive TPP greater than 50 daPa is recorded in approximately 1.7% of patients with middle-ear complaints and has been associated with acute otitis media (Ostergard and Carter, 1981). Marked positive TPP also is recorded in ears with pinhole perforations of the eardrum (Fowler and Shanks, 2002; Kessler et al., 1998; Kobayashi and Okitsu, 1986; Kobayashi et al., 1987). To differentiate between the two conditions, tympanograms with positive TPP should be recorded with descending pressure changes and then again with ascending changes. In an ear with a pinhole perforation, TPP from the two pressure sweeps can differ by several hundred daPa in comparison with an expected difference of less than 30 daPa in a normal ear.

PEAK COMPENSATED STATIC ACOUSTIC ADMITTANCE

The next calculation shown in Figure 9.2B is peak compensated static acoustic admittance (peak Y_{tm}). Peak Y_{tm} is the difference in admittance between the peak and 200 daPa. “Compensated” implies that the effects of the ear-canal volume have been removed from the measurement. Most new instruments have the option of recording tympanograms already corrected for ear-canal volume. To do so, the baseline function is turned on, and the tympanogram is zeroed at the selected starting pressure. If the tympanogram in Figure 9.2B had been recorded with the baseline on, the tympanogram would have been shifted down to 0 acoustic mmho at 200

daPa, and the peak Y_{tm} of 1 acoustic mmho could have been read directly from the y-axis.

Mean and 90% ranges for Y_{tm} are shown in Table 9.1. It has become commonplace to report 90% ranges rather than standard deviations or confidence intervals because peak Y_{tm} values are positively skewed (Shahnaz and Polka, 1997; Wilson et al., 1981; Zwislocki and Feldman, 1970). The values can be normalized by performing a logarithmic transformation or by simply reporting the 90% range. Peak Y_{tm} below the 5th percentile indicates an abnormally stiff middle ear. In children under seven years of age, the most common cause is MEE. Examples of low admittance in adults include MEE, otosclerosis, a thickened eardrum, and malleus fixation. High peak Y_{tm} is recorded from ears with eardrum pathology (e.g., atrophic scarring or tympanosclerotic plaques), ossicular replacement prostheses, or ossicular discontinuity. Abnormally high peak Y_{tm} is an indication for medical referral only in the presence of a significant conductive/mixed hearing loss.

The clinical value of peak Y_{tm} has been controversial since tympanometry became routine. The primary criticisms are high normal variability and significant overlap with pathological middle ears. Using consistent recording parameters can help to reduce normal variability. For example, Y_{tm} is greatly affected by the ear-canal pressure used to estimate V_{ea} ; peak Y_{tm} is lower when V_{ea} is estimated at 200 daPa than at −400 daPa. Because this is such an important variable, using the ANSI (1987) convention of specifying the ear-canal pressure used to calculate Y_{tm} is recommended, e.g., 200 Y_{tm} versus −400 Y_{tm} . The rate and direction of pressure change, and the number of consecutive pressure sweeps, also affect peak Y_{tm} . In general, Y_{tm} will increase slightly for ascending versus descending pressure sweeps, as the rate of pressure change increases, and as the number of consecutive tympanogram sweeps increases. These procedural variables have a greater effect on high frequency tympanometry and are discussed in more detail later.

TYMPANOGRAM WIDTH

Brooks (1968) was the first to suggest a measure that describes the sharpness of the tympanogram peak. This calculation was popularized when it was observed that tympanograms frequently were broadly rounded in ears with MEE (Brooks, 1969; Fiellau-Nikolajsen, 1983; Haughton, 1977; Paradise et al., 1976). Three measures of the steepness of the tympanogram peak have been suggested over the years (Brooks, 1969; de Jonge, 1986; Paradise et al., 1976). The calculation of tympanogram width (TW) proposed by de Jonge is easy to calculate and is the most widely used. TW demonstrated in Figure 9.2B is the pressure interval (in daPa) encompassing one half peak Y_{tm} . Koebshell and Margolis (1986) confirmed de Jonge’s findings. They compared eight different measures of tympanogram slope in 3 to 6 year olds. TW was the preferred measure because it had a narrow normal distribution that was independent of pump speed and a low correlation

with peak Y_{tm} that provided complementary rather than redundant information about the middle-ear transmission system. Of the four calculations depicted in Figure 9.2B, TW has been the single most valuable measure in identifying ears with MEE (Fiellau-Nikolajsen, 1983; Haughton, 1977; Nozza et al., 1994).

Effects of Gender, Age, and Race

Some investigators have reported slightly lower peak Y_{tm} and broader TW in females versus males (Jerger et al., 1972; Roup et al., 1998; Wiley et al., 1996; Zwislocki and Feldman, 1970), whereas others have found no gender effect (Holte, 1996; Margolis and Goycoolea, 1993; Margolis and Heller, 1987; Wan and Wong, 2002). Even if present, gender effects are clinically insignificant and do not warrant the added inconvenience of separate norms for males and females.

The effect of race on acoustic admittance measures recently has been investigated. Two studies comparing Caucasian and Chinese adults concluded that a decrease in peak Y_{tm} and an increase in TW in Chinese adults could be attributed to a smaller body size (Shahnaz and Davies, 2006; Wan and Wong, 2002). Different norms, therefore, may be required for Chinese children enrolled in screening programs. Wan and Wong (2002) pointed out that 48% of Chinese children failed tympanometry screening when norms developed on Caucasians were used.

Countless studies also have focused on the effect of age on peak Y_{tm} and TW (Blood and Greenberg, 1977; Holte, 1996; Jerger et al., 1972; Margolis and Heller, 1987; Wiley et al., 1996). Although some small age differences have been reported for adults, any clinically significant differences appear to be masked by the large range of normal variability. Convincing age effects in infants and small children, however, have resulted in separate norms for infants (ASHA, 1997). The ASHA recommendation is based on the data of Roush et al. (1995) who measured broader TW and lower peak Y_{tm} in children less than 1 year of age. They attributed the difference to residual effects of otitis media so prevalent in that age group. DeChicchis et al. (2000) similarly reported a systematic increase in peak Y_{tm} and decrease in TW with increasing age from 6 months to 5 years of age, with the biggest changes occurring during the first 3 years. These authors attributed the differences to anatomic and physiologic changes in the developing ear.

Screening For Middle Ear Effusion

Despite the high prevalence of MEE in children, particularly during the winter months, most cases of MEE are transient and do not necessitate medical intervention (Antonio et al., 2002; Bluestone, 2004; Fiellau-Nikolajsen, 1983; Tos and Poulsen, 1980). For example, Bluestone (2004) reported that 80% of ears with MEE resolved within two months without any medical treatment. A small number of cases, however, do become chronic and require medical management.

Differentiating between these two populations is the challenge facing programs that screen for middle-ear disease and is one reason why the ASHA guideline has undergone three revisions.

The goal of the initial ASHA (1979) screening guideline was to identify children with MEE. Immediate medical referral was recommended for children with flat tympanograms or $TPP < -200$ daPa with an absent 1000 Hz acoustic reflex (AR). By the time many of these children received medical follow up, the MEE had resolved, and the screening protocol was criticized for unacceptably high over-referral rates (Lous, 1982; Roush and Tait, 1985).

A major goal of the revised ASHA (1990) guideline was to decrease the high over-referral rate by eliminating medical referral for transient, self-resolving, secretory otitis media. Both TPP and the AR were dropped from the revised guideline. As previously discussed, TPP fluctuates widely and is a poor determinant of MEE, and the AR can be absent for reasons other than middle-ear disease (e.g., sensorineural hearing loss, a poor seal, or excessive baseline noise). These two admittance measures were replaced with the following three quantitative measures: $200Y_{tm}$, V_{ea} , and TW. A second major change to the screening protocol was the addition of the following three components: history of otalgia (ear pain) or otorrhea (ear discharge); visual inspection for structural defects; and screening audiometry. Rescreening in 4–6 weeks was recommended when any of the tympanometry measures was abnormal in an attempt to exclude self-resolving cases of MEE. Immediate medical referral without conducting tympanometry was recommended only for previously undetected/untreated structural defects, perforated eardrum, otalgia, or otorrhea.

One problem encountered at the time of the revision was that large scale, quantitative tympanometry studies had been conducted on children with normal middle ears but not with confirmed middle-ear disease. Publication of the ASHA (1990) screening guideline was the catalyst for a series of studies on the sensitivity and specificity of various tympanometry measures in identifying children with confirmed MEE (Nozza et al., 1992, 1994; Roush et al., 1992; Roush et al., 1995; Silman et al., 1992; Silverman and Silman, 1995).

Perhaps the most important finding of the Nozza et al. (1992, 1994) studies that compared tympanometry with myringotomy results was that there are not two but three distinct populations encountered when screening for middle-ear disease: normal middle ears from the general population, ears with MEE, and ears without MEE but with a history of chronic or recurrent otitis media. The pass-fail criteria are different for each population. When the interim norms from the general population were applied to ears with a history of chronic or recurrent MEE, the false-positive rate increased from 5% to 75% (Nozza et al., 1994). In comparison with the general population, children with a history of recurrent otitis media with effusion have lower Y_{tm} and wider TW even when the middle ears were effusion free. $TW > 275$ daPa was the single best (81% sensitivity and 82% specificity)

TABLE 9.2 ASHA (1997) and AAA (1997) screening criteria for abnormal V_{ea} , Y_{tm} , and TW in children from 4 months to 7 years of age

	Study	Age	V_{ea} (cm ³)	Y_{tm} (mmho)	TW (daPa)
ASHA (1997)	Roush et al., 1995	4–12 mos		<0.2	>235
	Nozza et al., 1992; 1994	1–7 yrs		<0.3	>200
	Shanks et al., 1992	1–7 yrs	>1.0		
AAA (1997)		3–7 yrs		<0.2	>250

ASHA, American Speech-Language-Hearing Association; AAA, American Academy of Audiology; V_{ea} , ear canal volume; Y_{tm} , admittance in the plane of the tympanic membrane; TW, tympanogram width.

tympanometry measure to differentiate between ears with and without MEE when a history of chronic or recurrent otitis media was present, followed by peak Y_{tm} , the AR, and TPP.

Silman et al. (1992) tested the ASHA (1990) tympanometry criteria using an experienced otoscopist as the gold standard. The ultimate “gold standard” for studies on MEE is myringotomy and aspiration, although this is not always practical. Otoscopy and surgical findings agree 79% to 86% of the time (Paradise et al., 1976; Nozza et al., 1994). Otoscopy misses few ears with MEE, but has a slightly higher false-positive rate.

Silman et al. (1992) reported that the ASHA (1990) protocol had 81.5% sensitivity and 79% specificity, and that performance improved to 90% sensitivity and 92.5% specificity when TPP and the ipsilateral AR were put back into the screening protocol. There still is no consensus on the value of including TPP and AR in screening programs. Nozza et al. (1992) and Silman et al. (1992) reported good performance of these two measures, whereas other investigators reported excessive false-positive rates (Roush and Tait, 1985). Although the AR performed well, its use in screening is limited because 28% of the normal population either failed the test or could not be tested (Nozza et al., 1992).

An interesting finding of the Silman et al. (1992) study was that when MEE resulted in failed hearing screening, all of the tympanometry measures were effective in identifying MEE. MEE, however, does not always result in significant hearing loss. Estimates of failed hearing screenings with verified MEE have ranged from 35% to 80% (Lyons et al., 2004; Roush and Tait, 1985; Silman et al., 1992). The condition of the middle ear changes along a continuum from a normal ear to a completely fluid-filled ear with significant hearing loss. All tympanometry screening measures were effective in correctly identifying the two extremes, but were less successful in identifying ears falling in the middle. Ears with a history of chronic or recurrent otitis media but without MEE also fall into this challenging middle range and tend to have high false-positive rates for both tympanometry and otoscopy (Nozza et al., 1992, 1994; Roush et al., 1995; Silverman and Silman, 1995). These same ears probably account for the 15–27% incidence of dry taps in ears undergoing myringotomy (Saeed et al., 2004; Watters et al., 1997).

In response to the growing database of sensitivity data, the ASHA guideline for middle-ear screening underwent another revision in 1997. The targeted population is children under seven years of age with chronic or recurrent middle-ear effusion with the potential to cause significant medical and developmental consequences. The primary change is that the tympanometry criteria have been modified to reflect the age and population specific data now available. The new ASHA (1997) and the AAA (1997) criteria are shown in Table 9.2. The AAA criteria are more conservative than the ASHA criteria in minimizing false-positive rates in 3 to 7 year olds. The consensus reflected in all the guidelines is that a test-battery approach is better than any single measure in identifying children requiring medical intervention. As such, the guidelines are changing more from screening to identification guidelines. The guidelines also recognize that the criteria used to identify MEE are highly dependent on the population being tested and recommend that program administrators continually monitor and adjust referral criteria for acceptable pass-fail rates.

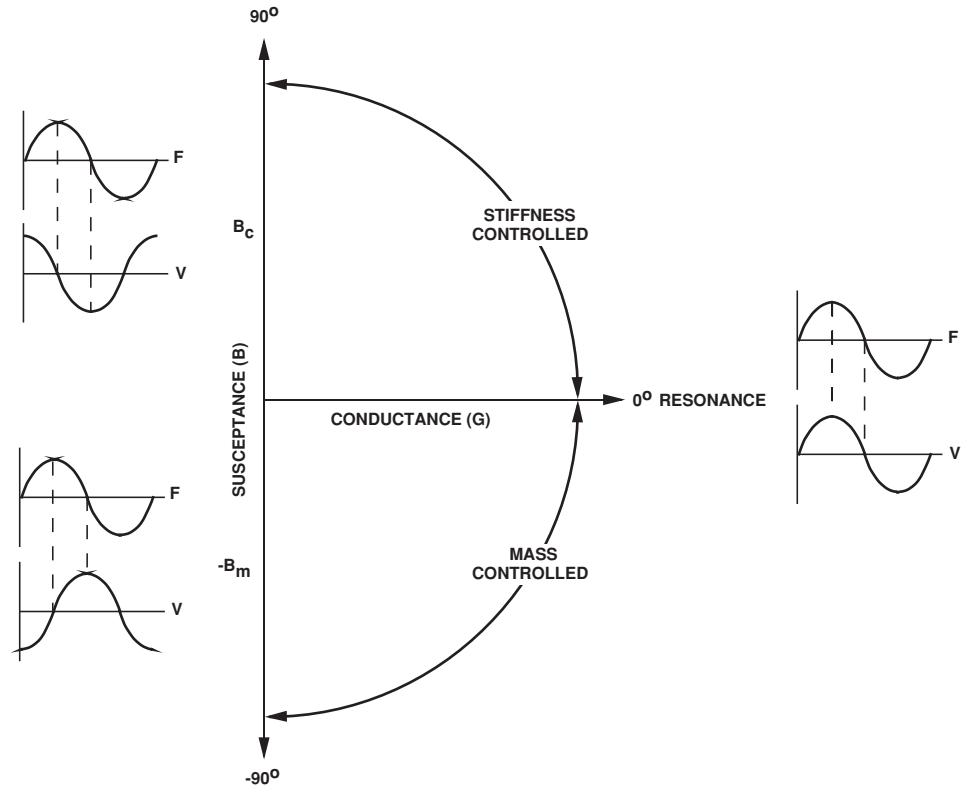
REVIEW OF ACOUSTIC IMMITTANCE PRINCIPLES

The remainder of the chapter focuses on multiple-frequency tympanometry (MFT). The principles discussed in Chapter 8 will be reviewed briefly. Additional recommended readings in this area include an excellent chapter by Margolis (1981) and chapters by Lilly (1972a,b) and Van Camp et al. (1986). For students who would like a more basic review of acoustic immittance measurements with an extensive bibliography, a book by Wiley and Fowler (1997) is recommended.

Recall from the preceding chapter that three elements contribute to admittance, spring (also termed compliance or inversely stiffness), mass, and resistance. When an identical sinusoidal force (F) is applied to these three elements, each responds with a unique velocity (V). These unique force velocity relationships are depicted in Figure 9.3 (adapted from Fowler and Shanks, 2002).

An element with pure resistance dissipates or absorbs energy due to friction. The velocity of a purely resistive element is in phase with the applied force. In admittance

FIGURE 9.3 Force (F) – velocity (V) relationships for compliant susceptance (B_c), mass susceptance ($-B_m$), and conductance (G) depicted on x-y coordinates. Total susceptance (B) determines whether the middle ear transmission system is stiffness controlled ($>0^\circ$), mass controlled ($<0^\circ$), or at resonance (0°) (after Fowler and Shanks, 2002).



terminology, the “real” or in phase component of complex acoustic admittance is called acoustic conductance (G_a) and is represented along the x-axis in Figure 9.3. Resistance can never be negative, so only the positive x-axis is shown in the figure.

The other two elements, a spring (or compliant) element and a mass element, both store energy, and their responses are out of phase with the applied force. The velocity of a spring or compliance element *leads* the applied force by 90° whereas the velocity of a mass element *lags* force by 90° . In other words, for the same applied force, the velocities of spring and mass elements are 180° out of phase, and are similarly represented at 90° and -90° on the y-axis. Compliant or positive susceptance (B_c) is the admittance offered by a compliant element, and mass or negative susceptance ($-B_m$) is the admittance offered by a mass element.

A system like the ear, that contains both compliant and mass susceptance, functions like a system with a single element whose total susceptance (B_a) is equal to the algebraic sum of the two out-of-phase components. All two-component admittance instruments measure total susceptance. If compliant susceptance (B_c) is larger than mass susceptance ($-B_m$), total susceptance is positive. The ear is described as stiffness controlled, and the admittance vector will be in the upper quadrant of Figure 9.3. Conversely, if mass susceptance ($-B_m$) is larger than compliant susceptance (B_c), total susceptance is negative. The ear is described as mass controlled, and the admittance vector will be in the lower quadrant of Figure 9.3. When compliant and mass susceptance are equal, total susceptance is 0 acoustic mmho

and the middle ear is in resonance. The resonant frequency (f_0) of the normal middle ear is between 800 and 1200 Hz. Below f_0 , the ear is described as stiffness controlled, and above f_0 , the ear is described as mass controlled.

If pathology such as otosclerosis increases the stiffness of the middle-ear transmission system, the ear remains stiffness controlled at higher than normal frequencies and f_0 increases. Conversely, if pathology such as ossicular discontinuity increases the mass (or decreases the stiffness) of the middle-ear transmission system, the ear becomes mass controlled at a lower than normal frequency and f_0 decreases. These effects frequently are reflected in the audiogram. An increase in stiffness decreases transmission for low frequencies, and the audiogram demonstrates a stiffness tilt. Conversely, an increase in mass decreases transmission for high frequencies, and the audiogram demonstrates a mass tilt.

NORMAL 226 AND 678 HZ TYMPANOGRAMS

Figure 9.4 shows uncompensated acoustic admittance (Y_a), susceptance (B_a), and conductance (G_a) tympanograms at 226 Hz (top) and 678 Hz (bottom) from a normal 40-year-old male. Peak compensated static acoustic admittance is plotted in both polar and rectangular formats to the right of the tympanograms. First, calculate peak $200B_{tm}$ and $200G_{tm}$, i.e., subtract the admittance values at 200 daPa from the respective peak values. At 226 Hz, B_{tm} is 0.57

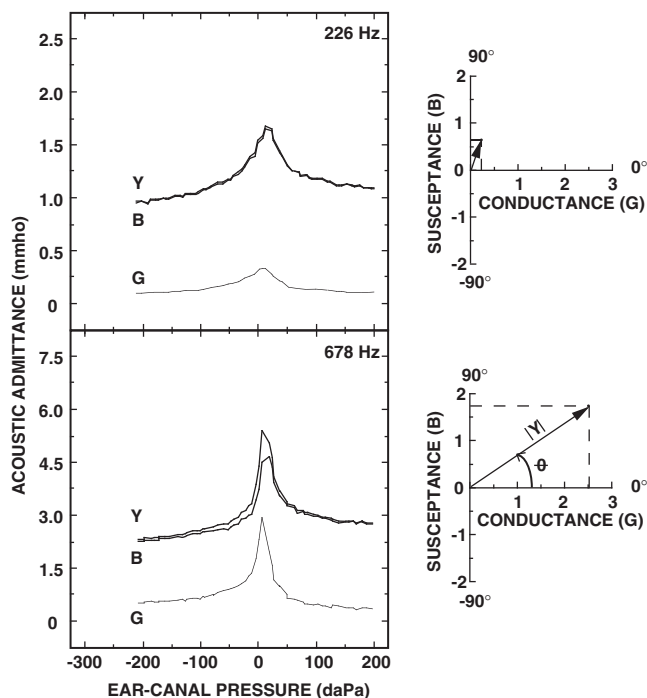


FIGURE 9.4 Normal uncompensated acoustic admittance (Y), susceptance (B), and conductance (G) tympanograms at 226 Hz (top panel) and 678 Hz (bottom panel) with corresponding peak compensated static acoustic admittance plotted in both rectangular (susceptance [B] and conductance [G]) and polar (admittance [$|Y|$] and phase angle [ϕ]) formats on the right.

(i.e., 1.68 – 1.11) and G_{tm} is 0.21 (i.e., 0.32 – 0.11) acoustic mmho. B_{tm} is plotted on the y-axis and G_{tm} is plotted on the x-axis to the right of the 226 Hz tympanogram. Make the same two calculations on the 678 Hz B_a and G_a tympanograms in the lower panel; $200B_{tm}$ and $200G_{tm}$ are 1.72 (i.e., 4.50 – 2.78) and 2.58 (i.e., 2.96 – 0.38) acoustic mmho, respectively. Perpendicular lines drawn from the x and y axes intersect at a point in the complex admittance plane that describes the peak compensated static acoustic admittance of this 40-year-old male. The intersecting lines form a rectangle when B_a and G_a are plotted in *rectangular* format.

The point where the two perpendicular lines intersect also can be described in *polar* format by drawing a vector from the origin, or x-y intercept, to the same point in the complex plane. The length or magnitude of the admittance vector (Y_{tm} or $|Y|$) and the phase angle (ϕ) of Y_{tm} with respect to the x-axis describe acoustic admittance in *polar* format. The vertical bars sometimes are used to indicate that the admittance vector is an absolute value and has no sign. The rectangular components of acoustic admittance can be transformed to polar format using the Pythagorean theorem or Equation 8.5 and converting from radians to degrees. At 226 Hz, Y_{tm} is 0.60 acoustic mmho with a phase angle of 70° , and at 678 Hz, Y_{tm} is 3.10 acoustic mmho with a phase an-

gle of 34° . Measuring only Y_{tm} without phase information is analogous to giving someone directions to your home by saying drive 5 miles. Similarly, specifying only Y_{tm} puts the vector anywhere between the 90° axis (straight up) and the -90° axis (straight down). It is common practice, however, to measure only Y_{tm} when a 226 Hz probe signal is used because the ear is very stiffness dominated, and the admittance vector lies close to the 90° axis. Note in the top panel of Figure 9.4 that the Y_a and B_a tympanograms at 226 Hz basically overlap and G_a contributes minimally to Y_a . This is not the case at 678 Hz, however, where the Y_a tympanogram deviates significantly from the B_a tympanogram near the peak. When high-frequency probe tones are used, both B_a and G_a contribute to complex acoustic admittance and must be specified; phase angle can no longer be assumed to approximate 90° . Although B_{tm} and Y_{tm} are essentially equal at 226 Hz, they are much different at 678 Hz, and in fact, Y_{tm} is closer in magnitude to conductance than to susceptance. Remember that Y_{tm} is a vector quantity and will *always* be larger than either of the contributing rectangular components (B or G). Susceptance is the primary contributor to Y_{tm} at 226 Hz, but conductance is the primary contributor at 678 Hz. If only Y_{tm} were measured at 678 Hz, you would have no idea which component, B_{tm} or G_{tm} , contributed more to its magnitude. Specifying only Y_{tm} at high frequencies presents an incomplete description of the acoustic admittance of the middle ear.

MULTIPLE-FREQUENCY TYMPANOMETRY

Normal 226 Hz tympanograms, no matter which admittance component is measured, are always single peaked. As probe frequency increases, however, tympanograms begin to notch in a systematic and predictable manner. The normal multiple-frequency tympanograms in Figure 9.5 were recorded from the same 40-year-old man as shown in Figure 9.4. Notice the change in the shapes of each of the four uncompensated admittance component tympanograms (G_a , B_a , Y_a , ϕ_a) as probe frequency increases from 226 to 1243 Hz². At low frequencies, the tympanograms all are single peaked; the amplitude at the peak gradually increases with frequency and then develops a notch at the peak. As frequency increases further, the central notch deepens. For the B_a tympanograms, the notch deepens and then develops a secondary notch at the peak. Next, notice the probe frequency at which notching first occurs for each of the admittance components, i.e., B_a at 904 Hz, G_a and Y_a at 1017 Hz, and ϕ_a at 1130 Hz³.

² An upper range of 1243 rather than 2000 Hz was used because of unstable calibration at higher frequencies.

³ All the tympanograms in Figure 9.5 are uncompensated for ear-canal volume. Although the shapes of the susceptance and conductance tympanograms are the same compensated and uncompensated, ear-canal volume does not have the same linear effect on admittance and phase angle tympanograms. See Shanks and Lilly (1981) and Shanks et al. (1988) for further discussion.

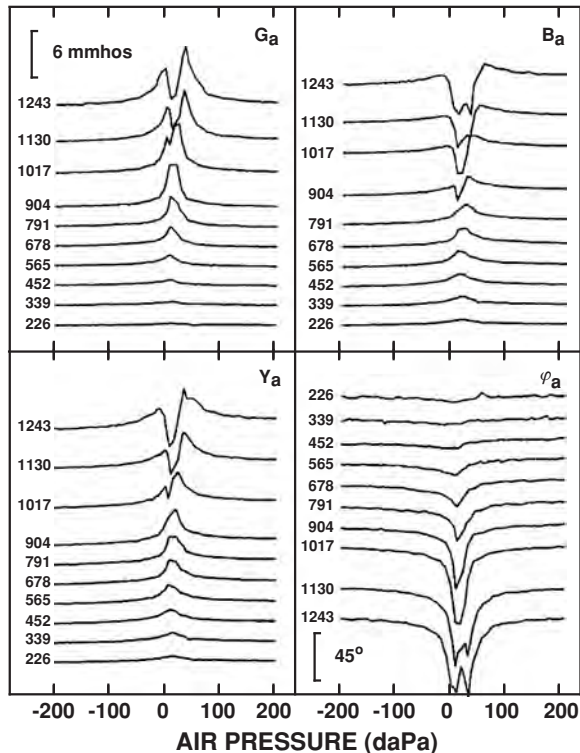


FIGURE 9.5 Uncompensated multiple frequency conductance (G_a), susceptance (B_a), admittance (Y_a), and phase angle (ϕ_a) tympanograms recorded from a normal, 40-year-old male.

Figure 9.6 demonstrates two ways to plot peak compensated static acoustic admittance for the family of normal tympanograms shown in Figure 9.5. Panel A shows $200B_{tm}$, G_{tm} , and Y_{tm} as a function of frequency.⁴ First examine the magnitude relationship between B_{tm} (open squares) and G_{tm} (filled diamonds). B_{tm} is larger than G_{tm} for probe frequencies less than 678 Hz. Stated differently, the peak-to-tail amplitudes of the susceptance tympanograms in Figure 9.5 are larger than the peak-to-tail amplitudes of the conductance tympanograms for probe frequencies of 226 through 565 Hz. The B_{tm} and G_{tm} functions in Figure 9.6A cross when the amplitudes of the B and G tympanograms are equal, i.e., when the admittance vector is at 45° as indicated by the dashed vertical line near 580 Hz. Beginning at 678 Hz, the amplitude of the G_{tm} tympanogram now is greater than the B_{tm} tympanogram, and the conductance function (filled diamonds) now rises above the susceptance function (open squares). As the amplitude of the conductance tympanogram continues to increase, the susceptance tympanogram develops a notch and begins to decrease to 0 acoustic mmho near 850 Hz. Recall that the resonant frequency (f_0) of the middle ear, indicated by the second vertical dashed line, occurs when stiffness susceptance (B_c) and mass susceptance (B_m)

⁴ Although frequency typically is incremented and plotted logarithmically, linear scaling was chosen to enhance plotting resolution.

are equal. The ear is stiffness controlled below f_0 and mass controlled above f_0 . Stated differently, peak B_{tm} is positive below resonance and negative above resonance. In contrast, G_{tm} increases until a notch develops at 1017 Hz and then decreases sharply but can never be negative. Now examine the Y_{tm} function (filled circles) in Figure 9.6A. Recall that both susceptance and conductance contribute to Y_{tm} , so admittance magnitude will always be greater than the larger of the two rectangular components. For low frequencies such as 226 Hz, Y_{tm} is similar in magnitude to B_{tm} , but for frequencies near f_0 , Y_{tm} is similar to G_{tm} and reaches a maximum near f_0 . As frequency increases above f_0 , G_{tm} decreases and Y_{tm} again is similar in magnitude to B_{tm} , but of course will never be negative.

Figure 9.6B shows exactly the same data as shown in Panel A, but plotted in polar format. This format clearly shows that the admittance vector rotates in a very orderly pattern as probe frequency increases from 226 Hz to 1243 Hz. When the admittance vector is above the 0° axis (i.e., between 90° and 0°), B_{tm} is positive, and the middle ear is described as stiffness controlled. At resonance, the admittance vector lies along the x-axis at 0° . Although the probe frequency did not coincide exactly with f_0 in this particular ear, the admittance vector crosses the x-axis at about 850 Hz. Above 850 Hz, the admittance vector rotates into the negative susceptance quadrant between 0° and -90° , and the middle ear is described as mass controlled. If only Y_{tm} without phase angle is measured, you cannot tell whether the ear is stiffness controlled or mass controlled, i.e., the admittance vector could be anywhere in the 180° plane.

Plotting only what happens at a single point on the tympanogram ignores valuable information about tympanogram shape. In contrast, plotting the entire tympanogram allows you to analyze both the shape and static admittance. 226 Hz Y_a tympanograms provide information on the stiffness characteristics of the middle ear including volume estimates, and 678 Hz B_a and G_a tympanograms provide more information on the mass characteristics and resonance of the middle ear. At 226 Hz, Y_a tympanograms are almost always single peaked and require calculations to differentiate between normal and pathological middle ears. Analysis of 678 Hz tympanograms is much different, but also much simpler, thanks to a model developed by Vanhuyse and his Belgian colleagues (1975).

The Vanhuyse Model

Notched 678 Hz tympanograms were not completely understood until the paper of Vanhuyse et al. (1975). The Vanhuyse model began with some basic assumptions about the normal shapes and magnitude relationships of acoustic resistance (R_a) and acoustic reactance (X_a) tympanograms recorded by Moller (1965). The R_a tympanogram decreased monotonically from high negative pressures to high positive pressures, and the X_a tympanogram was a parabolic function

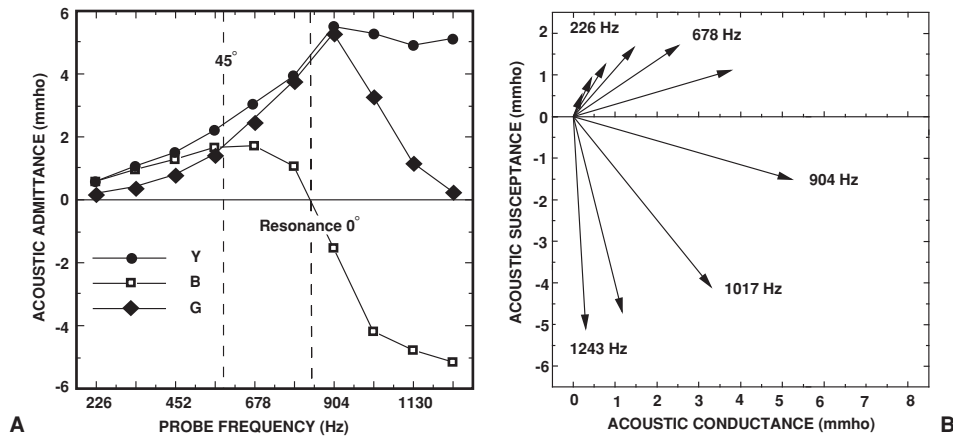


FIGURE 9.6 Peak compensated static acoustic admittance calculated from the normal multiple frequency tympanograms in Figure 9.5. Peak $200B_{tm}$ (open squares), $200G_{tm}$ (filled diamonds), and $200Y_{tm}$ (closed circles) are plotted in rectangular format as a function of probe frequency in Panel A; the rectangular admittance values corresponding to phase angles of 45° and 0° are indicated by dashed lines. Rotation of the admittance vector as a function of probe frequency is plotted in polar format in Panel B.

symmetric around 0 daPa. The R_a tympanogram was kept constant, and the X_a tympanogram was progressively shifted from stiffness-controlled to mass-controlled conditions. For each of four shifts in the X_a tympanogram, B_a and G_a tympanograms were calculated (Eq. 8.40) and replotted. Although the shapes of the R_a and X_a tympanograms remained constant, the four resultant B_a and G_a tympanograms became increasingly complex with each shift in X_a . [See Margolis and Shanks (1985), Shanks (1984), Shanks et al. (1988) and Van Camp et al. (1986) for a more in depth review of the Vanhuyse model].

The left half of Figure 9.7 shows the four tympanogram patterns predicted by the Vanhuyse model. The B_a and G_a tympanograms change shape for each 45° rotation of the admittance vector. When the admittance vector is between 90° and 45° , both the B and G tympanograms are single peaked and are designated 1B1G. When the vector rotates between 45° and 0° , a notch develops in the B tympanogram, but the G tympanogram remains single peaked; this pattern is designated 3B1G. The “3” indicates that there are 3 extrema or direction changes in the peak of the B tympanogram. The vertical lines with the arrows indicate the “peak” of the notched tympanograms and the pressure at which peak compensated static acoustic admittance is calculated. Because acoustic resistance tympanograms are markedly asymmetric, peak pressures for B_a and G_a tympanograms differ slightly (Margolis and Popelka, 1977; Moller, 1965; Van Camp et al., 1978; Van Camp et al., 1986). The horizontal lines with the arrows indicate that the tympanograms have been compensated for ear-canal volume, i.e., baseline corrected. Note that the notch in the 3B tympanogram is still above the baseline, and therefore, peak $200B_{tm}$ is positive and the middle-ear is stiffness controlled. If the center of the notch had fallen exactly on the baseline, peak $200B_{tm}$ would equal 0 acoustic mmho and indicate resonance.

As the admittance vector rotates between 0° and -45° , the conductance tympanogram also develops a notch and is designated 3B3G. Look at the susceptance (B) tympanogram and note that the center notch now is below the zero baseline. $200B_{tm}$ is negative, and the middle ear is mass controlled. When the admittance vector rotates between -45° and -90° , a 5B3G tympanogram is recorded. The notch in the conductance tympanogram deepens and the susceptance tympanogram develops a secondary notch. Note that static acoustic susceptance is calculated using the peak value in the secondary notch as indicated by the vertical arrow. Again, susceptance is negative, and the ear remains mass controlled.

The Vanhuyse model also predicted that a normal admittance magnitude tympanogram (Y_a) has only two shapes: a single-peaked tympanogram in a stiffness-controlled middle ear when the vector is $>0^\circ$ and a notched tympanogram in a mass-controlled middle ear when the vector is $<0^\circ$. In other words, the Y_a tympanogram notches at or above f_0 .

All four tympanogram patterns in the left half of Figure 9.7 are normal. Tympanograms recorded with descending pressure changes conform best to the Vanhuyse model (Margolis et al., 1985). At 678 Hz, normal B/G tympanograms are distributed as follows: 75% 1B1G, 8% 3B1G, 4% 3B3G, and 13% 5B3G. Notch width for 3B and 5B tympanograms is 36 and 51 daPa, respectively (Creten et al., 1985). In polar format, 83% are 1Y and 89% are 1ϕ (Creten et al., 1985; Wilson et al., 1984).

Although the Vanhuyse model originally was developed to explain the four normal tympanogram patterns recorded at 678 Hz, the model also is useful in accounting for changes in tympanogram shape as a function of frequency (Margolis et al., 1985). The incidence of notching increases for higher frequency probe tones. For example, at 1000 Hz approximately 80% of normal adult ears have 3B1G tympanograms (Calandrucchio et al., 2006).

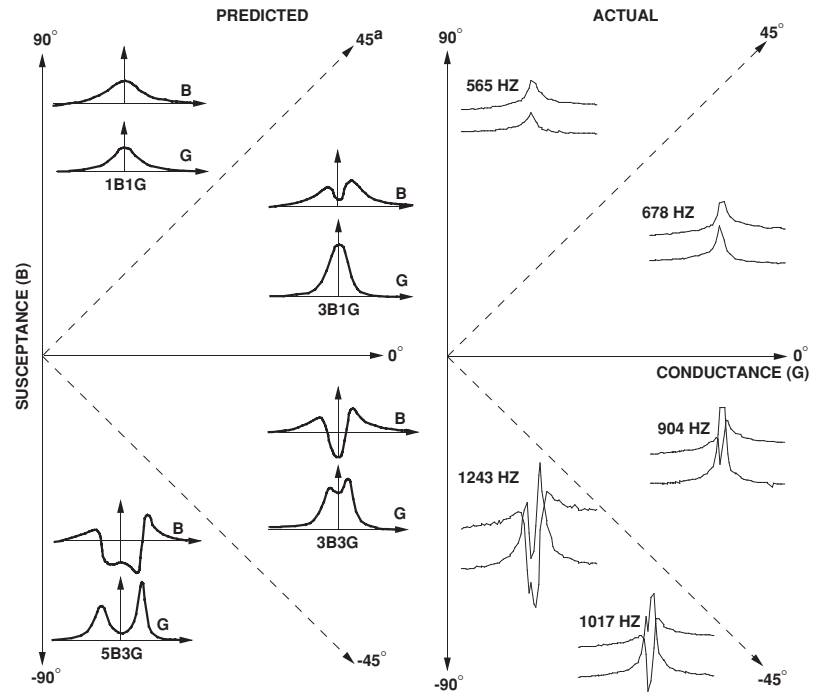


FIGURE 9.7 A comparison of susceptance (B) and conductance (G) tympanograms predicted by the Vanhuysse et al. (1975) model (Predicted) versus actual tympanograms measured from a 40-year-old male with a normal middle-ear transmission system (Actual). The actual tympanograms are from the same person whose data are depicted in Figures 9.4 through 9.6.

In the right half of Figure 9.7, the predicted Vanhuysse patterns are compared with samples of actual MFT previously shown in Figure 9.5. In each tympanogram pair, susceptance (B) is always the top function and conductance (G) is the lower function. Deviations from the predicted model tend to occur at the transitions between patterns, particularly for the 1B1G and 5B3G patterns (Margolis et al., 1985). One very common pattern that is not accounted for by the Vanhuysse model was recorded at 678 Hz. Note that G_{tm} is greater than B_{tm} , although the model predicts that the B_a tympanogram should notch when the admittance vector rotates below 45° . In this example, the phase angle is 34° , but the B_a tympanogram remains single peaked. Similarly, a 3B1G pattern was recorded at 904 Hz when the admittance vector was at an angle of -16° ; the model predicts a 3B3G pattern when the middle ear is mass controlled. At 1017 Hz, a 3B3G pattern was recorded even though the phase angle was at -51° ; the Vanhuysse model predicts a 5B3G pattern when the admittance phasor is between -45° and -90° . In all cases, the small deviations from the model occurred close to the transition angles of 45° , 0° , and -45° . Despite these small differences, the Vanhuysse model has been invaluable in understanding changes in the shapes of 678 Hz tympanograms across normal subjects and as a function of probe frequency.

This same group of Belgian researchers defined abnormal notching of B_a and G_a tympanograms. Simply determining whether an ear is stiffness controlled, mass controlled, or at resonance is not very diagnostic because the range of normal variability is so large. Examining the characteristic of the notching, however, can be diagnostic. Van de Heyning et al. (1982) quantified the number of extrema and the pressure

interval between the outermost extrema to define abnormal notching as follows:

- more than 5 extrema in the susceptance (B) tympanogram,
- more than 3 extrema in the conductance (G) tympanogram,
- notch width >75 daPa for 3B tympanograms,
- notch width >100 daPa for 5B tympanograms, or
- notch width >60 daPa for 3Y tympanograms.

A simple shape analysis also can be used to identify 1B1G tympanograms that are abnormally stiff. Simply compare the relative peak-to-tail amplitudes of the single-peaked B and G tympanograms. Only one of the following three magnitude relationships is possible:

- If $B_{tm} > G_{tm}$, then the admittance vector is $>45^\circ$.
- If $B_{tm} < G_{tm}$, then the admittance vector is $<45^\circ$.
- If $B_{tm} = G_{tm}$, then the admittance vector is $=45^\circ$.

Clinical experience with a 678 Hz probe tone in an adult male population shows that the admittance vector normally is $\leq 45^\circ$. If the vector is closer to the positive y-axis, i.e., between 90° and 45° , then $B_{tm} > G_{tm}$ and the ear is abnormally stiff. Refer again to the 678 Hz susceptance (B) and conductance (G) tympanograms in the lower half of Figure 9.4. In this example, $B_{tm} < G_{tm}$ at 678 Hz, the admittance vector is $<45^\circ$, and the middle ear is normally stiffness controlled. In contrast to 226 Hz tympanograms, 678 Hz tympanograms can be classified as normal, abnormally stiff, or abnormally mass controlled from a simple visual comparison of the magnitude relationship of B_a and G_a .

The Vanhuyse model lays the foundation for understanding other MFT procedures. Lilly (1984) provides an excellent review of experimental MFT procedures that were the precursors to current clinical techniques. In general, MFT can be categorized as sweep-pressure or sweep-frequency procedures. Traditional tympanometry uses a sweep-pressure technique. In sweep-frequency tympanometry, the probe-tone frequency is swept at discrete ear-canal pressures. Although the shapes of the tympanograms look similar, the estimates of f_0 for the two techniques are different.

Sweep-Pressure Tympanometry

Colletti (1975, 1976, 1977) introduced a sweep-pressure procedure for estimating the resonant frequency of the middle-ear transmission system. He used a custom device to measure uncompensated Z_a tympanograms (i.e., inverted Y_a tympanograms) for probe frequencies of 200 to 2000 Hz. Colletti noted that the shape of the Z_a tympanograms varied systematically from single peaked, to notched, to inverted as frequency increased. The frequency range corresponding to the emergence of the notched tympanogram was the most distinctive and easiest to classify. Recall from the Vanhuyse et al. (1975) model that a notched 3Y pattern emerges near the resonant frequency (f_0) of the middle ear. In normal ears, notched Z_a tympanograms were observed over a 300 Hz frequency interval beginning at an average of 1000 Hz with a normal range of 650 to 1400 Hz. The same three tympanogram patterns were recorded in pathological ears, but the frequency range where the notched pattern first emerged was different from normal ears. In general, the notched pattern emerged at higher than normal frequencies (850 to 1650 Hz) in ears with increased stiffness due to otosclerosis and lower than normal (500 to 850 Hz) in ears with increased mass due to ossicular discontinuity. Although considerable overlap occurred between the normal and otosclerotic ears, little overlap was found between ears with otosclerosis and ossicular discontinuity.

Sweep-Frequency Tympanometry

The MFT option on the Virtual (Model 310) and Grason Stadler (Model 33 and Tymstar) instruments is based on a sweep frequency procedure developed by Funasaka et al. (1984). They used a custom-built instrument to measure the difference in the sound pressure level and phase of a sweep frequency probe tone at 0 daPa versus -200 daPa. They identified four characteristics of the difference curves that were effective in differentiating among middle-ear pathologies. One of the most effective measures was the 0 crossing of the SPL difference curve, which averaged 1500 Hz in normal ears, <720 Hz in ears with ossicular discontinuity, and >1880 Hz in ears with ossicular fixation (Funasaka and Kumakawa, 1988). Their results again reflect an increase in f_0 for pathologies that increase stiffness and a decrease in f_0 for patholo-

gies that increase mass or conversely, decrease stiffness. 83% of ears with ossicular discontinuity and malleus/incus fixation and 55% of ears with stapedial fixation were correctly identified. Malleus and incus fixations result in very high impedance and typically are easier to detect than the more lateral pathology of stapes fixation. In contrast, at 220 Hz, only 32% of ears with ossicular fixation and 42% with ossicular discontinuity were identified correctly. Overall, 220 Hz tympanometry identified 35% of patients with ossicular discontinuity and fixation, whereas sweep-frequency measures identified 68%.

F_0 estimated in the Funasaka studies (1984, 1988) is lower than that reported in the Colletti studies (1975, 1976, 1977) because measurements were made at ambient rather than at peak pressure. The steep slope of tympanograms near the peak results in large differences between Y_{tm} calculated at peak versus 0 daPa. Y_{tm} is about 20% lower at 0 daPa than at peak, so estimates of f_0 will be higher at 0 daPa than at peak (Wilson et al., 1984). Although some investigators logically argue that measurements at 0 daPa present the most accurate picture of how an ear functions in the daily environment, measurements at peak are preferred because of better intra- and inter-test agreement (Margolis and Popelka, 1975; Porter and Winston, 1973; Wilson et al., 1984).

Both the Virtual (Model 310) and Grason Stadler (Model 33 and TymStar Version 2) instruments have a test option similar to that described by Funasaka et al. (1984), except the measurements are done at TPP rather than at 0 daPa. To run the multiple-frequency feature on the Grason-Stadler instruments, acoustic admittance first is measured as probe frequency is increased from 250 Hz to 2000 Hz at the selected starting pressure, typically at 200 daPa. Next, a 226 Hz tympanogram is recorded to identify TPP, and then a second frequency sweep is performed at peak. The Grason-Stadler instrument always plots the difference in phase angle between the starting and peak pressures ($\Delta\phi$) and plots a second difference function of your choice, which logically should be susceptance (ΔB). The difference in acoustic susceptance between peak and 200 daPa is simply a plot of $200B_{tm}$ as a function of frequency (see Fig. 9.6A). F_0 is indicated at the 0 crossing of the ΔB function.

The value of the $\Delta\phi$ function, however, is questionable. Valvik et al. (1994) concluded that $\Delta\phi$ provides no information of any value. Although *compensated* phase angle is 0° at resonance, $\Delta\phi$ is derived by simply subtracting two uncompensated phase angles and is not a measure of the phase of the middle-ear transmission system. In contrast, the Virtual instrument plotted true peak compensated phase angle by correcting for ear-canal volume in rectangular format (susceptance and conductance) and then converting back to polar format. In this case, the probe frequency corresponding to a phase angle of 0° provided an estimate of f_0 . Although this is a mathematically sound procedure, in practice the procedure was susceptible to artifacts and the test-retest reliability was not very good. Holte (1996) noted errors in estimating f_0 with this procedure by as much as 2 octaves.

The mathematics governing complex numbers can be confusing. In contrast to the rectangular components of acoustic admittance (B_a and G_a), the polar components (Y_a and ϕ_a) cannot be compensated to the plane of the tympanic membrane by simply subtracting the values at 200 daPa from the peak values (Shanks et al., 1993). Stated differently, a Y_a tympanogram baselined at 200 daPa ($200Y_a$) is not the same as $200Y_{tm}$ particularly when high-frequency probe tones are used. Simply baselining a Y_a tympanogram at 200 daPa can result in a $200Y_a$ value that is 0 acoustic mmho or even negative. You know from previous discussions that Y_a is a magnitude value that can never be equal to 0 or negative. Unlike B_a and G_a , the shapes of Y_a and phase-angle tympanograms are different in the plane of the probe tip versus the plane of the tympanic membrane, and must be compensated using vector mathematics (Shanks et al., 1988).

Age and Gender Effects

Wiley et al. (1996; 1999) reported small gender differences with slightly higher f_o in women than in men, except among the 80 to 90 year olds. Other studies reported no age or gender effects on f_o (Ferekidis et al., 1999; Hanks and Mortenson, 1997; Hanks and Rose, 1993; Holte, 1996; Margolis and Goycoolea, 1993; Shahnaz and Davies, 2006). In view of the wide normal range for f_o , small gender and age effects are not clinically significant and do not justify the inconvenience of separate norms.

Procedural Variables

Several procedural variables affect tympanogram amplitude and shape and must be considered when norms are established. In general, procedural variables have a greater effect on the tympanogram peak than on the tails, and have a greater effect at high frequencies than at low frequencies.

RATE AND DIRECTION OF PRESSURE CHANGE

Pump speed has a significant effect on Y_{tm} but little effect on V_{ea} and TW (Creten and Van Camp, 1974; Koebshell and Margolis, 1986; Margolis and Heller, 1987; Shanks and Wilson, 1986). Single-peaked tympanograms increase in amplitude with increasing rates of pressure change. For example, Margolis and Heller (1987) measured a 10–14% increase in Y_{tm} for an increase in pump speed from 200 daPa/s to 400 daPa/s. In addition, the incidence, depth, and complexity of notching increases at high rates of pressure change.

The amplitude and shape of tympanograms also are affected by the direction of pressure change, particularly at high frequencies. Tympanograms recorded in the negative to positive direction ($-/+$) typically result in higher Y_{tm} than the positive to negative ($+/-$) direction and exhibit more complex notching (Alberti and Jerger, 1974; Creten and Van Camp, 1974; Margolis and Smith, 1977; Shanks and Wilson, 1986; Van Camp et al., 1980; Wilson et al., 1984). Wilson et al. (1984) and Shanks and Wilson (1986) reported

that 38% to 46% of 678 Hz tympanograms were notched for ascending pressure changes and only 21% to 25% were notched for descending pressure changes. The descending pressure change is preferred because the notched patterns are more consistent with the Vanhuysse et al. (1975) model. More recently, other studies have shown that ranges of f_o are smaller and test-retest reliability is higher for descending ($+/-$) than for ascending ($-/+$) pressure changes (Margolis and Goycoolea, 1993; Wiley et al., 1999).

COMPENSATION OF EAR-CANAL VOLUME

The pressure used to estimate V_{ea} is the single most important procedural variable to control in establishing norms. Because of tympanogram asymmetry, V_{ea} almost always is larger when estimated from the positive rather than the negative tail of the tympanogram. This in turn results in lower peak Y_{tm} and a lower f_o by 150 to 400 Hz (Hanks and Mortensen, 1997; Holte 1996; Margolis and Goycoolea, 1993; Shahnaz and Polka, 1997; Shanks et al., 1993; Wiley et al., 1999). More importantly, these same studies found that the 90% range for f_o is smaller, with smaller inter- and intra-subject variability for positive versus negative pressure compensation. This provides additional strong support for ear-canal compensation at a positive starting pressure.

NUMBER OF CONSECUTIVE PRESSURE SWEEPS

Y_{tm} also increases with the number of pressure sweeps, especially during the first three to five tympanograms (Osguthorpe and Lam, 1981; Vanpeperstraete et al., 1979; Wilson et al., 1984). An 18% increase in Y_{tm} at 226 Hz from sweep one to sweep ten was attributed to alterations in the viscoelastic properties of the eardrum and middle ear with repeated pressurization (Wilson et al., 1984). This variable is a consideration when sweep-pressure MFT is employed.

SWEEP-PRESSURE VERSUS SWEEP-FREQUENCY TYMPANOMETRY

Margolis and Goycoolea (1993) discussed a myriad of procedural variables that affect the estimate of f_o . They compared eight tympanometry estimates of f_o in order to identify the measure with the highest test-retest reliability and lowest inter-subject variability. For both sweep-pressure and sweep-frequency tympanometry, f_o was 78 to 151 Hz higher for Y_a versus B_a tympanograms. In addition, sweep-pressure tympanometry produced lower estimates of f_o than sweep-frequency tympanometry. Mean f_o was 990 Hz with a 90% range of 630 to 1400 Hz and 1135 Hz with a 90% range of 800 to 2000 Hz for sweep-pressure and sweep-frequency tympanometry, respectively. Similarly, Hanks and Mortensen (1997) reported a 400 Hz difference in f_o between sweep-pressure and sweep-frequency tympanometry in young adults. The difference in f_o could result from multiple consecutive pressure sweeps during sweep-pressure tympanometry (Osguthorpe and Lam, 1981; Vanpeperstraete et al., 1979; Wilson et al., 1984). To maximize the range of

possible abnormal values, Margolis and Goycoolea (1993) suggested using sweep-pressure tympanometry to evaluate stiff ears and sweep-frequency tympanometry to evaluate mass-controlled ears. Another consideration in choosing between the two options is time; sweep-frequency tympanometry is faster, and the effects of the direction and rate of pressure change are not issues.

Even when procedural variables are controlled, the normal 90% range of f_0 is wide. In general, f_0 is shifted higher when stiffness is increased and is shifted lower when mass is increased or conversely, stiffness is decreased. Although the f_0 can help to categorize middle-ear pathologies into two broad categories, i.e., stiffness- versus mass-related pathologies, f_0 is not very useful in differentiating among pathologies within each broad category. As will be demonstrated with several case studies in the final section of this chapter, tympanogram morphology, particularly TW and notch width, can be more discriminating than f_0 .

TESTING NEONATES

Several investigators in the early 1970's noted that conventional 226 Hz tympanometry yielded different patterns in newborns compared with older infants and adults. 226 Hz tympanograms frequently were notched and resembled patterns usually recorded at higher probe-tone frequencies in adults (Bennett, 1975; Keith, 1973, 1975). The atypical, notched tympanograms were hypothesized to reflect hypermobile eardrums (Bennett, 1975; Keith, 1973; 1975), distensible ear-canal walls (Paradise et al., 1976), or developmental changes in the ear (Keith, 1973). In contrast, single-peaked tympanograms were recorded from some infants with otoscopically/surgically confirmed middle-ear effusion (Paradise et al., 1976; Shurin et al., 1976). As a result of these early studies, low-frequency tympanometry was not considered reliable in neonates and infants less than 6 months of age.

Later studies used high-frequency probe tones and the Vanhuyse model to quantify developmental changes in the tympanogram patterns of neonates. At 226 Hz, notched tympanograms (e.g., 3B1G, 3B3G, 5B3G) were recorded in 50% to 83% of neonate ears (Himelfarb et al., 1979; Holte et al., 1991; McKinley et al., 1997; Sprague et al., 1985). Examination of even the 1B1G tympanograms at 226 Hz showed that the magnitude relationship between B_a and G_a was similar to that typically recorded in adult ears at 678 Hz, that is, phase angle was $<45^\circ$ rather than $>70^\circ$ typically found in older children and adults. In contrast, 678 Hz tympanograms in neonates frequently were described as flat or "bizarre" and could not be classified by the Vanhuyse model (Himelfarb et al., 1979; McKinley et al., 1997; Sprague et al., 1985).

These atypical tympanograms converted quite rapidly from notched to 1B1G during the first few weeks of life. All 226 Hz tympanograms were notched in 5 to 11 hour old newborns, decreasing to 70% notched at one week, to 30%

to 46% at two weeks, and to 24% at 1 to 2 months (Bennett, 1975; Calandruccio et al., 2006; Holte et al., 1991). By 2 to 4 months, most 226 Hz tympanograms converted to 1Y or 1B1G and were similar to patterns recorded in adult ears (Himelfarb et al., 1979; Holte et al., 1991; Marchant et al., 1986; Meyer et al., 1997).

The reason for the low-frequency notched tympanograms in neonates is still being debated. One possible difference, in addition to the obvious size difference, is that the ear canal of neonates is entirely cartilaginous at birth and highly distensible under pressure from pneumatic otoscopy or tympanometry. Holte et al. (1991) conducted a study to examine the effect of ear-canal distention on the shapes of multiple-frequency tympanograms. Pneumatic video otoscopy revealed up to a 70% change in ear-canal diameter in 1- to 5-day-old newborns. Although the osseous portion of the ear canal is not fully formed until about 1 year of age, only a minimal change in ear-canal dimension was measured beyond one month of age. Importantly, Holte et al. found no correlation between the distention of the ear canal and the complexity of 226 Hz tympanograms. Ear-canal effects were primarily evidenced in tympanogram asymmetry at the tails but not at the peak.

Although flaccidity of the ear canal did not account for the notching of 226 Hz tympanograms, it did frequently cause ear-canal collapse around the probe tip, particularly for high negative pressures and ascending pressure changes (Holte et al., 1991; Sprague et al., 1985). Collapse occurs most frequently during the first week after birth and is indicated when acoustic admittance abruptly decreases toward 0 acoustic mmho when negative ear-canal pressures are applied during tympanometry. Another effect of the cartilaginous ear canal in newborns is that the ear canal cannot be modeled as a hard-walled cavity or pure acoustic compliance at extreme pressures as it can be in adults. Recall that phase angle at extreme ear-canal pressures should approach 90° in order to accurately estimate the acoustic admittance of the ear canal and compensate measures to the plane of the eardrum. This condition frequently is not met in neonates and makes the calculation of peak Y_{tm} suspect when ear-canal phase angle is less than about 70° (Holte et al., 1991; Sprague et al., 1985). A tympanometry procedure that does not rely on compensated measures, therefore, may be preferable in neonates.

Holte et al. (1991) concluded that the notched 226 Hz tympanograms reflected increased mass and resistance with a concomitant decrease in resonant frequency in newborns. They reported the occurrence of two resonances at 450 and 710 Hz that increased to a single resonance at 900 Hz in older infants. A possible contributor to the decreased f_0 in neonates is the presence of fluid/debris in the ear canal and middle ear. At birth, the ear canal contains varying amounts of vernix caseosa, and the middle ear contains residual mesenchyme and amniotic fluid (Eavey, 1993). Renvall et al. (1975) demonstrated in temporal bones that low fluid levels in the middle ear result in broadly notched, mass-controlled

tympanograms. Both the vernix and mesenchyme decrease precipitously in the first few days after birth and likely contribute to the shift toward single-peaked tympanograms and increase in f_0 in the first few weeks of life (Himelbarb et al., 1979; Holte et al., 1991; Keith, 1975; Meyer et al., 1997). Whether notched 226 Hz tympanograms common in the first few days after birth are due to anatomical/developmental differences or simply to the effects of residual ear-canal and middle-ear fluid is unknown.

The ideal probe-tone frequency and admittance component for use in neonates and infants is likely to be debated for some time. Despite the notching of low-frequency tympanograms in neonates, Holte et al. (1991) recommended a 226 Hz probe tone because tympanogram patterns were the least affected by maturational changes and were most consistent with the Vanhuysse model. Other investigators have argued in favor of high-frequency probe tones after noting that many newborns who failed OAE/ABR screening had flat B_a or Y_a tympanograms at 678, 800, or 1000 Hz (Hirsch et al., 1992; Kei et al., 2003; Marchant et al., 1986; Margolis et al., 2003; McKinley et al., 1997; Rhodes et al., 1999; Sutton et al., 1996). Although sensitivity was higher for high-frequency probe tones in comparison with 226 Hz, flat tympanograms also were recorded from many ears that passed OAE screening (McKinley et al., 1997; Sutton et al., 1996). For example, Sutton et al. (1996) found that half of the ears with flat Y 678 Hz tympanograms passed OAE testing. Their results showed high sensitivity but low specificity for probe-tone frequencies around 678 Hz. Specificity was improved by increasing the probe tone to 1000 Hz (McKinley et al., 1997; Rhodes et al., 1999; Sutton et al., 1996).

As a result of these findings, admittance magnitude (Y_a) measures at 1000 Hz are becoming the measure of choice in testing neonates and young infants (Kei et al., 2003; Margolis et al., 2003; Purdy and Williams, 2000; Sutton et al., 1996). This measure is especially appealing because Y_a tympanograms at 1000 Hz tend to be single peaked or flat, making it easier to differentiate between normal and abnormal ears (Kei et al., 2003; McKinley et al., 1997; Rhodes et al., 1999). Kei et al. (2003) recorded 1Y tympanograms in 92% of newborns and flat tympanograms in 6%. Although all newborns passed TEOAE screening, the newborns with flat tympanograms were associated with less robust TEOAEs. Similarly, Rhodes et al. (1999) reported that 92% of 1000 Hz tympanograms were single peaked and that three ears with flat 1000 Hz tympanograms failed hearing screening.

A quantitative analysis of Y_a at 1000 Hz as suggested by Margolis et al. (2003) is more challenging, largely because it relies on an accurate estimate of ear-canal volume, agreement on what constitutes tympanogram peak, and compensation of a single-component magnitude measurement. Holte et al. (1991) calculated Y_{tm} only when the phase angle at the tail value exceeded 70° , i.e., a B:G magnitude relationship of ≥ 3 . This condition frequently is not met in neonates. Although V_{ca} routinely is estimated at 200 daPa, Margolis et al. (2003) recommended a pressure of -400 daPa so that V_{ca} would

be smaller, and therefore Y_{tm} would be larger with a wider range of normal values.

The second consideration is what to designate as “peak” on notched tympanograms. Sutton et al. (2002) recommended the negative notch peak, Margolis et al. (2003) used the positive notch peak, and the Vanhuysse model uses the center notch. In order to minimize variability, a consistent procedure must be adopted.

Third, compensation of admittance magnitude theoretically is not possible without phase information. Recall from the previous discussion that vector quantities like Y_a cannot be subtracted using simple mathematics. Because both components of complex acoustic admittance were not available, Margolis et al. (2003) chose an alternative method of expressing tympanogram amplitude by simply reporting the peak: tail amplitude. Although this may prove to be a valid measure, it is important to recognize that this is not equivalent to $-400Y_{tm}$. To avoid confusion, this calculation should be designated as $-400Y_a$ to indicate that it is an expression of uncompensated Y_a amplitude.

Reaching consensus on a tympanometry protocol for neonates is extremely challenging because “normal” is difficult to define and “abnormal” is nearly impossible to verify. Sensitivity/specificity data are difficult to establish in newborns because there is no “gold standard” for this population. Although otoscopy and surgical findings traditionally are used to validate tympanometry in children and adults, neither is appropriate in newborns. Myringotomies typically are not performed in children under one year of age and otoscopy is unreliable in virtually all newborns either because a view of the eardrum is obscured by vernix or because the eardrum is not “normal” in appearance (Eavey, 1993; Himelfarb et al., 1979; Rhodes et al., 1999; Shurin et al., 1976). Shurin et al. (1976) reported that 5 of 10 newborns identified with MEE otoscopically had dry ears on tympanocentesis.

In the absence of a gold standard, many of the recent studies have used the presence of OAE as an indication of normal middle-ear function. Vernix and residual mesenchyme, however, also are confounding factors in screening newborns with ABR and OAE and have been linked to failed screening (Keefe et al., 2000; McKinley et al., 1997). Pass rates tend to be higher in newborns a few days to two weeks old, after the ear canals and middle ears have had a chance to clear (Margolis et al., 2003; McKinley, et al., 1997; Purdy and Williams, 2000; Sining, 2003; Sutton et al., 2002).

Considerable work remains before a tympanometry protocol for newborns and infants under four months of age can be established. Special attention should be placed on age *in days* because the ears of neonates change so rapidly. Beyond one month of age, when most of the debris has cleared from the ear and the canal walls show little movement ($<10\%$) under pressurization, peak Y_{tm} may be a more valid measure. It may be necessary to have one protocol for neonates and another for infants over one month. By 3 to 4 months of age, conventional tympanometry may be appropriate (Himelfarb et al., 1979; Holte et al., 1991; Marchant et al., 1986).

CASE STUDIES

The final section of the chapter will reinforce the principles and applications discussed throughout the chapter. Case history, otoscopy, audiometric tuning forks, acoustic reflexes, and the configuration and magnitude of the conductive component are used together in evaluating tympanograms. A test battery approach is advocated because there is not a 1:1 correspondence between a specific tympanometry pattern and middle-ear pathology. Middle-ear pathology changes along a continuum and can involve multiple pathologies. When all available information is used together, however, tympanometry can contribute unique information about the middle-ear transmission system that is not available from any other audiometric test.

Otosclerosis

Otosclerosis is a progressive disease manifesting in early adulthood that results from abnormal otic capsule bone remodeling (Cherukupally et al., 1998; Chole and McKenna, 2001; Shohet and Sutton, 2001). The first stage of the disease is characterized by resorption of bone and replacement with spongy, highly vascularized bone. The disease progresses to bony fixation of the anterior stapes footplate and then diffuse ankylosis of the entire circumference of the footplate. Hearing loss also progresses over several years from a mild low-frequency conductive hearing loss ≤ 30 dB HL to a flat, severe conductive/mixed hearing loss in the late stages of the disease. Although otosclerosis most commonly leads to a conductive hearing loss, a sensorineural component also can result, possibly through enzymatic secretion of the disease into the membranous labyrinth. A positive family history is present in approximately 50% of cases, and 70% to 80% are bilateral. Otosclerosis affects females twice as often as males and is rare in Blacks and Asians. Clinical otosclerosis develops in less than 1% of the population, although 8–11% of temporal bones show histological evidence of the disease at autopsy.

Considerable attention has focused on otosclerosis because of its relatively high prevalence and frustration over an inability to consistently differentiate this population from normal, even in the presence of large air-bone gaps. On a group basis, ears with otosclerosis demonstrate significantly lower Y_{tm} and higher f_0 than normal ears. On an individual basis, however, there is 26% to 56% overlap with the normal population. Muchnik et al. (1989) reported that only 25% of ears with otosclerosis exhibited increased stiffness and 25% actually had higher than normal admittance; results were equally discouraging at 220 and 660 Hz. Several other investigators, however, reported that the two populations are best differentiated when high-frequency probe tones are used (Jacobsen and Mahoney, 1977; Shahnaz and Polka, 2002; Wada et al., 1998; Zhou et al., 2002). Other studies focused on identifying the optimal acoustic immittance components to measure. Burke and Nilges (1970) reported that peak compensated static acoustic compliance and resistance below

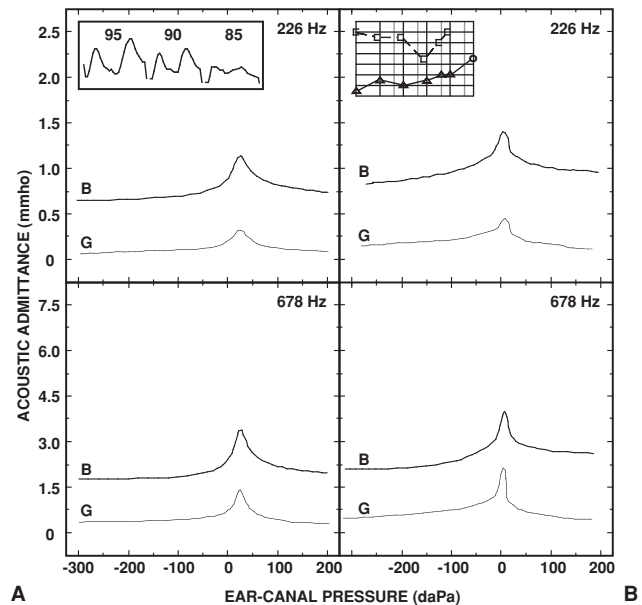


FIGURE 9.8 Susceptance (B) and conductance (G) tympanograms at 226 Hz (top panels) and 678 Hz (bottom panels) recorded from DK with otosclerosis. Panel A shows tympanograms recorded in the early stages of the disease with a biphasic acoustic-reflex response shown in the inset, and Panel B shows tympanograms recorded 18 years later with a pure tone audiogram shown in the inset.

750 Hz differentiated 76% to 89% of the otosclerotic ears from normal. Others reported that φ_{tm} was the single best component to identify otosclerosis (Shahnaz and Polka, 1997; Shanks, 1990; Van Camp and Vogeleeer, 1986). In the end, a diagnosis of otosclerosis is based on a finding of normal-appearing tympanograms in conjunction with large air-bone gaps, typically greater in the low-frequency range. In general, tympanometry is less effective in identifying pathologies like otosclerosis that do not directly affect the eardrum.

Figure 9.8 shows tympanograms from DK, a 32-year-old female, who volunteered for a research study on MFT in normal ears. Preliminary screening for study inclusion revealed normal hearing in the left ear and a mild hearing loss in the right ear with air-bone gaps of 15 to 20 dB at 250 and 500 Hz, a classic stiffness tilt on the audiogram. Upon questioning, DK reported that family members across several generations had otosclerosis. 226 Hz and 678 Hz tympanograms were recorded from the right ear at the initial evaluation (Panel A) and again 18 years later (Panel B). The tympanograms in the Panel A are typical of early otosclerosis. At first glance, the tympanograms appear completely normal. At 226 Hz, $200B_{tm}$ (or $200Y_{tm}$) is stiffer than average, but falls well within the normal 90% ranges for adults in Table 9.1. Analysis of the 678 Hz tympanograms, however, shows that the ear is abnormally stiff. A comparison of the peak-to-tail (i.e., compensated) amplitudes of the susceptance (B) and conductance (G) tympanograms reveals that

peak $200B_{tm}$ is greater than $200G_{tm}$. In other words, the admittance vector is at $>45^\circ$ and the ear is abnormally stiff. A second finding seen only in early otosclerosis is shown in the inset of Panel A. Although no significant air-bone gaps were measured, biphasic or “on-off” acoustic reflexes were recorded from the right ear (Bennett and Weatherby, 1979). This reflex pattern, characterized by a brief increase in admittance with a return to baseline both at the onset and the offset of the eliciting tone, has been attributed to the elasticity of the stapes (Bel et al., 1975; Ciardo et al., 2005).

The audiogram inset in Figure 9.8B shows that hearing loss in the right progressed considerably in 18 years. The bone conduction thresholds show a classic Carhart notch at 2000 Hz that is attributed to a shift in the resonance of the ossicular chain caused by the disease process (Carhart, 1962). A Carhart notch is not a definitive indication of otosclerosis; as subsequent cases will show, most middle-ear pathology will alter the normal resonance characteristics of the ossicular chain and will result in the smallest air-bone gaps near 2000 Hz.

A comparison of 678 Hz peak $200B_{tm}$ versus $200G_{tm}$ in Panel B shows that conductance is greater than susceptance, and therefore, the admittance vector now is in the normal region at $<45^\circ$. This change in admittance seems counter-intuitive; the middle ear is actually less stiff now than it was 18 years ago. Note also that the TW, especially at 678 Hz, is very narrow. Several reports of “peaky” tympanograms in ears with ossicular fixation have been reported in the literature and provide additional evidence for a diagnosis of otosclerosis. An example of narrow tympanogram width can be found in Shanks (1984) and Shanks and Shelton (1991), and also has been noted by Ivey (1975), Dieroff (1978), and Shahnaz and Polka (1997).

In summary, tympanograms in ears with otosclerosis can be normal, abnormally stiff, or have a narrow TW. Any of these patterns in combination with a significant low-frequency conductive hearing loss with normal TPP and otoscopy, and in some cases a positive family history, point to a diagnosis of otosclerosis.

Ossicular Discontinuity

In the absence of a history of otologic surgery or trauma, patients with ossicular discontinuity are difficult to distinguish from those with otosclerosis simply on the basis of case history and otoscopy. Although high resolution CT scanning (Meriot et al., 1997) may aid in the diagnosis, tympanometry with a suggestive history are almost pathognomonic.

Ossicular discontinuity is encountered frequently in the clinical setting, but typically not as an isolated pathology. Discontinuity at the level of the incus most commonly occurs as a sequela of chronic otitis media (COM) and more rarely due to head trauma. Discontinuity associated with COM results from decalcification of the long process of the incus, often leaving only a fibrous band connecting the incus to the stapes (Terkildsen, 1976). Cholesteatoma, eardrum

perforation, and otorrhea also may occur as complications of COM. Surgical intervention in these patients very often is two-staged to remove the disease and repair the eardrum perforation in the first surgery, and to reconstruct the ossicular chain with either a total or partial ossicular replacement prosthesis (TORP or PORP) or bone cements in the second surgery (Babu and Seidman, 2004; Fisch, 1980). Hearing frequently is poorer than pre-surgical levels after the first surgery and then improves following reconstruction of the ossicular chain during the second surgery. Longitudinal fracture of the temporal bone following head trauma also can result in ossicular discontinuity at the incudo-stapedial joint. Head trauma is one of the rare instances when ossicular discontinuity is seen as an isolated pathology.

Figures 9.9A and B show tympanograms recorded from two ears with ossicular discontinuity and intact eardrums. As shown on the inset audiograms, ossicular discontinuity gives rise to a maximum conductive hearing loss. Surprisingly, the cause of the ossicular discontinuity in these two cases was not clear. Figure 9.9A shows tympanograms from RP, a 21-year-old male who suffered head trauma at age 17, but reported that the hearing loss was present prior to the head trauma. Surgical findings revealed complete incudo-stapedial joint separation with degeneration of the long process of the incus. Figure 9.9B shows another case (GS) of ossicular discontinuity with unknown etiology in a 34-year-old male. Surgical intervention again revealed erosion of the long process of the incus, this time with fibrous connections to the head of the stapes.

First, examine the 226 Hz tympanograms in the upper halves of Figures 9.9A and B. The rectangular admittance components, acoustic susceptance (B_a) and conductance (G_a), were recorded in Panel A, whereas admittance magnitude (Y_a) was recorded in Panel B. Y_{tm} calculated using Equation 8.5 is 2.6 acoustic mmho for RP (Panel A) and 2.4 acoustic mmho for GS (Panel B). In this case, conversion of B_{tm} and G_{tm} to Y_{tm} was not necessary to determine whether or not Y_{tm} is abnormal. Recall that B_{tm} and Y_{tm} at 226 Hz are nearly equal because the middle-ear is very stiffness dominated. In addition, Y_{tm} will *always* be greater than either of the two rectangular components; if B_{tm} is abnormally high, Y_{tm} also must be abnormally high. In both cases of ossicular discontinuity, $200Y_{tm}$ is higher than 90% of normal adults, no matter whose norms in Table 9.1 are used. Although 226 Hz TW is wider for GS (Panel B) than for RP (Panel A), both are <200 daPa and fall well within the normal range (see Table 9.2).

Now examine the corresponding 678 Hz tympanograms for these two cases of ossicular discontinuity. Even a cursory glance at these tympanograms reveals that both sets of tympanograms are abnormally mass controlled. First, the 678 Hz susceptance tympanograms are mass controlled because $200B_{tm}$ is negative. Stated differently, B_a in the center notch falls below B_a at 200 daPa. This finding alone does not make these tympanograms abnormal, but the complexity of the notching does. The pattern of the notching is very irregular

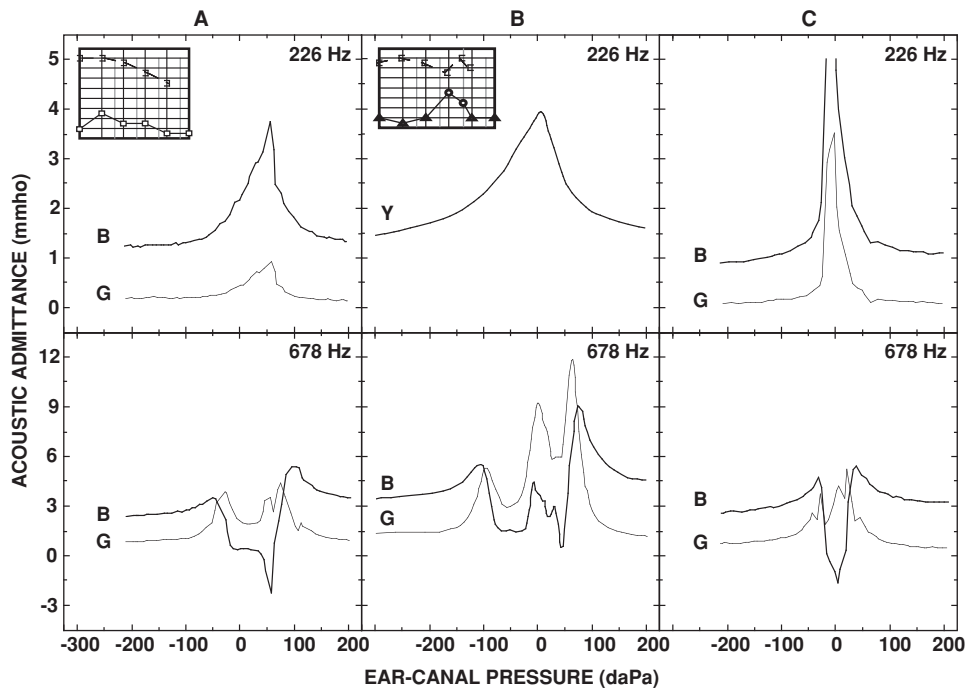


FIGURE 9.9 Susceptance (B) and conductance (G) tympanograms at 226 Hz (top panels) and 678 Hz (bottom panels) recorded from RP and GS with ossicular discontinuity (A and B) and from DJC with atrophic scarring of the eardrum (C). The audiograms for the two patients with ossicular discontinuity are shown as insets; the audiogram for DJC was normal and is not shown.

with more than five extrema for B_a and more than three extrema for G_a . In addition, the pressure interval between the outmost susceptance extrema exceeds 100 daPa. Both tympanograms are clearly outside the normal range defined by the Vanhuysse model (see Figure 9.7A). In contrast to 226 Hz, the pathology at 678 Hz is so obvious that no calculation is necessary. These abnormally mass-controlled 678 Hz tympanograms in conjunction with 60 to 70 dB air-bone gaps and normal otoscopy indicate ossicular discontinuity.

The presenting findings, however, are not always so clear-cut. As stated previously, many middle ears have multiple pathologies. When multiple pathologies are present, tympanometry will be dominated by the most lateral pathology (Chesnutt et al., 1975). As an example, a case of surgically confirmed ossicular discontinuity with intact eardrum was seen in a 16-year-old girl who suffered a temporal bone fracture with facial paralysis and bleeding into the middle-ear cavity following a motor-vehicle accident. When tympanograms were recorded shortly after the accident, she had a hemotympanum (i.e., blood filled middle-ear cavity) that immobilized the eardrum and completely masked the ossicular discontinuity. Her tympanograms were flat and reflected the immobile eardrum and not the more medial ossicular discontinuity that was confirmed at surgery. Similarly, when a perforated eardrum occurs in conjunction with ossicular discontinuity, tympanograms will reflect only the perforated eardrum and will be flat with a large V_{ea} .

Eardrum Pathology

Figure 9.9C is included in the same figure as ossicular discontinuity to emphasize the importance of otoscopy before analyzing tympanograms. Although eardrum pathology fre-

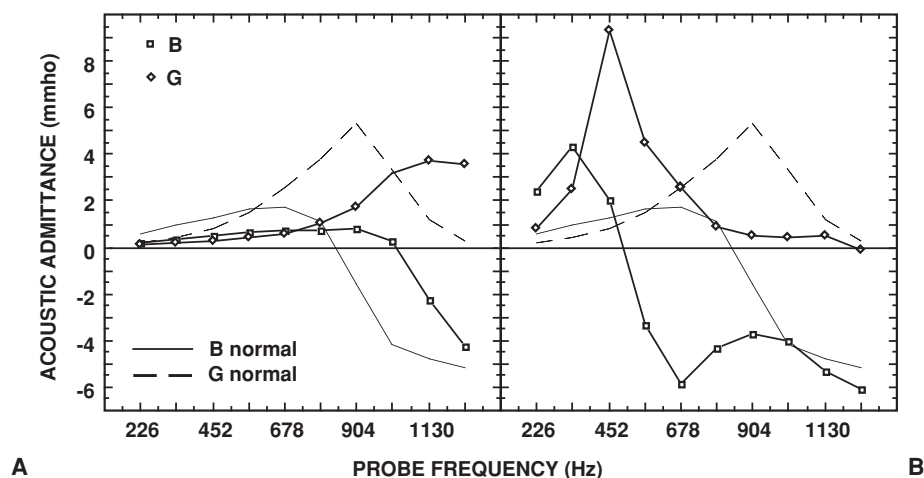
quently has little or no effect on hearing, it can have a dramatic effect on tympanogram shape. Two of the most common conditions are atrophic scarring secondary to healed eardrum perforation and tympanosclerosis of the eardrum. Several excellent examples of these eardrum pathologies are available in text form and online (Ballachanda, 1995; Hawke and McCombe, 1995; Kavanagh, 2006; Sullivan, 2006).

Small eardrum perforations usually heal spontaneously, typically within 6–8 weeks for ears with traumatic perforation and healthy middle ears, and within 3 to 4 months when associated with otitis media (Hawke and McCombe, 1995). The new membrane, referred to as a neomembrane, a monomeric eardrum, or atrophic scar, however, typically is thinner than the surrounding eardrum because the central, fibrous layer of the eardrum does not regenerate. A monomeric or atrophic eardrum is visible otoscopically, and in some cases, is so thin that it is difficult to differentiate from a perforation unless the eardrum is placed under pressure during pneumatic otoscopy or tympanometry.

In tympanosclerosis, an inflammatory process in the middle ear produces calcified plaques in the middle-ear space and on the medial surface of the eardrum (Forseni et al., 1997). With involved cases affecting the middle-ear space, conductive hearing loss can result that can be challenging to treat surgically. In severe cases, tympanoplasty with or without ossiculoplasty may be required (Bayazit et al., 2004). Frequently, however, tympanosclerotic plaques are a sequela of middle-ear disease in childhood, and are of little medical consequence in adulthood.

Figure 9.9C shows tympanograms from DJC, a 27-year-old male with a history of COM until age 7. During childhood, he was treated with multiple myringotomies without

FIGURE 9.10 Peak $200B_{tm}$ (open squares) and $200G_{tm}$ (open diamonds) as a function of probe-tone frequency from a patient with otosclerosis (A) and from a patient with ossicular discontinuity (B). For comparison, normal $200B_{tm}$ and $200G_{tm}$ functions are shown in both panels by the solid and dashed lines, respectively.



tympanostomy tubes (TT). Children with a history of TT in childhood, whether or not atrophic scarring is obvious during otoscopy, tend to have higher than average $200Y_{tm}$ in adulthood (de Beer et al., 2005). At age 20, DJC dove into a swimming pool and perforated the left eardrum, which healed spontaneously. Otoscopy revealed atrophic scarring in the posterior-superior quadrant of the left eardrum; no significant conductive component was measured.

Panel C shows mass-controlled tympanograms typical of an ear with eardrum pathology. Y_{tm} at 226 Hz is abnormally high (>4.0 acoustic mmho) and exceeded the upper range of the equipment. At 678 Hz, the tympanograms are mass controlled and irregular as they were in the two cases of ossicular discontinuity. The main difference in tympanogram morphology in cases with ossicular discontinuity versus eardrum pathology is the TW or notch width. 226 Hz TW typically is narrower in ears with eardrum pathology in comparison with ossicular discontinuity. Although all three tympanograms in this figure are abnormally mass controlled at 678 Hz, the pressure interval between the outermost susceptance extrema is abnormal in ossicular discontinuity (>125 daPa) but normal (80 daPa) in the ear with eardrum pathology. Similar tympanogram patterns are recorded from ears that have undergone stapedectomy or stapedotomy for surgical treatment of otosclerosis (Colletti, 1976; Colletti et al., 1993; Liden et al., 1970). Audiometry, case history, and otoscopy are crucial in differentiating eardrum pathology and post-surgical ears from ossicular discontinuity.

Feldman (1974) revealed an important consequence of eardrum pathology: high-admittance eardrum pathology can completely mask a more central low-admittance pathology. In an ear with ossicular fixation and eardrum pathology, for example, the high admittance eardrum will completely dominate tympanometry measures and mask the effect of ossicular fixation. [See Margolis (1981) for a discussion of input impedances in parallel versus series circuits.] The importance of otoscopy in interpreting tympanograms cannot be overstated.

OTOSCLEROSIS VERSUS OSSICULAR DISCONTINUITY

Figures 9.8B and 9.9A showed 226 and 678 Hz tympanograms in patients with otosclerosis and ossicular discontinuity. Figure 9.10 shows an alternative analysis of peak compensated static acoustic admittance at 10 frequencies in these same two ears. Peak $200B_{tm}$ (squares) and $200G_{tm}$ (diamonds) as a function of frequency are displayed for DK with otosclerosis (Panel A) and RP with ossicular discontinuity (Panel B). For comparison, $200B_{tm}$ and $200G_{tm}$ functions from the normal 40 year old depicted in Figures 9.4 to 9.6 are shown in both panels by the solid and dashed lines, respectively. Two points on the functions that are easy to compare are the frequencies corresponding to phase angles of 45° and 0° (resonance). Recall that the admittance vector is at 45° when $200B_{tm}$ and $200G_{tm}$ are equal, i.e., the frequency where the two-component functions cross near 580 Hz. Resonance (f_0) occurs when mass and stiffness susceptance are equal in magnitude and sum to 0 acoustic mmho, i.e., the frequency where the B functions cross the zero axis near 850 Hz. In contrast, the ear with otosclerosis in Panel A remained stiffness controlled over a broader frequency range than normal. This is reflected in higher frequencies for both the 45° point (~ 700 Hz) and resonance (~ 1020 Hz). When plotted in this format, the results show that the ear with otosclerosis (Fig. 9.10A) is more clearly separated from the normal ear at high probe frequencies, and that the separation is smallest for the most commonly used probe frequency of 226 Hz.

In contrast to otosclerosis, the ear with ossicular discontinuity (Fig. 9.10B) is mass controlled at a lower than normal frequency. The 45° point occurred near 345 Hz and resonance occurred near 500 Hz. There is clear differentiation between otosclerosis with an f_0 of 1020 Hz and disarticulation with an f_0 of 500 Hz.

Although the ear with the monomeric eardrum in Figure 9.9C is not shown in Figure 9.10, his B_{tm} and G_{tm} functions are indistinguishable from the ear with ossicular discontinuity. This demonstrates a strong advantage of recording tympanograms rather than only one point (peak)

from the tympanogram. Even a cursory glance at tympanogram shapes at 678 Hz differentiates all three patients from normal in Figure 9.9, with no calculation needed. When only peak compensated static acoustic admittance is plotted, the shape of the tympanogram and broadness of the notching is lost. Notch width is particularly important and helps to differentiate between an ear with eardrum pathology and one with ossicular discontinuity. As subsequent examples will show, the width and depth of the notch in 678 Hz tympanograms is relied on to differentiate between normal and abnormal mass-controlled ears.

Otitis Externa

Another example of mass-related pathology that can have a marked effect on high-frequency tympanometry, but little effect on hearing, is otitis externa (OE). Ear-canal infections, or OE, often occur in hearing-aid wearers or after water exposure, giving rise to the lay term “swimmer’s ear.” Common OE presents with significant otalgia, particularly with manipulation of the pinna. Moist, edematous skin with squamous debris or purulent material typically is evident with otoscopy (see examples in Ballachanda, 1995; Hawke and McCombe, 1995; Kavanagh, 2006; Sullivan, 2006). Bacteria are the most common infecting agent, although fungus is often found in chronic cases. Chronic dermatologic conditions of the external auditory canal skin can give rise to itching and occasionally acute infection. A history of trauma, either with a fingertip or Q-tip, preceding the infection often is elicited. Treatment is with appropriate ototopical antibiotics, occasionally with the addition of steroids, as well as water precautions, and pain medications. Diabetic patients with OE require medical referral. These patients may develop a more aggressive form of OE, malignant OE, in which the infection extends beyond the soft tissues of the ear canal and into the surrounding bone. Skull-base osteomyelitis is a sequela and often causes cranial-nerve deficits and even death if not treated expeditiously.

Figure 9.11A shows tympanograms from MR, a 49-year-old male swimmer who reported recurrent otalgia and discharge from his right ear over the past 2 years. Otoscopy revealed otitis externa with greenish discharge in the ear canal and against the eardrum. The audiogram in the inset shows a definite mass tilt. The 226 Hz tympanogram has a $200Y_{tm}$ of 0.35 acoustic mmho with a broad TW. This tympanogram demonstrates a problem encountered in calculating TW from asymmetric tympanograms. In this example, the negative tail never quite reaches the one-half peak value so TW cannot be calculated. The 678 Hz tympanogram shows a shallow, mass-controlled 5B3G pattern with an abnormal notch width in excess of 100 daPa. This pattern is typical of a mass-loaded eardrum and indicates that the discharge is against the eardrum.

Figure 9.11B shows another case of abnormal mass. These tympanograms were recorded from a 76-year-old male (JP) who was seen the day before for earmold impressions.

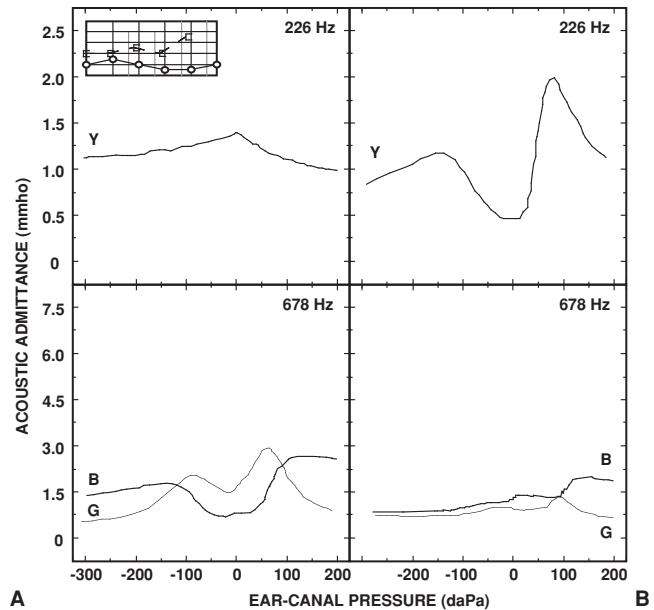


FIGURE 9.11 Susceptance (B), conductance (G), and admittance (Y) tympanograms at 226 Hz (top panels) and 678 Hz (bottom panels) recorded from MR with otitis externa and debris against the eardrum (A) and from JP with blood filling the ear canal (B); the audiogram for MR is shown in the inset but no audiogram was available for JP.

The patient returned to the clinic the next day and reported that on the drive home from his appointment, he noted bleeding from his right ear. Otoscopy showed fresh blood filling much of the ear canal and obscuring a view of the eardrum. Tympanometry was completed to document the status of the eardrum. The 226 Hz acoustic admittance tympanogram shows a V_{ea} of 1.1 cm^3 and verified an intact eardrum. What is so unusual in this case is that the 226 Hz tympanogram was broadly notched and mass controlled; notching typically is recorded only at high-probe frequencies. This patient was immediately referred to ENT. Examination under a microscope revealed bleeding along the inferior, lateral wall of the ear canal; the lesion was cauterized with AgNO.

Otitis Media

Otitis media (OM) associated with immature Eustachian tubes is the most common illness among children receiving medical care. Acute OM presents with otalgia and fever whereas COM presents with a conductive hearing loss and aural fullness. Otoscopy findings can range from bulging, erythematous eardrums with purulence in the middle ear to retracted, opaque eardrums with air-fluid levels or even bubbles in the middle ear. Recurrent or persistent disease is often an indication for myringotomy with TT. Acute OM is more appropriately treated with observation if it is non-suppurative, but is treated with antibiotics if fever and purulent material are noted in the middle ear.

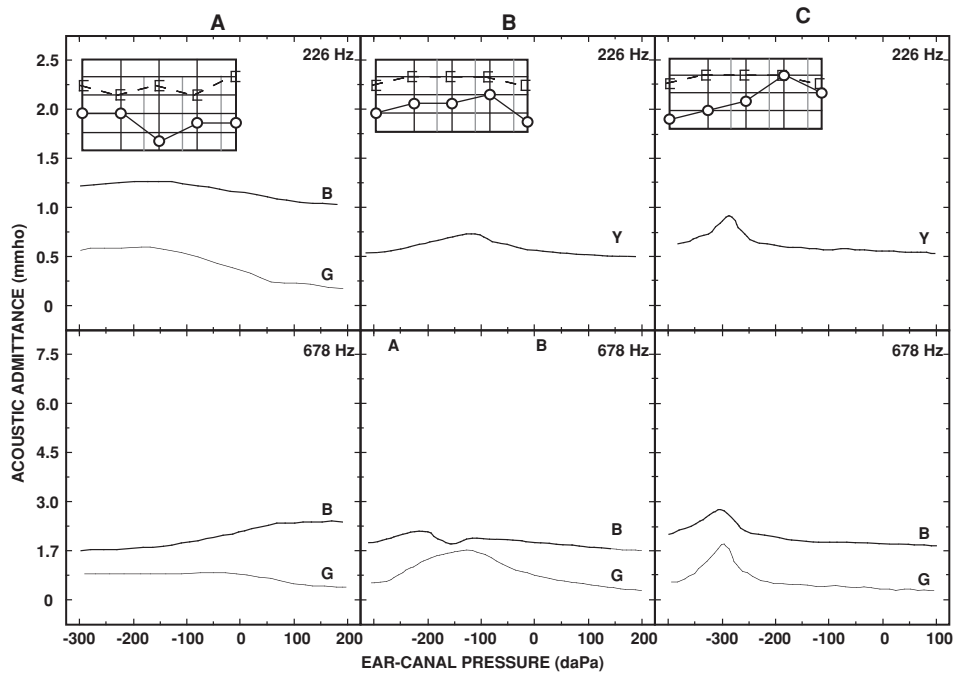


FIGURE 9.12 Susceptance (B), conductance (G), and admittance (Y) tympanograms at 226 Hz (*top panels*) and 678 Hz (*bottom panels*) recorded during three stages of otitis media, SC with a fluid-filled middle ear in Panel A; and CC with resolving, low level middle-ear fluid in Panel B, improving to resolution of middle-ear fluid with a slightly retracted eardrum in Panel C. The corresponding audiograms are shown as insets.

An adult who presents with a chronic serous otitis media should be evaluated for nasopharyngeal pathology. Masses in the nasopharynx can exert extrinsic pressure on the Eustachian tube and cause obstruction. Careful endoscopic examination or imaging can help to rule out significant pathology. Adults having undergone radiation therapy to the head and neck, especially the nasopharynx, parotid, or temporal bone often develop chronic serous otitis media as a result of deleterious effects on the Eustachian tube by the radiation therapy. Treatment often consists of placement of a TT.

Figure 9.12A shows tympanograms recorded from SC, a 6-year-old male with a history of bilateral MEE. Although this tympanogram pattern typically is described as flat with a normal V_{ea} of 1.0 cm^3 , the B_a and G_a tympanograms have a distinctive sloping pattern. Feldman (1976b) describes this tympanogram pattern, which is indicative of MEE, as “converging 220 Hz and 660 Hz susceptance tympanograms”, (p. 126). That is, the 226 Hz tympanograms rise and the 678 Hz tympanograms fall with decreasing pressures. Berry et al. (1975) reported that this distinctive sloping pattern at 660 Hz was indicative of effusion in 100% of the ears. They further concluded that 660 Hz tympanometry was a better indicator of MEE than 220 Hz.

Figure 9.12B and 9.12C show tympanograms recorded from my daughter CC when she was four years old. She had a cold and fever and briefly complained of an earache. All symptoms resolved within a few days. A short time later, her pediatrician saw her for a preschool check up and noted

that she had fluid in her right ear. She not only had flat tympanograms similar to those in Panel A, but she also had 45–55 dB air-bone gaps in the right ear; her left ear was normal. Although her hearing improved slightly and air-bone gaps decreased to 25 dB, her tympanograms remained flat for several months. After four months with no resolution of the fluid, she was scheduled to have TT. A few days prior to the scheduled surgery, the tympanograms shown in Figure 9.13B were recorded. The 226 Hz tympanogram was abnormally stiff (<0.3 acoustic mmho) with an abnormally wide TW (>250 daPa). The 678 Hz tympanograms indicated that finally there was some air in the middle-ear space, although fluid still was present as evidenced by the shallow, broadly notched 678 Hz tympanograms and by the conductive hearing loss greatest in the high-frequency range. Two weeks later, the tympanograms shown in the Figure 9.12C were recorded. Note that the hearing loss improved in the high frequencies, indicating resolution of the mass-loading fluid. Negative pressure of -300 daPa was present, resulting in small low-frequency air-bone gaps, but the amplitude of the tympanogram at 226 Hz was normal and the amplitude relationship between susceptance and conductance at 678 Hz was normal, i.e., $200G_{tm}$ was greater than $200B_{tm}$ and phase angle was $<45^\circ$. Both the audiogram and tympanograms in Figure 9.12C indicate that the pathology was strictly negative middle-ear pressure with no significant fluid.

Caution, however, must be taken in inferring etiology when interpreting tympanograms. Flat tympanograms with

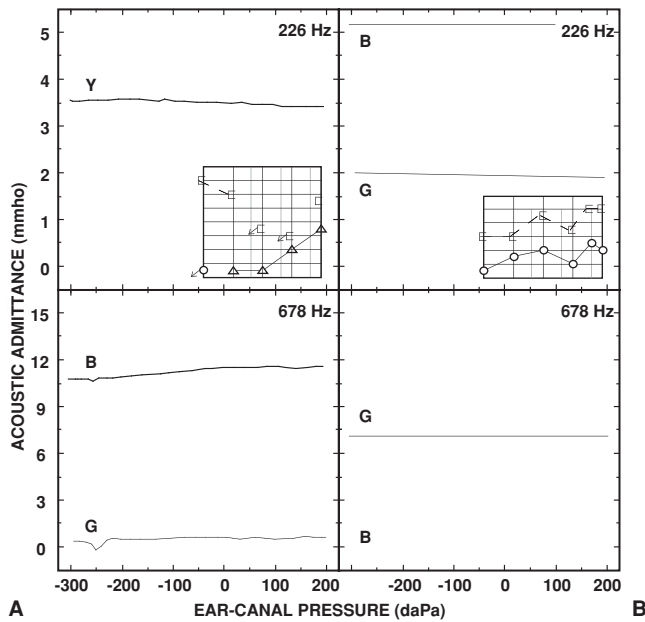


FIGURE 9.13 Susceptance (B), conductance (G), and admittance (Y) tympanograms at 226 Hz (*top panels*) and 678 Hz (*bottom panels*) recorded from AD with a perforated eardrum following ear lavage in an ear with a history of chronic otitis media and multiple TT (Panel A), and from BH with a traumatic eardrum perforation from a high-powered squirt gun (Panel B).

normal volumes are not always indicative of middle-ear effusion. Other causes of flat tympanograms include a thickened eardrum, malleus fixation, and middle-ear tumors that are discussed in a subsequent section. In other words, if the motion of the eardrum is completely restricted for any reason, the tympanograms will be flat.

Eardrum Perforation

Eardrum perforation can result as a complication of OM, from trauma, or from extruded TT. Historically, there has been little agreement on the relationship between the size and location of an eardrum perforation and the degree and configuration of hearing loss. With the exception of trauma, eardrum perforation rarely occurs as an isolated pathology. When perforation occurs as a sequela of OM, for example, other middle-ear structures such as the ossicles, middle-ear space, and mastoid air cell system also are involved and contribute to the magnitude of the conductive component. Most studies, however, do agree that the larger the perforation, the larger the air-bone gaps (Voss et al., 2001).

At least four different tympanogram patterns have been recorded from ears with eardrum perforation. Figure 9.13A shows tympanograms from AD, a 19-year-old male with a history of COM and multiple TT as a child. He reported to the clinic with a large, 40% posterior-inferior perforation of the right eardrum that occurred following cerumen removal by

lavage. When a flat tympanogram is recorded, the first observation should be the V_{ea} at 200 daPa. Remember from the calibration discussion that the volume of an enclosed cavity can be represented as pure acoustic compliance, and therefore, is most accurately estimated using a low-frequency probe tone such as 226 Hz. Also recall that an ideal hard walled cavity has $G_a = 0$ acoustic mmho, and therefore, V_{ea} will be the same whether estimated from a B_a or Y_a tympanogram. In this example, a V_{ea} of 3.5 cm^3 was estimated from $200Y_a$. When the eardrum is perforated, the volume is comprised of the ear canal, the middle-ear space, and the mastoid air cell system. $V_{ea} > 2.5 \text{ cm}^3$ in an adult male is consistent with eardrum perforation.

Tympanograms in Figure 9.13A are typical of those recorded in ears with a history of COM. V_{ea} estimated at both 226 and 678 Hz are similar to those measured in calibration cavities. V_{ea} estimated from the 678 Hz B_a tympanogram at 200 daPa is 3.7 cm^3 (i.e., 11.1 acoustic mmho divided by 3) and is essentially equal to the corresponding volume estimate of 3.5 cm^3 at 226 Hz. Conductance at 678 Hz is close to 0 acoustic mmho. In other words, this ear functioned like a calibration cavity (refer to Figure 9.1). This finding most likely occurs because the mastoid air cell system is poorly aerated in ears with COM. That is, the mastoid air cell system is functionally shut off from the rest of the middle ear so that the volume of the ear canal plus the middle-ear space closely resembles a hard walled cavity with a uniform cross-sectional area.

In contrast, Figure 9.13B depicts a traumatic eardrum perforation in BH, a 30-year-old male who was hit in the ear with water from a high powered squirt gun. Otologic exam showed a 50% perforation in the posterior-inferior quadrant. The inset audiogram shows a flat 25 dB conductive component. At 226 Hz, the B_a tympanogram was flat and exceeded equipment limits (i.e., >7 acoustic mmho) and G_a was flat at 2 acoustic mmho. This ear did not function like a calibration cavity, particularly at the high-probe frequency. At 678 Hz, B_a in a hard-walled cavity is three times greater than at 226 Hz, and G_a remains at 0 acoustic mmho. The 678 Hz B_a and G_a tympanograms in this ear, however, are the opposite of readings expected in a hard-walled cavity. The B_a tympanogram in this perforated ear was flat and off scale in the *negative* direction, and the G_a tympanogram was flat at 7.0 acoustic mmho.

This “atypical” pattern first was noted in 1982 when recording tympanograms with an Otolaryngology resident on a normal temporal bone following perforation of the eardrum. This pattern can only be seen if the two rectangular components of acoustic admittance are recorded and would not have appeared out of the ordinary if only Y_a had been recorded. Recall that Y_a is always larger than the larger of the two rectangular components (B_a and G_a), so the tympanogram would mirror the G_a tympanogram rather than the B_a tympanogram as typically occurs. The maximum susceptance reading at 226 Hz indicates that the volume being measured is very large. The minimum susceptance reading

at 678 Hz indicates that the volume being measured cannot be modeled as pure acoustic compliance. In an ear with a traumatic eardrum perforation, the volume being estimated is comprised of the ear canal, the middle-ear space, and narrow aditus leading to the mastoid air cell system. This combined volume does not begin to approximate a volume with a uniform cross-sectional area. These irregular cavities and narrow aditus introduce mass and resistance, and therefore, make an accurate estimate of volume impossible, particularly when using high-frequency probe tones.

This atypical tympanogram pattern is indicative of a “healthy” middle-ear transmission system and functional mastoid air cell system. Ears with large middle-ear volumes and a large, communicating mastoid air cell system have a better prognosis for successful tympanoplasty and less chance for recurrence of middle-ear disease and post-surgical complications than patients with small volumes (Andreasson, 1977; Holmquist, 1970; Holmquist and Bergstrom, 1977; Lindeman and Holmquist, 1982).

A second “bizarre” tympanogram pattern also was identified while making recordings on temporal bones. The question posed by a resident was, how large does a perforation have to be to result in a flat tympanogram? The resident made a tiny hole in the eardrum, decided it was too large, and then used a needle to rough up the edges of the perforation and draw them closer together. Surprisingly, the tympanogram, which was recorded from 200 daPa to –300 daPa, was not flat, but had a normal peak at an extreme *positive* pressure of 150 daPa. When the direction of the recording was reversed, the peak shifted to an extreme *negative* pressure of –250 daPa. When a high positive pressure is applied to an ear with a small perforation, the pressure stresses the perforation and allows positive pressure to momentarily flow into the middle-ear space. Similarly, an extreme negative starting pressure again stresses the perforation and allows negative pressure to enter the middle ear space. See Figure 12.7 in Fowler and Shanks (2002) for an example of this tympanogram pattern. Be alert to the presence of pinhole perforations. If a significant positive TPP is recorded, it is a simple matter to reverse directions and record a Y 226 Hz tympanogram using ascending pressures to rule out the presence of a pinhole perforation.

Middle Ear Tumors

Flat tympanograms also can be recorded from ears with middle-ear masses or tumors. Two examples are cholesteatoma with or without eardrum perforation and glomus tumor. The shape of the tympanograms is dependent on how much eardrum movement is restricted.

CHOLESTEATOMA

Cholesteatoma is a disorder characterized by squamous (skin) epithelium being trapped in the middle ear or mastoid. It can occur as a sequela of chronic suppurative otitis media by a migration of squamous epithelium from the ear

canal through an eardrum perforation, or from a deep retraction of the pars flaccida or other weakened area of the eardrum as a result of chronic Eustachian tube dysfunction. Cholesteatoma also can arise congenitally by means of an epithelial rest of tissue being trapped behind the eardrum during embryologic development (Shohet and de Jong, 2002). In either event, cholesteatoma has locally invasive properties and can destroy adjacent bone including the ossicles, middle and posterior fossa dura plates, and the bony labyrinth. A bacterial superinfection amplifies the destructive properties of a cholesteatoma and can present as chronic otorrhea refractory to antibiotics; it is usually painless. The treatment is surgical resection with a tympanoplasty or tympanomastoidectomy procedure and has a recurrence rate of between 30% and 50%.

Figure 9.14A shows the fourth tympanogram pattern that can be recorded from an ear with a perforated eardrum. BM is a 62-year-old male referred for pre-operative testing prior to undergoing a tympanomastoidectomy in the left ear. He had a history of eardrum perforation and COM, accompanied by periodic drainage for more than 20 years. His audiogram showed a maximum low-frequency conductive hearing loss with decreasing air-bone gaps with increasing frequency. Surgical findings revealed a cholesteatoma filling the middle-ear space and extending into the attic. The malleus was eroded and the incus was absent. The

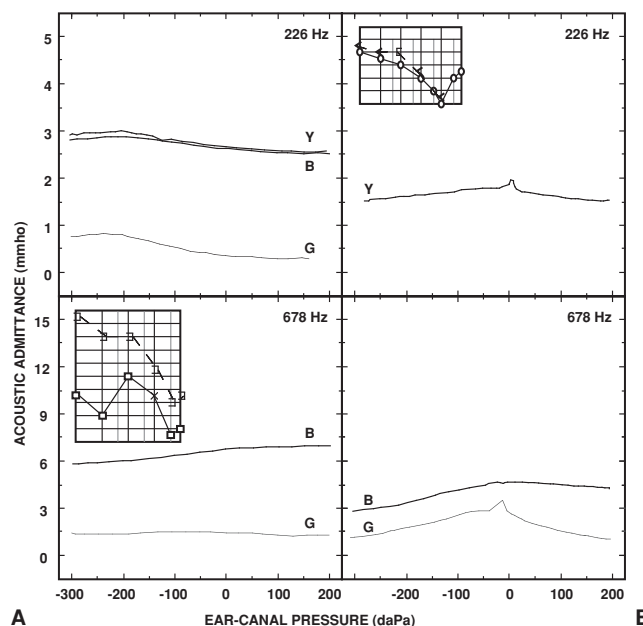


FIGURE 9.14 Susceptance (B), conductance (G), and admittance (Y) tympanograms at 226 Hz (*top panels*) and 678 Hz (*bottom panels*) recorded from BM with a perforated eardrum and cholesteatoma filling the middle-ear space and extending into the attic (Panel A) and from MC with a pulsating glomus tumor visible behind the eardrum (Panel B).

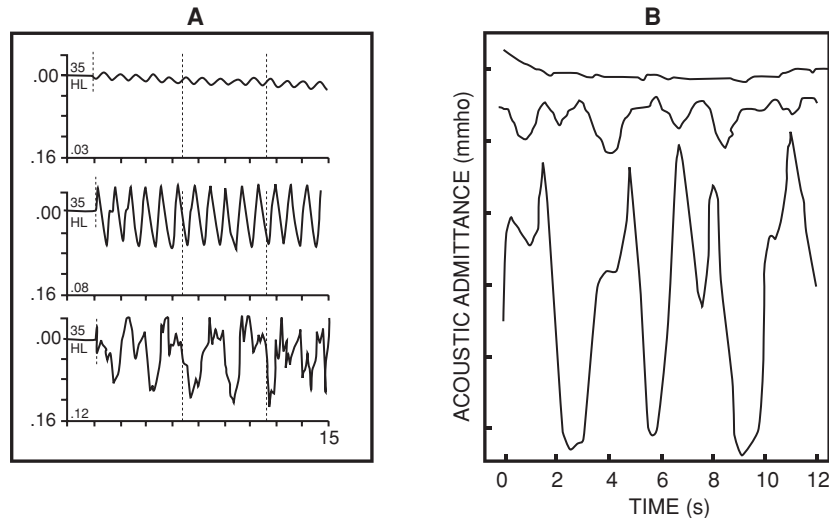


FIGURE 9.15 Changes in acoustic admittance at TPP recorded over 12-15 s in the acoustic reflex mode with the acoustic reflex stimulus at a minimum. The top trace in Panel A shows normal vascular perturbations, the middle trace shows high-magnitude vascular perturbations in MC with the glomus tumor in Figure 9.14B, and the bottom trace shows very large perturbations coincident with breathing in a patient with a patulous Eustachian tube. Panel B shows three tracing from RE with a patulous Eustachian tube. The top trace was recorded while holding his breath, the middle trace while breathing normally, and the bottom trace during forced, closed mouth breathing.

cholesteatoma was removed and ossicular reconstruction was planned for a second surgery. Y_a , B_a and G_a tympanograms at 226 and B_a and G_a tympanograms at 678 Hz are shown in Figure 9.14A. V_{ea} estimated from either the 226 Hz Y_a or B_a tympanogram was 2.5 cm^3 and was at the upper range for normal ear-canal volume in adult males. Although BM had a known eardrum perforation, the cholesteatoma filled the middle-ear space and effectively cut off the middle ear from the ear canal. This is not an uncommon finding with chronic middle-ear disease and serves as a caution when interpreting flat tympanograms. A flat tympanogram with a 226 Hz volume estimate exceeding 1.0 cm^3 in children <7 years of age and exceeding 2.5 cm^3 in adults indicates a perforated eardrum or patent TT, but a flat tympanogram with a normal V_{ea} does not necessarily rule out a perforated eardrum. Tympanometry in conjunction with otoscopy and an otologic exam, however, can be informative and alert the examiner to a space-occupying lesion.

GLOMUS TUMOR

Glomus tumors or paragangliomas are typically benign tumors derived from supporting nerve tissue around the jugular bulb or in the middle ear. Although approximately 95% of glomus tumors are benign and slow growing, they are highly vascular and invasive. Patients often present with pulsatile tinnitus and with conductive or complete hearing loss in approximately 61% of the patients (Manolidis et al., 1999). Otoscopy may reveal a red mass behind the tympanic membrane. Because of their location, these tumors can cause significant morbidity including difficulties swallowing and phonating. Treatment is usually surgical resection, although radiation

therapy may be reserved for patients unable to undergo a prolonged surgical procedure.

MC is a 72-year-old female who complained of aural fullness and a muffled quality to sound in her right ear for the past month. She also had roaring, pulsating tinnitus with vertigo and nausea. Otoscopy revealed a pulsating mass behind the right eardrum. A subsequent MRI showed a 2.4-cm mass in the right temporal bone extending into the right middle ear. Only minimal low-frequency air-bone gaps were measured. The tympanograms displayed in Figure 9.14B were atypical with low normal Y_{tm} at 226 Hz and restricted TW, but the recordings did not reflect the pulsating eardrum noted on otoscopic exam. Others have reported tympanograms in ears with glomus tumors ranging from flat to pulsatile (see Fig. 6.17 in Feldman, 1976b).

In this case, the pulsation was detected when the instrument was put in the AR mode. The AR mode can be used as a very sensitive extension of tympanometry. With the acoustic-reflex stimulus turned off during tone-decay testing, static acoustic admittance at TPP can be measured over time, typically 12 to 15 seconds. Figure 9.15 shows two applications of the AR mode. The top trace in Panel A shows normal vascular perturbations. The rate of oscillation in the top trace is 56 beats per minute and indicates a slight vascular change. The second trace was recorded from MC who complained of pulsatile tinnitus associated with the glomus tumor. When switched to the more sensitive AR mode, the vascular pulsation became very obvious and was greater in magnitude than normally recorded. In contrast to the vascular changes in the top two traces, the bottom trace of Figure 9.15A shows

perturbations coincident with breathing in another patient with a patulous Eustachian tube.

Patulous Eustachian Tube

While most Eustachian tube (ET) dysfunction involves a ET that does not open properly, a patulous ET is one that remains open. Movement of the eardrum coincident with breathing can sometimes be detected during otoscopy or noted during tympanometry and AR testing. Patients sometimes complain of aural fullness and autophonia, a sensation of hearing one's own voice louder than normal, although in many cases, the patient is symptom free. Patulous Eustachian tube occurs in approximately 7% of adults and frequently is misdiagnosed as OM because of the similarity in symptoms (Henry and DiBartolomeo, 1993). Precipitating factors include sudden weight loss, inappropriate use of decongestants, and fatigue. Some cases of patulous ET are transient and self-resolving. In persistent cases, treatment is challenging and not always effective. Several methods have been proposed to make the diagnosis, including video endoscopy of the nasopharyngeal opening of the ET and even audiometry of tones presented in the nasal cavity (Kano et al., 2004; Poe et al., 2001; Virtanen and Palva, 1982). The easiest method, however, is a tympanometry procedure that takes advantage of increased instrument sensitivity in the acoustic-reflex mode (Finkelstein et al., 1988; Henry and DiBartolomeo, 1993).

Figure 9.15B shows RE, a 40-year-old male who complained of decreased hearing in both ears and a constant, mild

headache for the past 3 to 4 months. Perturbations coincident with respiration were noted when attempting to measure AR thresholds. To verify patulous Eustachian tube, three traces were obtained at peak admittance with the reflex stimulus turned off. The top flat trace was recorded with RE holding his breath. The second trace was obtained during normal breathing, and the third trace was recorded during forced breathing with the mouth closed. Four to six large perturbations should be recorded during the 12- to 15-s time window. Occasionally, movement of the eardrum coincident with respiration can be noted otoscopically. One case encountered with a patulous Eustachian tube had a large atrophic area on the eardrum that moved in and out with normal respiration. That patient had no aural complaints and was not treated.

SUMMARY AND CONCLUSIONS

Universal hearing screening has had a positive impact on tympanometry and has focused renewed attention on high frequency measurements. Hopefully, the reader will finally be convinced of the power of high-frequency and MFT tympanometry in evaluating middle-ear disease. In almost every case, pathology that is either absent or subtle at 226 Hz is accentuated at 678 Hz or 1000 Hz. Prepare for the next generation of measurements by utilizing the MFT options already available on commercial instruments and realizing their advantages and applications.

REFERENCES

- American Academy of Audiology. (1997) Identification of hearing loss & middle-ear dysfunction in preschool & school-age children. *Audiol Today*. 9, 21–23.
- American National Standards Institute. (1987) Specifications for instruments to measure aural acoustic impedance and admittance (aural acoustic immittance). ANSI S3.39–1987. New York: American National Standards Institute.
- American Speech-Language-Hearing Association. (1979) Guidelines for acoustic immittance screening of middle-ear function. *ASHA*. 21, 283–288.
- American Speech-Language-Hearing Association. (1990) Guidelines for screening for hearing impairment and middle-ear disorders. *ASHA*. 32, 17–32.
- American Speech-Language-Hearing Association. (1997) Guidelines for audiologic screening. Rockville MD: American Speech-Language-Hearing Association.
- Alberti P, Jerger J. (1974) Probe-tone frequency and the diagnostic value of tympanometry. *Arch Otolaryngol*. 99, 206–210.
- Andreasson L. (1977) Correlation of tubal function and volume of mastoid and middle ear space as related to otitis media. *Acta Otolaryngol*. 83, 29–33.
- Andreasson L, Harris S. (1979) Middle ear mechanics and Eustachian tube function in tympanoplasty. *Acta Otolaryngol*. 360, 141–147.
- Antonio SM, Don D, Doyle WJ, Alper CM. (2002) Daily home tympanometry to study the pathogenesis of otitis media. *Pediatr Infect Dis J*. 21, 882–885.
- Ars B. (1989) Organogenesis of the middle ear structures. *J Laryng Otol*. 103, 16–21.
- Babu S, Seidman MD. (2004) Ossicular reconstruction using bone cement. *Otol Neurotol*. 25, 98–101.
- Ballachanda BB. (1995) The Human Ear Canal: Theoretical Considerations and Clinical Applications Including Cerumen Management. San Diego: Singular Publishing Group.
- Bayazit YA, Ozer E, Kara C, Gokpinar S, Kanlikama M, Mumbuc S. (2004) An analysis of the single-stage tympanoplasty with overlay grafting in tympanosclerosis. *Otol Neurotol*. 25, 211–214.
- Bel J, Causse J, Michaux P. (1975) Paradoxical compliances in otosclerosis. *Audiology*. 14, 118–129.
- Bennett M. (1975) Acoustic impedance bridge measurements with the neonate. *Brit J Audiol*. 9, 117–124.
- Bennett MJ, Weatherby LA. (1979) Multiple probe frequency acoustic reflex measurements. *Scand Audiol*. 8, 233–239.
- Bentler RA. (1989) External ear resonance characteristics in children. *J Speech Hear Res*. 54, 264–268.
- Beranek LL. (1954) *Acoustics*. New York: McGraw-Hill.
- Berry QC, Andrus WS, Bluestone CD, Cantekin EI. (1975) Tympanometric pattern classification in relation to middle ear effusion. *Ann Otol*. 84, 56–63.
- Blood I, Greenberg HJ. (1977) Acoustic admittance of the ear in the geriatric person. *J Am Aud Soc*. 2, 185–187.
- Bluestone CD. (2004) Studies in otitis media: Children's Hospital of Pittsburgh-University of Pittsburgh progress report-2004. *Laryngoscope*. 114: 1–26.
- Brooks DN. (1968) An objective method of determining fluid in the middle ear. *Int Audiol*. 7, 280–286.

- Brooks DN. (1969) The use of the electro-impedance bridge in the assessment of middle ear function. *Int Audiol*. 8, 563–565.
- Burke K, Nilges T. (1970) A comparison of three middle ear impedance norms as predictors of otosclerosis. *J Aud Res*. 10, 52–58.
- Calandrucchio L, Fitzgerald TS, Prieve BA. (2006) Normative multifrequency tympanometry in infants and toddlers. *J Am Acad Audiol*. 17, 470–480.
- Carhart R. (1962) Effects of stapes fixation on bone-conduction response. In: *Otosclerosis*. Boston: Little & Brown, 175–197.
- Cherukupally SR, Merchant SN, Rosowski JJ. (1998) Correlations between pathologic changes in the stapes and conductive hearing loss in otosclerosis. *Ann Otol Rhinol Laryngol*. 107, 319–326.
- Chesnutt B, Stream RW, Love JT, McLarey DC. (1975) Otoadmittance measures in cases of dual ossicular disorders. *Arch Otolaryn*. 101, 109–113.
- Chole RA, McKenna M. (2001) Pathophysiology of otosclerosis. *Otol Neurotol*. 22, 249–257.
- Ciarlo A, Garavello W, Leva M, Graziano B, Gaini RM (2005) Reversed ipsilateral acoustic reflex: A study on subjects treated with muscle relaxants. *Ear Hear*. 26, 96–103.
- Colletti V. (1975) Methodological observations on tympanometry with regard to the probe tone frequency. *Acta Otolaryn*. 80, 54–60.
- Colletti V. (1976) Tympanometry from 200 to 2000 Hz probe tone. *Audiology*. 15, 106–119.
- Colletti V. (1977) Multifrequency tympanometry. *Audiology*. 16, 278–287.
- Colletti V, Fiorino FG, Sittoni V, Policante A. (1993) Mechanics of the middle ear in otosclerosis and stapedoplasty. *Acta Otolaryngol*. 113, 637–641.
- Creten WL, Van Camp KJ. (1974) Transient and quasi-static tympanometry. *Scand Audiol*. 3, 39–42.
- Creten WL, Van de Heyning PH, Van Camp KJ. (1985) Immittance audiometry: Normative data at 220 and 660 Hz. *Scand Audiol*. 14, 115–121.
- de Beer B, Snik A, Schilder A, Graamans K, Zielhuis GA. (2005) The effect of otitis media in childhood on the development of middle ear admittance on reaching adulthood. *Arch Otolaryngol Head Neck Surg*. 131, 777–781.
- De Chicchis AR, Todd NW, Nozza RJ. (2000) Developmental changes in aural acoustic admittance measurements. *J Am Acad Audiol*. 11, 97–102.
- Decraemer WF, Creten WL, Van Camp KJ. (1984) Tympanometric middle ear pressure determination with two component admittance meters. *Scand Audiol*. 13, 165–172.
- Dieroff HG. (1978) Differential diagnostic value of tympanometry in adhesive processes and otosclerosis. *Audiol*. 17, 77–86.
- de Jonge R. (1986) Normal tympanometric gradient: A comparison of three methods. *Audiol*. 25, 299–308.
- Diamant M. (1965) The “pathologic size” of the mastoid air cell system. *Acta Otolaryn*. 60, 1–10.
- Dolan K. (1979) Mastoid pneumatization. In: Sade J, ed. *Secretory otitis media and its sequelae*. New York: Churchill Livingstone, 298–314.
- Eavey RD. (1993) Abnormalities of the neonatal ear: Otoloscopic observations, histological observations, and a model for contamination of the middle ear by cellular contents of amniotic fluid. *Laryngoscope*. 103, 1–31.
- Eby T, Nadol J. (1986) Postnatal growth of the human temporal bone: Implication for cochlear implants in children. *Ann Otol Rhinol Laryngol*. 95, 356–364.
- Eliachar I, Northern JL. (1974) Studies in tympanometry: Validation of the present technique for determining intra-tympanic pressures through the intact eardrum. *Laryngoscope*. 84, 247–255.
- Elner A, Ingelstedt S, Ivarsson A. (1971) The elastic properties of the tympanic membrane. *Acta Otolaryng*. 72, 397–403.
- Feldman AS. (1974) Eardrum abnormality and the measurement of middle-ear function. *Arch Otolaryn*. 99, 211–217.
- Feldman AS. (1976a) Tympanometry: Application and interpretation. *Ann Otol Rhinol Laryngol*. 85, 202–208.
- Feldman AS. (1976b) Tympanometry—procedures, interpretations and variables. In: Feldman AS, Wilbur LA, eds. *Acoustic Impedance and Admittance: The Measurement of Middle Ear Function*. Baltimore: Williams & Wilkins, 103–155.
- Ferekidis E, Vlachou S, Douniadakis D, Apostolopoulos N, Adamopoulos G. (1999) Multiple-frequency tympanometry in children with acute otitis media. *Otolaryn-Head Neck Surg*. 121, 797–801.
- Fiellau-Nikolajsen M. (1983) Tympanometry and secretory otitis media. Observations on diagnosis, epidemiology, treatment, and prevention in prospective cohort studies of three-year-old children. *Acta Otolaryn Suppl*. 394, 1–73.
- Finkelstein Y, Talmi Y, Rubel Y, Zohar Y. (1988) An objective method for evaluation of the patulous Eustachian tube by using the middle ear analyzer. *Arch Otolaryn*. 114, 1134–1138.
- Fisch U. (1980) *Tympanoplasty and Stapedectomy. A manual of techniques*. New York: Thieme-Stratton.
- Flisberg K, Ingelstedt S, Ortegren U. (1963) On middle ear pressure. *Acta Otolaryng*. 182, 43–56.
- Forseni M, Eriksson A, Bagger-Sjoberg D, Nilsson J, Hultcrantz M. (1997) Development of tympanosclerosis: Can predicting factors be identified? *Am J Otol*. 18, 298–303.
- Fowles CG, Shanks JE. (2002) Tympanometry. In: Katz J, ed. *Handbook of Clinical Audiology*. Baltimore: Lippincott Williams & Wilkins: 175–204.
- Funasaka S, Funai H, Kumakawa K. (1984) Sweep-frequency tympanometry: Its development and diagnostic value. *Audiology*. 23, 366–379.
- Funasaka S, Kumakawa K. (1988) Tympanometry using a sweep-frequency probe tone and its clinical evaluation. *Audiology*. 27, 99–108.
- Haapaniemi JJ. (1996) Immittance findings in school-aged children. *Ear Hear*. 17, 19–27.
- Hanks WD, Mortensen BA. (1997) Multifrequency tympanometry: effects of ear canal volume compensation on middle ear resonance. *J Am Acad Audiol*. 8, 53–58.
- Hanks WD, Rose KJ. (1993) Middle ear resonance and acoustic immittance measures in children. *J Speech Hear Res*. 36, 218–222.
- Haughton P. (1977) Validity of tympanometry for middle ear effusions. *Arch Otolaryn*. 103, 505–513.
- Hawke M, McCombe A. (1995) *Diseases of the Ear: A Pocket Atlas*. Ontario, Canada: Manticore Communication.
- Henry DF, DiBartolomeo JR. (1993) Patulous Eustachian tube identification using tympanometry. *J Am Acad Audiol*. 4, 53–57.
- Himelfarb MZ, Popelka GR, Shanon E. (1979) Tympanometry in normal neonates. *J Speech Hear Res*. 22, 179–191.
- Hirsch JE, Margolis RH, Rykken JR. (1992) Comparison of acoustic reflex and auditory brain stem response screening of high-risk infants. *Ear Hear*. 13, 181–186.
- Holmquist J. (1970) Size of mastoid air cell system in relation to healing after myringoplasty and to Eustachian tube function. *Acta Otolaryn*. 69, 89–93.
- Holmquist J, Bergstrom B. (1977) Eustachian tube function and size of the mastoid air cell system in middle ear surgery. *Scand Audiol*. 6, 87–89.
- Holte L. (1996) Aging effects in multifrequency tympanometry. *Ear Hear*. 17, 12–18.
- Holte L, Margolis RH, Cavanaugh RM. (1991) Developmental changes in multifrequency tympanograms. *Audiol*. 30, 1–24.
- Ikarashi H, Nakano Y. (1988) Relation between the onset of chronic middle ear inflammation and the development of the middle ear air cell system. *J Otorhinolaryngol Relat Spec*. 50, 306–312.
- Ivey R. (1975) Tympanometric curves and otosclerosis. *J Speech Hear Res*. 18, 554–558.
- Jackler RK, Schindler RA. (1984) Role of the mastoid in tympanic membrane reconstruction. *Laryng*. 94, 495–500.

- Jacobson JT, Mahoney TM. (1977) Admittance tympanometry in otosclerotic ears. *J Am Aud Soc.* 3, 91–98.
- Jerger J. (1970) Clinical experience with impedance audiometry. *Arch Otolaryn.* 92, 311–324.
- Jerger J, Jerger SJ, Mauldin L. (1972) Studies in impedance audiometry: I. Normal and sensorineural ears. *Arch Otolaryn.* 96, 513–523.
- Kano S, Kawase T, Baba Y, Sato T, Kobayashi T. (2004) Possible new assessment of patulous Eustachian tube function: audiometry for tones presented in the nasal cavity. *Acta Otolaryngol.* 124, 431–435.
- Kavanagh K. (2006) Eardrum & Middle Ear Photographs. Online at http://www.entusa.com/eardrum_and_middle_ear.htm.
- Keefe DH, Folsom RC, Gorga MP, Vohr BR, Buley JC, Norton SJ. (2000) Identification of neonatal hearing impairment: ear-canal measurements of acoustic admittance and reflectance in neonates. *Ear Hear.* 21, 443–461.
- Kei J, Allison-Levick J, Dockray J, Harrys R, Kirkegard C, Wong J, Maurer M, Hegarty J, Young J, Tudehope D. (2003) High-frequency (1000 Hz) tympanometry in normal neonates. *J Am Acad Audiol.* 14, 20–28.
- Keith R. (1973) Impedance audiometry with neonates. *Arch Otolaryngol.* 97, 465–467.
- Keith R. (1975) Middle ear function in neonates. *Arch Otolaryngol.* 101, 376–379.
- Kessler J, MacDonald CB, Cox LC. (1998) Bizarre “sawtooth” tympanogram in a patient with otitis media. *J Am Acad Audiol.* 9, 272–274.
- Kobayashi T, Okitsu T. (1986) Tympanograms in ears with small perforations of the tympanic membranes. *Arch Otolaryngol Head Neck Surg.* 112, 642–645.
- Kobayashi T, Okitsu T, Takasaka T. (1987) Forward-backward tracing tympanometry. *Acta Otolaryn.* 435:, 100–106.
- Koebell KA, Margolis RH. (1986) Tympanometric gradient measured from normal preschool children. *Audiology.* 25, 149–157.
- Liden G. (1969) Tests for stapes fixation. *Arch Otolaryn.* 89, 215–219.
- Liden G, Harford E, Hallen O. (1974) Automatic tympanometry in clinical practice. *Audiology.* 13, 126–139.
- Liden G, Peterson JL, Bjorkman G. (1970) Tympanometry: A method for analysis of middle ear function. *Acta Otolaryn.* 263, 218–224.
- Lildholdt T. (1980) Negative middle ear pressure: Variations by season and sex. *Ann Otol Rhinol Laryngol.* 89, 67–70.
- Lilly DJ. (1972a) Acoustic impedance at the tympanic membrane: A review of basic concepts. In: Rose D, Keating L, eds. *Proceedings of the Mayo Impedance Symposium*. Rochester, MN: Mayo Foundation, 1–34.
- Lilly DJ. (1972b) Acoustic impedance at the tympanic membrane: A overview of clinical applications. In: Rose D, Keating L, eds. *Proceedings of the Mayo Impedance Symposium*. Rochester, MN: Mayo Foundation, 51–74.
- Lilly DJ. (1984) Multiple frequency, multiple component tympanometry: New approaches to an old diagnostic problem. *Ear Hear.* 5, 300–308.
- Lilly DJ, Shanks JE. (1981) Acoustic immittance of an enclosed volume of air. In: Popelka GR, ed. *Hearing Assessment with the Acoustic Reflex*. New York: Grune & Stratton, 145–160.
- Lindeman P, Holmquist J. (1982) Volume measurement of middle ear and mastoid air cell system with impedance audiometry on patients with eardrum perforations. *Am J Otol.* 4, 46–51.
- Lous J. (1982) Three impedance screening programs on a cohort of seven-year-old children. *Scand Audiol Suppl.* 17, 60–64.
- Lyons A, Kei J, Driscoll C. (2004) Distortion product otoacoustic emissions in children at school entry: a comparison with pure-tone screening and tympanometry results. *J Am Acad Audiol.* 15, 702–715.
- Manolidis S, Shohet JA, Jackson CG, Glasscock ME. (1999) Malignant glomus tumors. *Laryngoscope.* 109, 30–34.
- Marchant CD, McMillan PM, Shurin PA, Johnson CE, Turczky VA, Feinstein JC, Panek DM. (1986) Objective diagnosis of otitis media in early infancy by tympanometry and ipsilateral acoustic reflex thresholds. *J Pediat.* 109, 590–595.
- Margolis RH. (1981) Fundamentals of acoustic immittance. In: Popelka GR, ed. *Hearing Assessment with the Acoustic Reflex*. New York: Grune & Stratton, 117–144.
- Margolis RH, Bass-Ringdahl S, Hanks WD, Holte L, Zapala DA. (2003) Tympanometry in newborn infants—1 kHz norms. *J Am Acad Audiol.* 14, 383–392.
- Margolis RH, Goycoolea HG. (1993) Multifrequency tympanometry in normal ears. *Ear Hear.* 14, 408–413.
- Margolis RH, Heller JW. (1987) Screening tympanometry: Criteria for medical referral. *Audiology.* 26, 197–208.
- Margolis RH, Hunter LL. (1999) Tympanometry: Basic principles and clinical implications. In: Musiek FE, Rintelmann WF, eds. *Contemporary Perspectives in Hearing Assessment*. Boston: Allyn & Bacon, 89–130.
- Margolis RH, Popelka GR. (1975) Static and dynamic acoustic impedance measurements in infant ears. *J Speech Hear Res.* 18, 435–443.
- Margolis RH, Popelka GR. (1977) Interactions among tympanometric variables. *J Sp Hear Res.* 20, 447–462.
- Margolis RH, Shanks JE. (1985) Tympanometry. In: Katz J ed. *Handbook of Clinical Audiology*, 3rd ed. Baltimore: Williams & Wilkins, 438–475.
- Margolis RH, Smith P. (1977) Tympanometric asymmetry. *J Speech Hear Res.* 20, 437–446.
- Margolis RH, Van Camp KJ, Wilson RH, Creten WL. (1985) Multifrequency tympanometry in normal ears. *Audiology.* 24, 44–53.
- McKinley AM, Grose, JH, Roush J. (1997) Multifrequency tympanometry and evoked otoacoustic emissions in neonates during the first 24 hours of life. *J Am Acad Audiol.* 8, 218–223.
- Meriot P, Veillon F, Garcia JF, Nonent M, Jezequel J, Bourjat P, Bellet M. (1997) CT appearances of ossicular injuries. *Radiographics.* 17, 1445–54.
- Meyer SE, Jardine CA, Deverson W. (1997) Developmental changes in tympanometry: A case study. *Br J Audiol.* 31, 189–195.
- Moller AR. (1960) Improved technique for detailed measurements of the middle ear impedance. *J Acoust Soc Am.* 32, 250–257.
- Moller AR. (1965) An experimental study of the acoustic impedance of the middle ear and its transmission properties. *Acta Otolaryngol.* 60, 129–149.
- Moller AR. (1972) The middle ear. In: Tobias J, ed. *Foundations of Modern Auditory Theory*. New York: Academic Press, 135–194.
- Molvaer O, Vallersnes F, Kringlebotn M. (1978) The size of the middle ear and the mastoid air cell. *Acta Otolaryngol.* 85, 24–32.
- Muchnik C, Hildesheimer M, Rubinstein M, Gleitman Y. (1989) Validity of tympanometry in case of confirmed otosclerosis. *J Laryngol Otol.* 103, 36–38.
- Nozza RJ, Bluestone CD, Kardatz D, Bachman R. (1992) Towards the validation of aural acoustic immittance measures for diagnosis of middle ear effusion in children. *Ear Hear.* 13, 442–453.
- Nozza RJ, Bluestone CD, Kardatz D, Bachman R. (1994) Identification of middle ear effusion by aural acoustic immittance measures for diagnosis of middle ear effusion in children. *Ear Hear.* 15, 310–323.
- O’Donoghue G, Jackler R, Jenkins W, Schindler R. (1986) Cochlear implantation in children: The problem of head growth. *Otolaryngol Head Neck Surg.* 94, 78–81.
- Osguthorpe JD, Lam C. (1981) Methodologic aspects of tympanometry in cats. *Otolaryngol Head Neck Surg.* 89, 1037–1040.
- Ostergard CA, Carter DR. (1981) Positive middle ear pressure shown by tympanometry. *Arch Otolaryngol.* 107, 353–356.
- O’Tuama LA, Swanson MS. (1986) Development of paranasal and mastoid sinuses: A computed tomographic pilot study. *J Child Neurol.* 1, 46–49.

- Paradise JL, Smith CG, Bluestone CD. (1976) Tympanometric detection of middle ear effusion in infants and young children. *Pediatrics*. 58, 198–210.
- Poe DS, Abou-Halawa A, Abdel-Razek O. (2001) Analysis of the dysfunctional eustachian tube by video endoscopy. *Otol Neurotol*. 22, 590–595.
- Porter TA, Winston ME. (1973) Methodological aspects of admittance measurements of the middle ear. The Reflex. Concord, MA: Grason-Stadler.
- Purdy SC, Williams MJ. (2000) High frequency tympanometry: A valid and reliable immittance test protocol for young infants? *New Zealand Audiol Soc Bull*. 10, 9–24.
- Rabinowitz WM. (1981) Measurement of the acoustic input immittance of the human ear. *J Acoust Soc Am*. 70, 1025–1035.
- Renvall U, Holmquist J. (1976) Tympanometry revealing middle ear pathology. *Ann Otol Rhinol Laryngol*. 85, 209–215.
- Renvall U, Liden G, Bjorkman G. (1975) Experimental tympanometry in human temporal bones. *Scand Audiol*. 4, 135–144.
- Rhodes MC, Margolis RH, Hirsch JE, Napp AP. (1999) Hearing screening in the newborn intensive care nursery: A comparison of methods. *Otolaryngol Head Neck Surg*. 120, 799–808.
- Roup CM, Wiley TW, Safady SH, Stoppenback DT. (1998) Tympanometric screening norms for adults. *Am J Audiol*. 7, 55–60.
- Roush J, Bryant K, Mundy M, Zeisel S, Roberts J. (1995) Developmental changes in static admittance and tympanometric width in infants and toddlers. *J Am Acad Audiol*. 6, 334–338.
- Roush J, Drake A, Sexton JE. (1992) Identification of middle ear dysfunction in young children: A comparison of tympanometric screening procedures. *Ear Hear*. 13, 63–69.
- Roush J, Tait CA. (1985) Pure tone and acoustic immittance screening of preschool-aged children: An examination of referral criteria. *Ear Hear*. 6, 245–249.
- Rubensohn G. (1965) Mastoid pneumatization in children at various ages. *Acta Otolaryngol*. 60, 11–14.
- Sade J, Amos AR. (1997) Middle ear and auditory tube: Middle ear clearance, gas exchange, and pressure regulation. *Otolaryngol Head Neck Surg*. 116, 499–524.
- Sade J, Fuchs C. (1996) Secretory otitis media in adults: I. The role of mastoid pneumatization as a risk-factor. *Ann Otol Rhinol Laryngol*. 105, 643–647.
- Saeed K, Coglianese CL, McCormick DP, Chonmaitree T. (2004) Otopscopic and tympanometric findings in acute otitis media yielding dry tap at tympanocentesis. *Ped Infect Dis J*. 23, 1030–1034.
- Sederberg-Olsen J, Sederberg-Olsen A, Jensen A. (1983) The prognostic significance of the air volume in the middle ear for the tendency to recurrence of secretory middle ear condition. *Int J Ped Otorhinolaryngol*. 5, 179–187.
- Shahnaz N, Davies D. (2006) Standard and multifrequency tympanometric norms for Caucasian and Chinese young adults. *Ear Hear*. 27, 75–90.
- Shahnaz N, Polka L. (1997) Standard and multifrequency tympanometry in normal and otosclerotic ears. *Ear Hear*. 18, 326–341.
- Shahnaz N, Polka L. (2002) Distinguishing healthy from otosclerotic ears: effect of probe-tone frequency on static immittance. *J Am Acad Audiol*. 13, 345–355.
- Shanks JE (1984). Tympanometry. *Ear Hear*. 5, 268–280.
- Shanks JE. (1985) Tympanometric volume estimates in patients with intact and perforated eardrums. Presented at the annual meeting of the American Speech-Language Hearing Association, Washington D.C.
- Shanks JE. (1990) Multiple frequency tympanometry: Findings in otosclerosis. Presented at the annual meeting of the American Academy of Audiology, New Orleans.
- Shanks JE, Lilly DJ. (1981) An evaluation of tympanometric estimates of ear canal volume. *J Speech Hear Res*. 24, 557–566.
- Shanks JE, Lilly DJ, Margolis RH, Wiley TL, Wilson RH. (1988) Tutorial: Tympanometry. *J Speech Hear Dis*. 53, 354–377.
- Shanks JE, Shelton C. (1991) Basic principles and clinical applications of tympanometry. *Otolaryngol Clin North Am*. 24: 299–328.
- Shanks JE, Stelmachowicz PG, Beauchaine KL, Schulte L. (1992) Equivalent ear canal volumes in children pre- and post-tympanostomy tube insertion. *J Speech Hear Res*. 35, 936–941.
- Shanks JE, Wilson RH. (1986) Effects of direction and rate of ear-canal pressure changes on tympanometric measures. *J Speech Hear Res*. 29, 11–19.
- Shanks JE, Wilson RH, Cambron NK. (1993) Multiple frequency tympanometry: Effects of ear canal volume compensation on static acoustic admittance and estimates of middle ear resonance. *J Speech Hear Res*. 36, 178–185.
- Shaw EAG. (1974) Transformation of sound pressure level from the free field to the eardrum in the horizontal plane. *J Acoust Soc Amer*. 56, 1848–1861.
- Shohet JA, de Jong AL. (2002) The management of pediatric cholesteatoma. *Otolaryngol Clin North Am*. 35, 841–51.
- Shohet JA, Sutton F. (2001) “Middle Ear, Otosclerosis”. *Emedicine Otolaryngology and Facial Plastic Surgery* Online Textbook.
- Shurin PA, Pelton SI, Klein JO. (1976) Otitis media in the newborn infant. *Ann Otol Rhinol Laryngol*. 85, 216–222.
- Silman S, Silverman CA, Arick DS. (1992) Acoustic-immittance screening for detection of middle-ear effusion in children. *J Am Acad Audiol*. 3, 262–268.
- Silverman CA, Silman S. (1995) Acoustic-immittance characteristics of children with middle-ear effusion: Longitudinal investigation. *J Am Acad Audiol*. 6, 339–345.
- Sininger YS. (2003) Audiologic assessment of infants. *Curr Opin Otolaryngol Head Neck Surg*. 11, 378–382.
- Sprague BH, Wiley TL, Goldstein R. (1985) Tympanometric and acoustic-reflex studies in neonates. *J Speech Hear Res*. 28, 265–272.
- Sullivan RE. (2006) Audiology Forum: Video Otoscopy. Online at www.rcsullivan.com/www/ears.htm.
- Sutton G, Baldwin M, Brooks D, Gravel J, Thornton R. (2002) Tympanometry in neonates and infants under 4 months: A recommended test protocol. The Newborn Hearing Screening Programme, UK; online at www.nhsp.info.
- Sutton GJ, Gleadle P, Rowe SJ. (1996) Tympanometry and otoacoustic emissions in a cohort of special care neonates. *Br J Audiol*. 30, 9–17.
- Tashima K, Tanaka S, Saito H (1986) Volumetric changes of the aerated middle ear and mastoid after insertion of tympanostomy tubes. *Am J Otolaryngol*. 7, 302–305.
- Terkildsen K. (1976) Pathologies and their effect on middle ear function. In: Feldman AS & Wilbur LA, eds. *Acoustic Impedance and Admittance: The Measurement of Middle Ear Function*. Baltimore: Williams & Wilkins, 78–102.
- Terkildsen K, Scott Nielsen S. (1960) An electroacoustic impedance measuring bridge for clinical use. *Arch Otolaryngol*. 72, 339–346.
- Terkildsen K, Thompsen KA. (1959) The influence of pressure variations on the impedance of the human ear drum. *J Laryngol Otol*. 73, 409–418.
- Tos M, Poulsen G. (1980) Screening tympanometry in infants and two-year-old children. *Ann Otol Rhinol Laryngol Suppl*. 68, 217–222.
- Uzun C, Adali MK, Korten, M, Yagiz R, Aydin S, Kahir B, Karasalioglu A. (2002) Relationship between mastoid pneumatization and middle ear barotraumas in divers. *Laryngoscope*. 112, 287–291.
- Valvik B, Johnson M, Laukli E. (1994) Multifrequency tympanometry. *Audiology*. 33, 245–253.
- Van Camp KJ, Creten WL, Vanpeperstraete PM, Van de Heyning PH. (1980) Tympanometry-detection of middle ear pathologies. *Acta Otol Rhinol Laryngol*. 34, 574–583.
- Van Camp KJ, Margolis RH, Wilson RH, Creten W, Shanks JE. (1986) Principles of tympanometry, (Monograph No. 24) Rockville MD: American Speech-Language-Hearing Association.

- Van Camp KJ, Vanhuyse VJ, Creten WL, Vanpeperstraete PM. (1978) Impedance and admittance tympanometry. II. Mathematical approach. *Audiology*. 17, 108–119.
- Van Camp KJ, Vogeeler M. (1986) Normative multifrequency tympanometric data on otosclerosis. *Scand Audiol*. 15, 187–190.
- Van de Heyning P H, Van Camp K J, Creten WL, Vanpeperstraete PM. (1982) Incudo-stapedial joint pathology: A tympanometric approach. *J Speech Hear Res*. 25, 611–618.
- Vanhuyse VJ, Creten WL, Van Camp KJ. (1975) On the W-notching of tympanograms. *Scand Audiol*. 4, 45–50.
- Vanpeperstraete PM, Creten WL, Van Camp KJ. (1979) On the asymmetry of susceptance tympanograms. *Scand Audiol*. 8, 173–179.
- Virtanen H, Palva T. (1982) The patulous Eustachian tube and chronic middle ear disease. *Acta Otolaryngol*. 93, 49–53.
- Voss SE, Rosowski JJ, Merchant SN, Peake WT. (2001) Middle-ear function with tympanic-membrane perforations. I. Measurements and mechanisms. *J Acoust Soc Am*. 110, 1432–1444.
- Wada H, Koike T, Kobayashi T. (1998) Clinical applicability of the sweep frequency measuring apparatus for diagnosis of middle ear diseases. *Ear Hear*. 19, 240–249.
- Wan IK, Wong LL. (2002) Tympanometric norms for Chinese young adults. *Ear Hear*. 23, 416–421.
- Watters GWR, Jones JE, Freeland AP. (1997) The predictive value of tympanometry in the diagnosis of middle ear effusion. *Clin Otolaryngol*. 22, 343–345.
- Wilber LA, Feldman AS. (1976) The middle ear measurement battery. In: Feldman AS and Wilbur LA, eds. *Acoustic Impedance and Admittance: The Measurement of Middle Ear Function*. Baltimore: Williams & Wilkins, 345–377.
- Wiley TL, Cruickshanks KJ, Nondahl DM, Tweed TS. (1999) Aging and middle ear resonance. *J Am Acad Audiol*. 10, 173–179.
- Wiley TL, Cruickshanks KJ, Nondahl DM, Tweed TS, Klein R, Klein BEK. (1996) Tympanometric measures in older adults. *J Am Acad Audiol*. 7, 260–268.
- Wiley TL, Fowler CG. (1997) *Acoustic Immittance Measures in Clinical Audiology: A Primer*. San Diego: Singular Publishing Group.
- Wilson RH, Shanks JE, Kaplan SK. (1984) Tympanometric changes at 226 Hz and 678 Hz across 10 trials and for two directions of ear canal pressure change. *J Speech Hear Res*. 27, 257–266.
- Wilson RH, Shanks JE, Velde TM. (1981) Aural acoustic-impedance measurements: Inter-aural differences. *J Speech Hear Dis*. 46, 413–421.
- Zhao F, Wada H, Koike T, Ohyama K, Kawase T, Stephens D. (2002) Middle ear dynamic characteristics in patients with otosclerosis. *Ear Hear*. 23, 150–158.
- Zwislocki J. (1962) Analysis of the middle-ear function. Part I: Input impedance. *J Acoust Soc Am*. 34, 1514–1523.
- Zwislocki J. (1976) The acoustic middle ear function. In: Feldman AS and Wilbur LA, eds. *Acoustic Impedance and Admittance: The Measurement of Middle Ear Function*. Baltimore: Williams & Wilkins, 66–77.
- Zwislocki J, Feldman AS. (1970) *Acoustic impedance of pathological ears*. Monograph No. 15. Rockville, MD: American Speech-Language-Hearing Association.