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Middle ear mechanics in normal, diseased and reconstructed ears

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Abstract

A review of the structure-function relationships in normal, diseased and reconstructed middle ears is presented. Variables used to describe the system are sound pressure, volume velocity and acoustic impedance. We discuss the following:

(1) Sound can be transmitted from the ear canal to the cochlea via two mechanisms: the tympano-ossicular system (ossicular coupling) and direct acoustic stimulation of the oval and round windows (acoustic coupling). In the normal ear, middle-ear pressure gain, which is the result of ossicular coupling, is frequency-dependent and smaller than generally believed. Acoustic coupling is negligibly small in normal ears, but can play a significant role in some diseased and reconstructed ears.

(2) The severity of conductive hearing loss due to middle-ear disease or after tympanoplasty surgery can be predicted by the degree to which ossicular coupling, acoustic coupling, and stapes-cochlear input impedance are compromised. Such analyses are used to explain the air-bone gaps associated with lesions such as ossicular interruption, ossicular fixation and tympanic membrane perforation.

(3) With type IV and V tympanoplasty, hearing is determined solely by acoustic coupling. A quantitative analysis of structure-function relationships can both explain the wide range of observed post-operative hearing results and suggest surgical guidelines in order to optimize the post-operative results.

(4) In tympanoplasty types I, II and III, the hearing result depends on the efficacy of the reconstructed tympanic membrane, the efficacy of the reconstructed ossicular chain and adequacy of middle-ear aeration. Currently, our knowledge of the mechanics of these three factors is incomplete. The mechanics of mastoidectomy and stapedectomy are also discussed.

Key words: Ear, middle; Hearing loss, conductive; Tympanoplasty

Introduction

Joseph Toynbee was a remarkable person: an accomplished clinician and otopathologist, a dedicated and insatiable scientist, and a philanthropist. His classic book *Diseases of the Ear* laid the foundations for scientific otology in the last century (Toynbee, 1860). He devised an artificial tympanic membrane in 1853, and thus, he can also be regarded as one of the pioneers of middle-ear reconstructive surgery. It is a great privilege for me (SNM) to present the Sixteenth Biennial Toynbee Memorial Lecture, and I am grateful to the selection committee, as well as to the Royal Society of Medicine and to the Midland Institute of Otology, for this honour.

The work reported here is the result of a fruitful collaboration between clinicians and basic scientists. Our group seeks understanding of the mechanical processes of the normal middle ear and an application of concepts to diseased and reconstructed ears. An important impetus for this research is the unsatisfactory hearing results that are often obtained after tympanoplasty, especially with advanced lesions of the ossicles. The summary in Table I of post-surgical hearing results from eight large clinical series spanning the past 30 years demonstrates that results are often less than satisfactory. When the ossicular chain has to be reconstructed, long-term closure of the air-bone gap to ≤20 dB occurs in 40–70 per cent of cases when the stapes is intact, and

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TABLE I
POST-TYMPANOPLASTY HEARING RESULTS. % OF CASES WITH POST-OPERATIVE AIR-BONE GAPS ≤ 20 dB

Authors	No. of cases	Minor columellas, including PORPs	Major columellas, including TORPs
1. Lee and Schuknecht (1971)	936	40%	—
2. Pennington (1973)	216	70%	—
3. Jackson *et al.* (1983)	417	64%	43%
4. Brackmann *et al.* (1984)	1042	73%	55%
5. Lau and Tos (1986)	229	54%	40%
6. Ragheb *et al.* (1987)	455	52%	37%
7. Colletti *et al.* (1987)	832	45%	30%
8. Goldenberg (1992)	262	57%	58%

in only 30–55 per cent of cases when the stapes superstructure is missing (Lee and Schuknecht, 1971; Pennington, 1973; Jackson *et al.*, 1983; Brackmann *et al.*, 1984; Lau and Tos, 1986; Ragheb *et al.*, 1987; Colleti *et al.*, 1987; Goldenberg, 1992).

It seems likely that one major factor responsible for the modest nature of post-tympanoplasty hearing results is lack of quantitative understanding of structure-function relationships in the mechanical response of reconstructed ears. The need for improved understanding of middle-ear mechanics is clearly shown by the clinical occurrence of many instances in which anatomical differences between a good and poor hearing result are seemingly minor. For example, Liston *et al.* (1991), with the use of intra-operative monitoring by auditory evoked responses during ossiculoplasty, found that changes in prosthesis position by 0.5–1.0 mm had effects on hearing as large as 20 dB. It is also a common clinical observation that post-surgical ears, which seem identical in structure, can exhibit markedly differing degrees of conductive hearing loss. In other words, small changes in structure can have large effects on function. Other major factors contributing to unsatisfactory post-surgical hearing results are incomplete knowledge of the biology of chronic middle-ear disease (including pathology of middle-ear aeration and Eustachian tube function), and a lack of control over the histopathological and tissue responses of the middle ear to surgery; these factors are outside the scope of this paper.

In our laboratory, we use three approaches to understand middle-ear mechanics: quantitative physics-based models, acoustic measurements in cadaveric temporal bone preparations and clinical assessment of hearing results (Figure 1). *A model of the middle ear* is essentially a set of mathematical equations that relate the physical structure of the ear to its acoustic function. *The cadaveric temporal bone preparation* is used to test predictions of a given model through acoustical measurements of middle-ear sound transmission before and after lesions and before and after simulated surgical procedures. There is good evidence that the mechanical properties of the cadaver human middle ear are representative of the *in vivo* condition (Rosowski *et al.*, 1990; Goode *et al.*, 1996; Puria *et al.*, 1997). The use of temporal bones allows one to make repeated measurements of acoustic and mechanical function, to make well controlled structural alterations and to measure response variables that are not accessible in living subjects such as stapes motion and intra-cochlear pressure. *Analysis of clinical hearing results* is used to determine the relevance both of our modelling and of our temporal bone experiments to clinical experience.

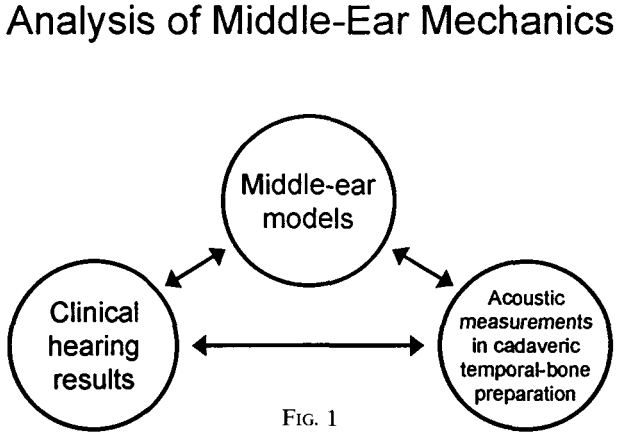

FIG. 1
Interactive approaches used to analyse middle-ear mechanics.

There is constant interplay between these three approaches. For instance, we first develop a model for a particular tympanoplasty reconstruction and make predictions of the air-bone gap under different conditions. These predictions are tested in the temporal bone preparation and with post-surgical audiometric data. The test results are then used to amend the model so that it relates structure to function more precisely. This sequence allows us to improve understanding of the mechanical processes involved in the reconstruction in question, and in turn, to modify surgical techniques so as to improve the post-operative hearing results. This review of the ideas involved in our models and measurements is designed to communicate clearly with clinicians. Previously published work contains more detailed information (Rosowski *et al.*, 1990; Peake *et al.*, 1992; Rosowski and Merchant, 1995; Merchant *et al.*, 1995; Rosowski *et al.*, 1995; Merchant *et al.*, 1996; Voss *et al.*, 1996; Merchant *et al.*, 1997a; Merchant *et al.*, 1997b; Puria *et al.*, 1997; Voss *et al.*, 1997; Voss, 1998; Whittemore *et al.*, 1998).

The normal middle ear

Sound can be conducted into the cochlea by two distinct mechanisms (Figures 2A and B), termed 'ossicular coupling' and 'acoustic coupling' (Peake *et al.*, 1992). Ossicular coupling occurs by way of the tympanic membrane and ossicles and results in an increase of sound pressure applied to the stapes ($\mathbf{P_S}$) compared to the sound pressure in the ear canal ($\mathbf{P_{EC}}$). At the same time, motion of the tympanic

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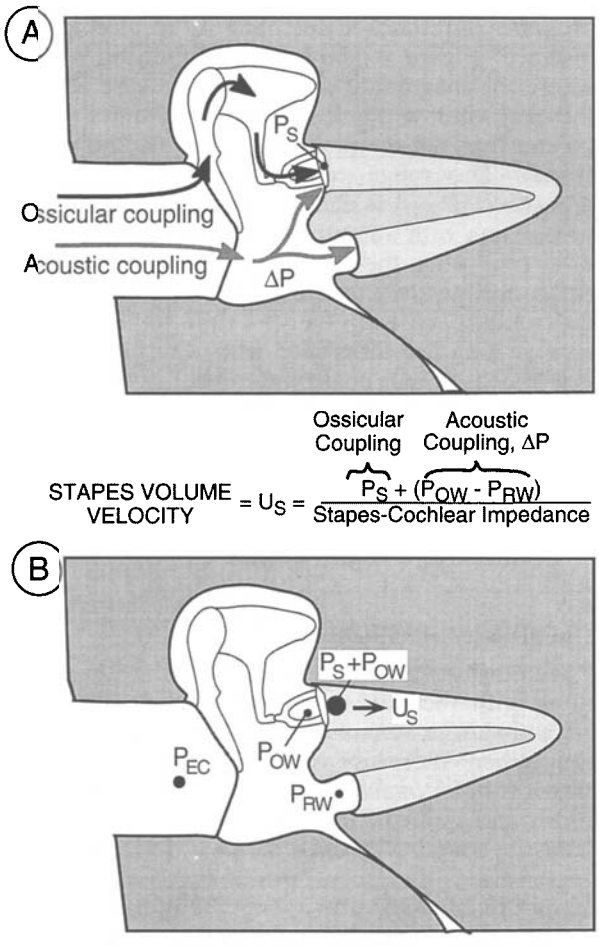

FIG. 2

Representation of two mechanisms of middle-ear sound transmission in the normal ear.

A. Schematic showing Ossicular coupling, $\mathbf{P_S}$ and Acoustic coupling, $\mathbf{\Delta P}$.

B. Schematic showing important acoustic variables in middle-ear sound transmission: $\mathbf{P_{EC}}$ = the stimulus sound pressure in the ear canal; $\mathbf{P_{OW}}$ = the sound pressure at the oval window; $\mathbf{P_{RW}}$ = the sound pressure at the round window; $\mathbf{P_S}$ = the ossicularly derived sound pressure of the stapes; $\mathbf{P_S + P_{OW}}$ = the total sound pressure at the oval window; $\mathbf{U_S}$ = the stapes volume velocity (the output of the middle ear).

membrane also creates a sound pressure in the middle-ear cavity. Because the cochlear windows are separated spatially, the sound pressures (within the middle-ear cavity) that act at the oval and round windows, $\mathbf{P_{OW}}$ and $\mathbf{P_{RW}}$ respectively, are not identical. The sound pressure difference between the windows is termed 'acoustic coupling' $\mathbf{\Delta P}$, where $\mathbf{\Delta P = P_{OW} - P_{RW}}$. Ossicular coupling and acoustic coupling add to produce a net sound pressure difference between the oval and round windows, to which the cochlea responds (Voss *et al.*, 1996).

Both ossicular and acoustic coupling result in motion of the stapes which can be quantified as the volume velocity of the stapes $\mathbf{U_S}$ (Figures 2A and B) (Peake *et al.*, 1992; Rosowski and Merchant, 1995). The stapes *volume* velocity is equal to its piston-like *linear* velocity multiplied by the area of the stapes footplate. The velocity $\mathbf{U_S}$ is 'opposed' by the impedances of the annular ligament and of the cochlea, together termed $\mathbf{Z_{SC}}$, the stapes-cochlear input impedance. Mathematically,

$$\mathbf{U_S} = \frac{\mathbf{P_S} + \mathbf{\Delta P}}{\mathbf{Z_{SC}}}. \qquad \text{(Equation 1)}$$

This equation describes middle-ear sound transmission in terms of the three quantities: $\mathbf{P_S}$, the pressure applied to the oval window by the stapes that results from ossicular coupling, $\mathbf{\Delta P}$, the acoustically-coupled stimulus, and $\mathbf{Z_{SC}}$, the input impedance of the stapes and cochlea. A loss in middle-ear sound transmission will occur if a lesion compromises any one of these three quantities, and the resulting air-bone gap can be predicted by comparing the altered $\mathbf{U_S}$ to the normal $\mathbf{U_S}$ and converting to decibels (Rosowski and Merchant, 1995). Thus, Equation 1 can be used to predict the conductive hearing loss occurring in a diseased or reconstructed ear.

Measured magnitudes of ossicular and acoustic coupling relative to sound pressure in the ear canal are shown in Figure 3. The solid line labelled 'Ossicular coupling, $\mathbf{P_S}$' is the mean of measurements of ossicular coupling made in six normal human temporal bones by Kurokawa and Goode (1995), and is similar to results obtained in our laboratory (Puria *et al.*, 1997). The magnitude of ossicular coupling, which is commonly called the middle-ear 'gain', is about 20 dB between 250 and 500 Hz, is a

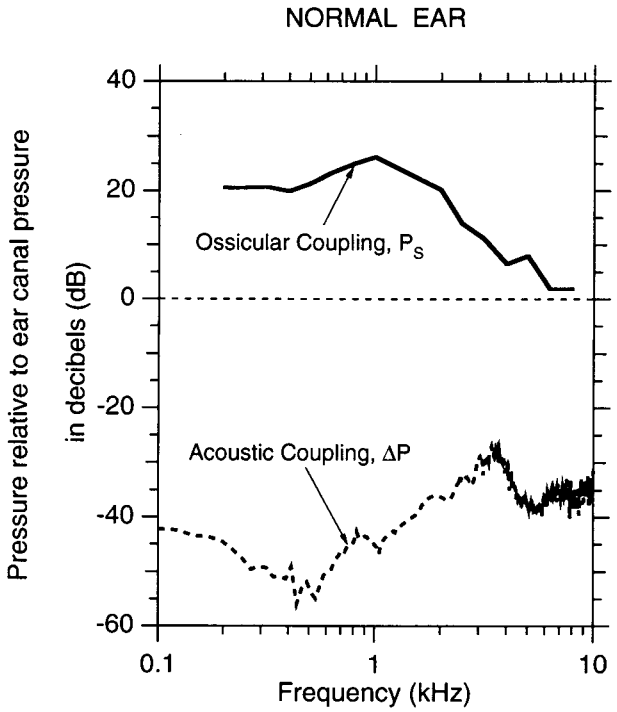

FIG. 3

Relative magnitudes of ossicular and acoustic coupling in a normal ear. The abscissa is a logarithmic frequency scale and the ordinate is a decibel scale on which both ossicular coupling ($\mathbf{P_S}$) and acoustic coupling ($\mathbf{\Delta P}$) pressures are plotted relative to the ear canal sound pressure. The horizontal dashed line at 0 dB represents a sound pressure level at the cochlear input equal to the sound pressure in the ear canal.

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maximum of about 25 dB around 1 kHz, and then decreases by about 6–8 dB per octave at frequencies above 1 kHz. Thus, the conventional transformer model of the middle ear which predicts a frequency independent middle-ear gain of 27–34 dB (Shambaugh, 1967; Schuknecht, 1974; Goodhill, 1979; Austin, 1990) is not quite accurate. The dashed line labelled 'Acoustic coupling, ΔP' is the mean of measurements in eight normal temporal bones (Voss, 1998). The measurements show that acoustic coupling is relatively small in the normal ear, on the order of 60 dB below ossicular coupling. Hence, one can ignore acoustic coupling in the normal ear. However, we will show later that acoustic coupling plays an important role in some diseased and reconstructed ears.

The cochlea responds to the *difference* in sound pressure between the cochlear windows (Peake *et al.*, 1992; Voss *et al.*, 1996). It is important to understand how this difference depends on the relative *magnitude* and *phase* of these two sound pressures. The window pressure difference $\mathbf{P_{WD}}$ is the sum of the ossicularly-coupled stapes sound pressure $\mathbf{P_S}$ and acoustically-coupled sound pressure ($\mathbf{P_{OW}} - \mathbf{P_{RW}}$) so that $\mathbf{P_{WD}} = \mathbf{P_S} + \mathbf{P_{OW}} - \mathbf{P_{RW}}$. (The total pressure acting at the oval window is $\mathbf{P_S} + \mathbf{P_{OW}}$, and the total pressure acting at the round window is $\mathbf{P_{RW}}$.) When there is a significant difference in magnitude of the ossicularly and acoustically coupled sound as in the normal ear or after successful tympanoplasty (types I, II and III), where $\mathbf{P_S} >> (\mathbf{P_{OW}} - \mathbf{P_{RW}})$, differences in phase have little effect in determining the window pressure difference (Merchant *et al.*, 1997b). For instance, Figure 4 shows a hypothetical situation where the magnitude of the total pressure acting at the oval window (i.e. $\mathbf{P_S} + \mathbf{P_{OW}}$) is 10 times (20 dB) greater than the sound pressure at the round window ($\mathbf{P_{RW}}$). The range of possible window pressure difference ($\mathbf{P_{WD}}$) is shown in Figure 4 by two sine waves, one with an amplitude of 9 representing the difference when the total oval window pressure and the round window pressure are in-phase (0° phase difference), and the other with an amplitude of 11 representing the difference when the pressures are out-of-phase (180° phase difference). Even with this maximum effect of varying the phase difference, the two curves shown in Figure 4 are similar in magnitude (within 2 dB). With larger magnitude differences, e.g. the factor of 100–1000 (i.e. 40–60 dB) expected in the normal ear, phase variations have a negligible effect.

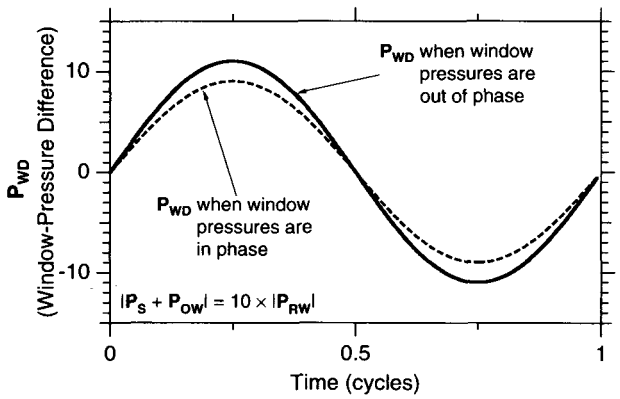

FIG. 4

Schematic showing that if there is an appreciable difference in magnitude between the window pressures, the difference in phase is of little importance in determining the difference between the sound pressures. In this specific case, one cycle of the waveform of the window-pressure difference $\mathbf{P_{WD}} = \mathbf{P_S} + \mathbf{P_{OW}} - \mathbf{P_{RW}}$ is plotted for two circumstances. In each circumstance, the total oval-window pressure $\mathbf{P_S} + \mathbf{P_{OW}}$ is a sine wave with an amplitude of 10, while the sound pressure at the round window $\mathbf{P_{RW}}$ is a sine wave with an amplitude of 1. The dashed line shows the window pressure difference $\mathbf{P_{WD}}$ when the total oval window pressure and round window pressure are in-phase, and the result is a $\mathbf{P_{WD}}$ wave of amplitude 9 = 10–1. The solid line shows $\mathbf{P_{WD}}$ when the pressures at the two windows are out-of-phase, and the result is a $\mathbf{P_{WD}}$ wave of amplitude 11 = 10–(–1). Note that the two $\mathbf{P_{WD}}$ pressures differ by less than 2 dB [i.e., $20\log_{10}(11/9) = 1.7$ dB], even though this phase variation produces the largest possible magnitude difference.

The diseased middle ear

We now use the model of Figure 2 and Equation 1, along with the data of Figure 3 to predict sound transmission in some abnormal states of the middle ear. When there is *ossicular interruption in the presence of an intact drum,* ossicular coupling is lost and sound input to the cochlea results solely from acoustic coupling (Peake *et al.*, 1992). Figure 3 demonstrates that acoustic coupling is about 60 dB smaller than ossicular coupling; therefore, we would predict that ossicular interruption results in a conductive hearing loss of 60 dB. This prediction is consistent with clinical observations (Figure 5A). Note that the consistency of the clinical results with our model of acoustic coupling suggests that stimuli reaching the inner ear through bone-conduction mechanisms in this particular condition are small enough to be ignored. This simplifying conclusion is helpful as the transmission of air-borne sound to the inner ear through bone conduction is poorly understood.

Loss of the tympanic membrane, malleus and incus abolishes ossicular coupling and leads to an enhancement of acoustic coupling of about 10–20 dB as compared to the normal ear (Peake *et al.*, 1992). This enhancement results from the loss of the shielding effect of the tympanic membrane which in the normal ear attentuates the middle-ear sound pressure by 10–20 dB relative to the ear-canal sound pressure. Therefore, for the missing tympanic membrane, malleus and incus condition, the air-bone gap predicted by Equation 1 is 40–50 dB. The predicted gap is similar to that measured in patients (Figure 5B). The increase in acoustic coupling due to loss of tympanic-membrane shielding also explains why the hearing of a patient with an interrupted ossicular chain and an intact tympanic membrane is improved by 10–20 dB with the creation of a perforation in the tympanic membrane.

Fixation of the stapes footplate due to otosclerosis results in a conductive hearing loss that can range from 5 to 50 dB (Shambaugh, 1967; Schuknecht, 1974; Goodhill, 1979). Otosclerotic fixation of the

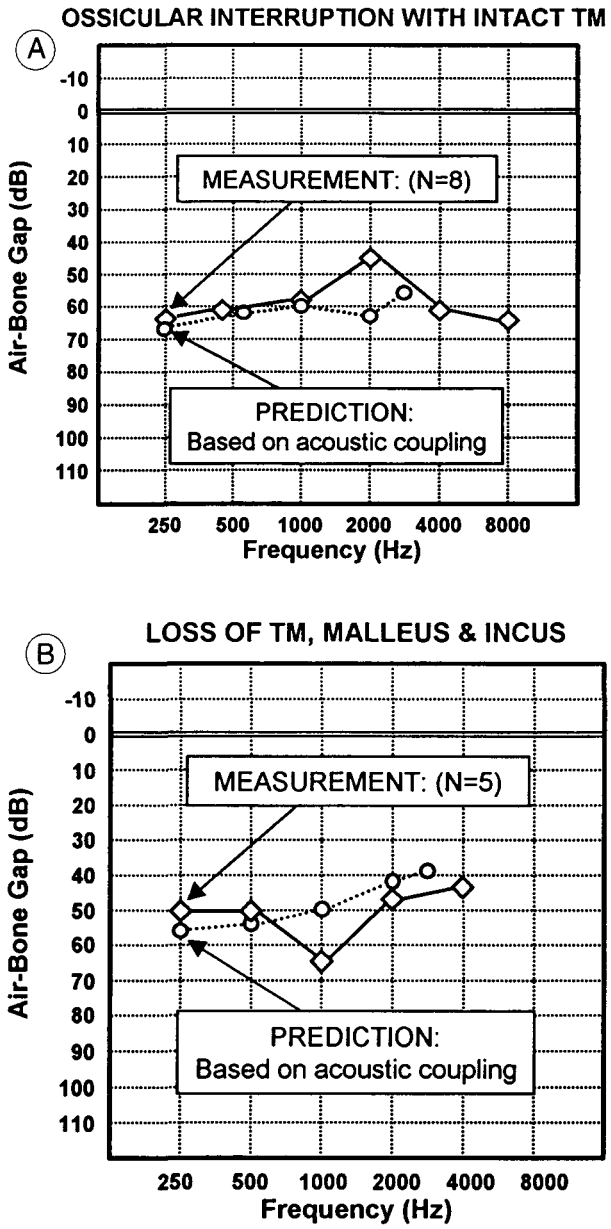

FIG. 5

Comparison of mean measured air-bone gaps with model predictions for two pathological conditions:
A. Ossicular interruption with intact tympanic membrane (TM): mean air-bone gaps measured in eight cases with surgically confirmed ossicular interruption are compared to air-bone gaps predicted by Equation 1. (Modified from Peake *et al.*, 1992.)
B. Loss of tympanic membrane (TM), malleus and incus: mean air-bone gaps measured in five cases are compared to air-bone gaps predicted by Equation 1. (Modified from Peake *et al.*, 1992.)

stapes footplates causes an increase in $\mathbf{Z_{SC}}$ (which is made up of the impedance of the annular ligament plus the impedance of the cochlea). The amount by which $\mathbf{Z_{SC}}$ is increased depends on the degree of fixation, and can account for the wide variation in the observed hearing loss. For low frequencies, $\mathbf{Z_{SC}}$ is determined mainly by the impedance of the annular ligament rather than the impedance of the cochlea (Merchant *et al.*, 1996). Therefore, any increase in ligament impedance will be seen first at low frequencies, which is consistent with the initial appearance of a low frequency hearing loss in otosclerosis. In contrast, the conductive hearing loss associated with so-called *malleus fixation* is only 15–25 dB (Schuknecht, 1993). Malleus fixation is generally the result of a bony spur that extends from the epitympanic wall to the malleus head (Schuknecht, 1993). Since the point of fixation lies near the axis of rotation of the malleus, and since this spur attaches to the malleus head only over a relatively small surface area, it is likely that true 'fixation' of the malleus does not occur and some sound is still transmitted across the incudo-malleal joint (Merchant *et al.*, 1997b). Hence, so-called malleus fixation leads to a reduction in ossicular coupling which is consistent with the 15–25 dB conductive losses that are observed clinically.

Perforations of the tympanic membrane cause a conductive hearing loss that can range from negligible to 50 dB (Anthony and Harrison, 1972; Austin, 1990). The primary mechanism of the air-bone gap due to a perforation is a reduction in ossicular coupling that is caused by a loss in pressure-difference across the tympanic membrane, and the magnitude of the gap is proportional to the degree of reduction in ossicular coupling (Voss *et al.*, 1997; Voss, 1998). The perforation also leads to an increase in acoustic coupling by 10–20 dB, because of loss of the shielding effect of the intact tympanic membrane (Voss *et al.*, 1997; Voss, 1998). The increase in acoustic coupling allows one to predict that the maximum conductive loss from a perforation will be no more than 40–50 dB, which is consistent with clinical observations.

The reconstructed middle ear

Our research to date has enabled us to develop a good understanding of the mechanics of type IV and V tympanoplasties, and we concentrate on these two examples in this review. In addition, we report a preliminary analysis of types I, II and III tympanoplasties for which our knowledge is far from complete at the present time. Finally, we discuss the mechanics of mastoidectomy and stapedectomy.

Type IV and V tympanoplasty

Amongst tympanoplasties, the simplest reconstruction is perhaps the Type IV procedure (Wullstein, 1956). It is a surgical option in patients with a canal wall down mastoidectomy where the tympanic membrane and ossicles are missing and the stapes footplate is mobile. In this situation, incoming sound from the ear canal acts directly on the footplate, while the round window is acoustically shielded by a tissue graft such as temporalis fascia (Figures 6A and B). The tissue graft that protects the round window is also called a 'shield' or 'graft-shield'. The air space between the shield and the round window is termed the 'cavum minor'. With no ossicular coupling, residual hearing depends solely on acoustic coupling (Peake *et al.*, 1992).

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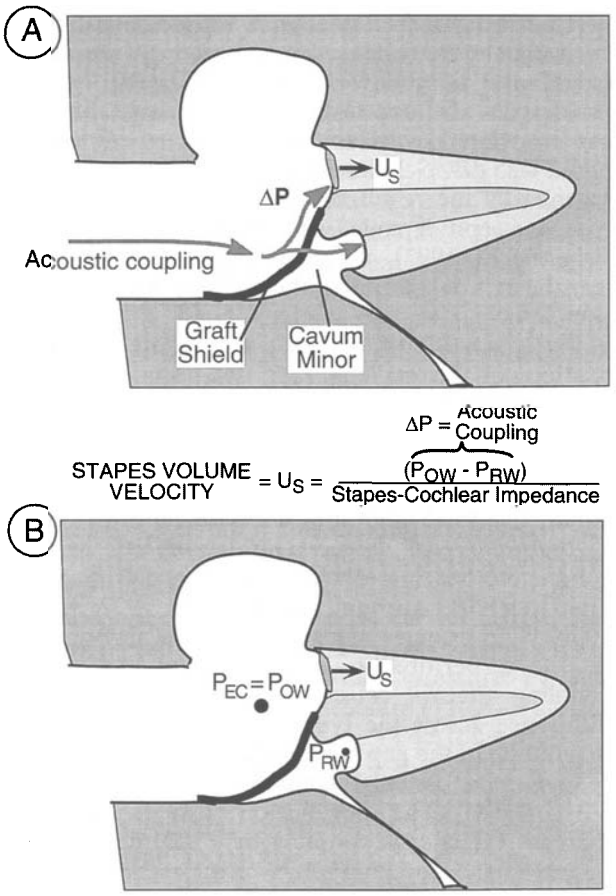

FIG. 6

Schematic drawing of type IV tympanoplasty.
A. Incoming sound from the ear canal is allowed to impinge directly on a mobile stapes footplate, while the round window is acoustically protected by a graft-shield. With no ossicular coupling, cochlear stimulation depends on acoustic coupling ΔP alone. U_S = volume velocity of the stapes.
B. The shield reduces the sound pressure at the round window, schematized by the smaller size of the P_{RW} dot, increasing the window pressure difference $\Delta P = P_{OW} - P_{RW}$ and enhancing acoustic coupling. P_{EC} = sound pressure in the ear canal, which is the same as the oval window sound pressure P_{OW}.

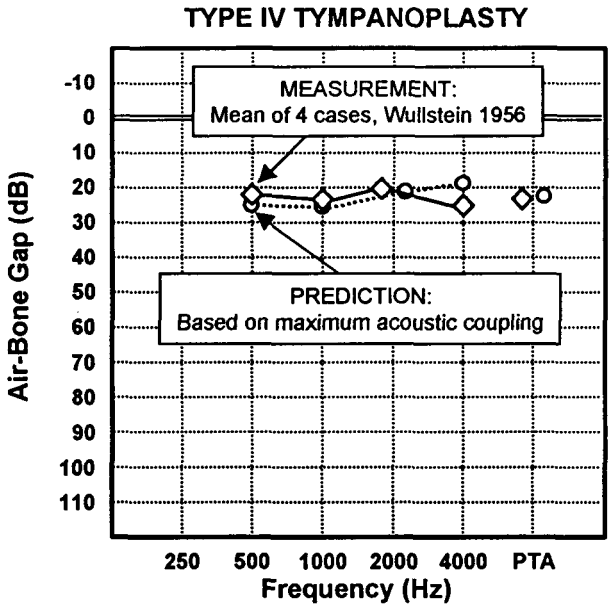

FIG. 7

Air-bone gaps after type IV tympanoplasty. Wullstein's best results (Wullstein, 1956) are compared with a prediction based on 'maximum' acoustic coupling. PTA = pure tone average of thresholds at 500 Hz, 1000 Hz and 2000 Hz. (Modified from Merchant *et al.*, 1995.)

The introduction of a tissue graft to shield the round window enhances acoustic coupling by increasing the sound pressure difference between the two cochlear windows. If the graft makes the round window sound pressure much less than the oval window pressure, then one achieves the condition of 'maximum' acoustic coupling, wherein acoustic coupling becomes equal to the oval window sound pressure. In terms of our model quantities, if P_{RW} is much smaller in magnitude than P_{OW}, then ΔP (= $P_{OW} - P_{RW}$) approximates P_{OW}. In a type IV tympanoplasty, the oval window sound pressure is the same as the ear-canal sound pressure, so that $\Delta P/P_{EC}$ = 1 or 0 dB. Therefore, in Figure 3, the acoustic coupling in an ideal type IV tympanoplasty would be represented by the dotted line at 0 dB. Because normal ossicular coupling P_S provides about 20 dB of pressure gain relative to the ear-canal sound pressure, maximum acoustic coupling is generally about 20 dB smaller than the normal P_S. Hence, the model predicts that type IV reconstructions that maximize acoustic coupling should produce an air-bone gap of only 20 dB. This prediction is consistent with the best clinical hearing results observed after Type IV tympanoplasty (Figure 7).

In clinical practice, results after type IV tympanoplasty vary widely, and all published clinical series report a wide range of air-bone gaps from 10 dB to 60 dB (Wullstein, 1960; Proctor, 1964; Lee and Schuknecht, 1971; Gotay-Rodriguez and Schuknecht, 1977; Merchant *et al.*, 1995). Satisfactory gaps of ⩽30 dB occur only in about 50 per cent of cases. We have been interested in determining the reasons for this variation and also for the occasional type IV patient with a 10 dB air-bone gap, which is better than the theoretically best result of 20 dB.

To these ends, we developed a simple model that directly relates the structure of the ear after type IV tympanoplasty to variations in the acoustic coupling of sound to the cochlea (Peake *et al.*, 1992; Rosowski *et al.*, 1995). The model is diagrammed in Figure 8A as an analogous electric circuit in which sound pressures are associated with voltages at 'nodes' (connection points denoted by circles) and acoustic volume velocities are related to circuit currents (denoted by arrows). The labelled pressures include the sound pressure at the oval window P_{OW} and at the round window P_{RW}. The labelled volume velocities include the volume velocity of the stapes U_S which is assumed to flow through the inner ear and leave the cochlea via the round window, and the volume-velocity of the shield U_{SH}. The model blocks represent impedances that describe the acoustic properties of the stapes Z_S, cochlea Z_C, acoustic shield Z_{SH} and the cavum minor air space Z_{CM}.

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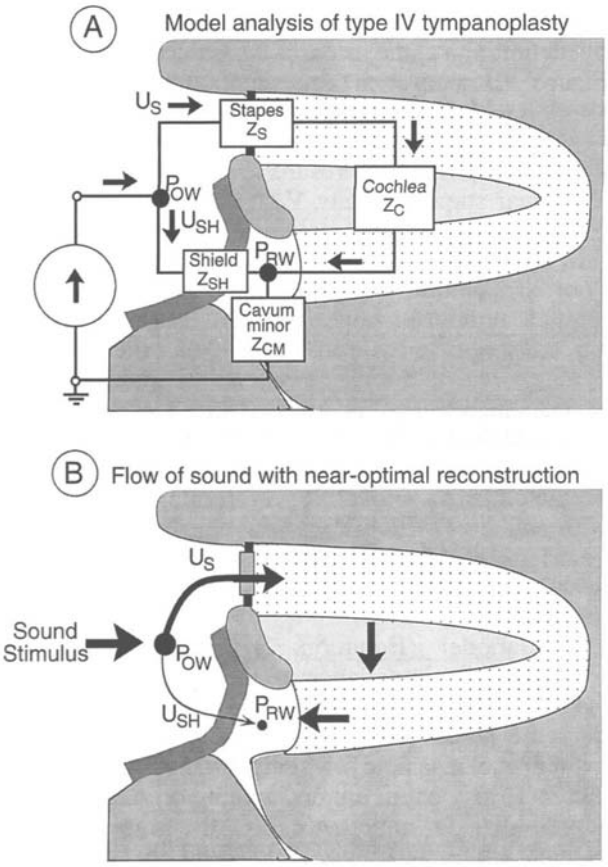

FIG. 8

Representations of the interaction of acoustic stimuli with the structures of a type IV tympanoplasty.

A. A lumped-element model. The model is diagrammed as an analogous electric circuit in which sound pressures are associated with voltages at 'nodes' (connection points denoted by circles) and acoustic volume velocities are related to circuit currents (denoted by arrows). See text for explanation of the model. $\mathbf{U_S}$ = stapes volume velocity, $\mathbf{U_{SH}}$ = volume velocity of the shield, $\mathbf{P_{OW}}$ = sound pressure at oval window, $\mathbf{P_{RW}}$ = sound pressure at round window, $\mathbf{Z_S}$ = impedance of the stapes footplate and annular ligament, $\mathbf{Z_C}$ = impedance of the cochlea, $\mathbf{Z_{SH}}$ = impedance of the graft-shield and $\mathbf{Z_{CM}}$ = impedance of the cavum minor. (Modified from Peake *et al.*, 1992 and Rosowski *et al.*, 1995.)

B. A schematic illustration of volume velocities and sound pressure magnitudes in a 'near' optimum type IV tympanoplasty. The thickness of the arrows schematizes the relative magnitudes of the volume velocities through the shield and stapes, while the size of the black circles schematizes the relative sizes of the sound pressures at the oval and round windows. The thick and stiff shield results in a small shield velocity $\mathbf{U_{SH}}$ while the large cavum minor air space results in a small round window pressure $\mathbf{P_{RW}}$. The net result is that the pressure difference $\Delta\mathbf{P} = \mathbf{P_{OW}} - \mathbf{P_{RW}}$ approximates $\mathbf{P_{OW}}$ and acoustic coupling is near maximum.

The way the impedance boxes are connected represents the physical constraints of the ear's structure. Sound coming in from the ear canal creates a pressure at the oval window ($\mathbf{P_{OW}}$) lateral to the footplate and the graft-shield. The sound pressure at the round window $\mathbf{P_{RW}}$ results from two mechanisms: displacements of the graft-shield change the volume of the cavum minor, thereby compressing the gas inside the closed volume and generating a sound pressure near the round window; similarly, sound driven displacements of the stapes and cochlear fluid move the round window and also change the cavum minor volume, thereby adding to $\mathbf{P_{RW}}$. Regardless of the source of $\mathbf{P_{RW}}$, it is the pressure difference between the oval and round window (acoustic coupling = $\Delta\mathbf{P} = \mathbf{P_{OW}} - \mathbf{P_{RW}}$) which drives the cochlea, and the greater this pressure difference, the larger the resulting stapes motion, and the better the hearing.

Optimum acoustic coupling to the normal cochlea and stapes requires that we maximize the pressure difference between the oval and round window $\Delta\mathbf{P} = \mathbf{P_{OW}} - \mathbf{P_{RW}}$ by making $\mathbf{P_{RW}}$ small. This optimum can be achieved by (1) restricting the flow of sound through the shield into the cavum minor, where a perfectly rigid high-impedance shield ($\mathbf{Z_{SH}}$ is approximately infinite) would reduce volume velocity flows across the shield to zero, $\mathbf{U_{SH}} = 0$, and by (2) producing a near zero impedance of the cavum minor ($\mathbf{Z_{CM}} \approx 0$) such that the pressure at the round window is small, $\mathbf{P_{RW}} \approx 0$, regardless of how much volume velocity flows into the cavum minor. The impedance of the cavum minor is inversely related to the air volume within it, and a small enough cavum minor impedance can be produced by an air volume of 0.1 ml or larger (Merchant *et al.*, 1997a). These predictions can also be derived from analysis of the analogue model which allows us to explicitly describe how each of the impedance boxes affects the stapes volume velocity that is produced by a given stimulus pressure, i.e.

$$\frac{\mathbf{U_S}}{\mathbf{P_{OW}}} = \frac{1}{\mathbf{Z_S} + \mathbf{Z_C} + \mathbf{Z_{CM}}\left(1 + \frac{\mathbf{Z_S}+\mathbf{Z_C}}{\mathbf{Z_{SH}}}\right)}. \qquad \text{(Equation 2)}$$

Equation 2 predicts that given normal $\mathbf{Z_S}$ and $\mathbf{Z_C}$, the largest stapes velocities will occur when (1) the shield impedance $\mathbf{Z_{SH}}$ is of much greater magnitude than $\mathbf{Z_S}$ and $\mathbf{Z_C}$, and (2) the cavum minor is aerated such that its impedance $\mathbf{Z_{CM}}$ is small compared to that magnitudes of the stapes and cochlea, i.e. $\mathbf{Z_{CM}} \approx 0$.

Figure 8B shows a more schematic representation of the model for a circumstance where the structural impedances are near optimal, the shield is nearly rigid but not perfectly so, and the cavum minor air volume is adequately large. The thickness of the arrows represents the relative magnitudes of the volume velocities through the shield and stapes, while the size of the black circles represents the relative sizes of the sound pressures at the oval and round windows. The near-rigid shield only allows a small volume-velocity to flow across it, while the relatively large cavum minor air space takes in the volume velocity of the shield and stapes without the generation of a large sound pressure.

We have developed a cadaveric temporal bone preparation to simulate the Type IV procedure, and to test the predictions of our model (Merchant *et al.*, 1997a). In our temporal bone preparation, the ear canal, tympanic membrane, malleus and incus were removed so as to expose the stapes and round window to the sound stimulus. A cavum minor

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chamber was constructed around the round window niche with dental cement. The surface of the chamber was covered by an acoustic shield that could be removed and replaced with shields made of different materials. We made measurements of the sound pressures at the oval and round windows ($\mathbf{P_{OW}}$ and $\mathbf{P_{RW}}$ respectively), and we also measured stapes velocity using an optical sensor. The value of the preparation was that we could individually vary each anatomical structure and test the predictions of the model on the measured variables.

Both the model analysis (Equation 2) and physical intuition predict that increasing stapes impedance decreases stapes volume velocity even though the acoustic coupling is large (Figure 9A; impedance is, by definition, a measure of resistance to motion). Figure 9B shows an experimental result from a temporal bone preparation showing stapes velocity ($\mathbf{V_S}$) plotted versus frequency (Note: All the experimental temporal bone results are plotted in terms of the linear stapes velocity $\mathbf{V_S}$ for convenience; $\mathbf{V_S} \times$ footplate area = stapes volume velocity $\mathbf{U_S}$. Further, only data points more than 15 dB above the noise floor are plotted in figures that show the experimental temporal bone results.) Baseline stapes velocity was measured, and then the annular ligament was allowed to become dry which made it stiffer, increasing its impedance (as has been demonstrated in live cat ears) (Lynch *et al.*, 1982). After drying, the stapes velocity decreased, as predicted by the model. Re-moistening the ligament with saline caused the stapes velocity to return to near baseline levels, demonstrating that the effect of decreased velocity was a consequence of drying of the ligament.

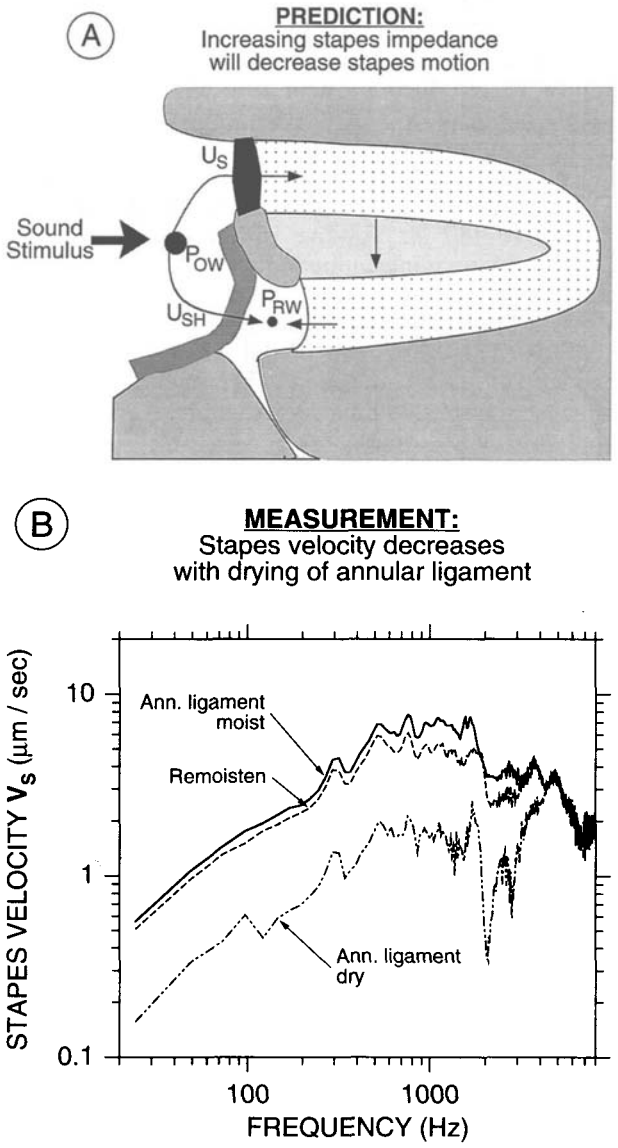

FIG. 9

Type IV tympanoplasty – The effect of increase in impedance of the stapes footplate:

A. A schematic showing the prediction that increasing the impedance of the stapes, e.g. by fixation, will lead to a reduction in stapes volume velocity $\mathbf{U_S}$.

B. Measurements in the temporal-bone preparation demonstrating that reversible stiffening of the annular ligament by drying leads to a reduction in stapes velocity. The y-axis shows magnitude of stapes velocity produced by a sound pressure difference of 1 pascal (94 dB SPL) between the oval and round windows. (Modified from Merchant *et al.*, 1997a.)

The model (Equation 2) also predicts that increasing the impedance of the cavum minor (for instance, by reducing its volume by saline filling) will decrease stapes velocity (Figure 10A) even in the presence of a shield of adequate impedance. The increase in cavum minor impedance adds to the stapes-cochlear impedance, i.e. the stapes has to compress the 'incompressible' fluid in the cavum minor as well as the cochlear fluid and thereby stapes velocity decreases. Also, with a fluid-filled cavum minor, more of the input pressure goes into compressing the fluid in the cavum minor with the result that the window pressure difference $\mathbf{P_{OW}} - \mathbf{P_{RW}}$ decreases. In the temporal-bone preparation, increasing the cavum minor impedance by filling with normal saline (the impedance of saline is much greater than that of air) results in a greatly reduced stapes velocity (Figure 10B).

An additional prediction of the model is that the addition of air bubbles to a fluid-filled cavum minor will restore detectable stapes motion by restoring some compressibility to the cavum minor impedance (Figure 11A). The prediction was tested in the temporal bone preparation, and the effect of an air bubble within the fluid was found to vary with location and size of the bubble (Figure 11B). For example, a 1 to 2 μl bubble at the round window membrane was sufficient to restore stapes velocity for frequencies >700 Hz, whereas the same sized bubble at the shield had only a small effect (Figure 11B). On the other hand, a 20 μl air bubble at any location within the cavum minor was sufficient to restore stapes velocity at all frequencies (data not shown in Figure 11B). Dependence of the effect of an air bubble on position in a fluid-filled cavum minor is not predicted by our simple model. It indicates that our model which treats the entire cavum minor as a single space must be modified to represent this feature of the results.

Another prediction of the model is that stapes velocity will decrease if the shield impedance is decreased (Equation 2 and Figure 12A). An

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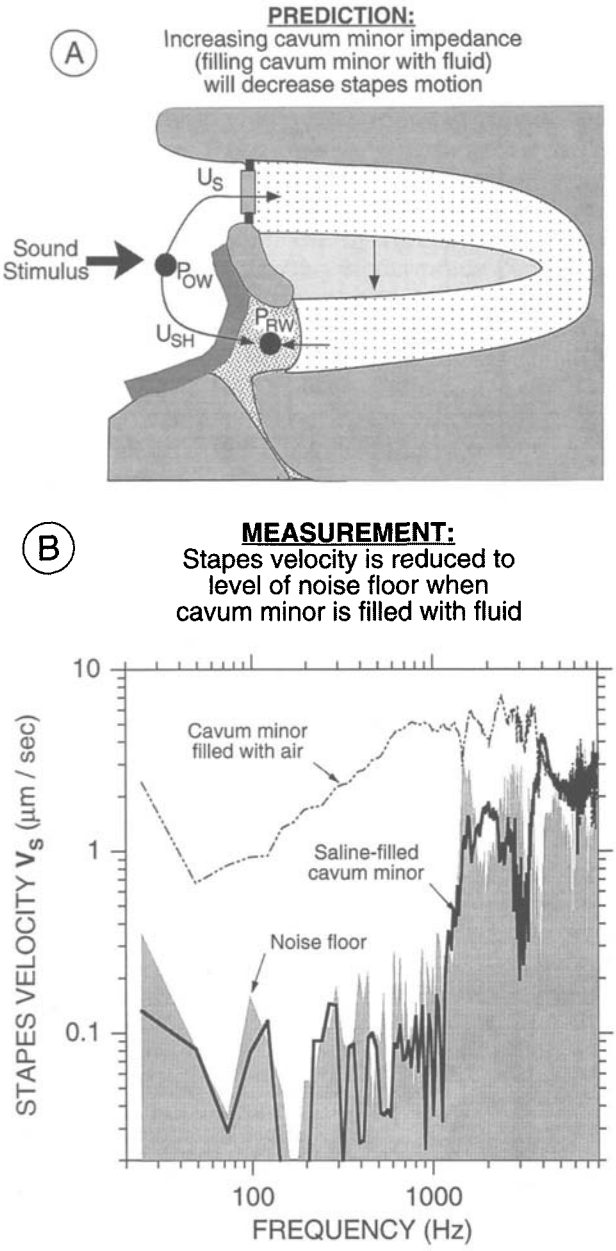

FIG. 10

Type IV tympanoplasty – The effect of increase in impedance of the cavum minor:

A. A schematic showing the prediction that increasing the impedance of the cavum minor (e.g., by filling the cavum minor with saline) will reduce the difference in pressure between the cochlear windows and thereby reduce stapes volume velocity U_S, even if the shield is very stiff.

B. Measurements in the temporal-bone preparation demonstrating that filling the cavum minor with physiological saline reduces stapes velocity to the level of the noise floor. The y-axis shows magnitude of stapes velocity produced by an oval window sound pressure of 1 pascal (94 dB SPL). (Modified from Merchant *et al.*, 1997a.)

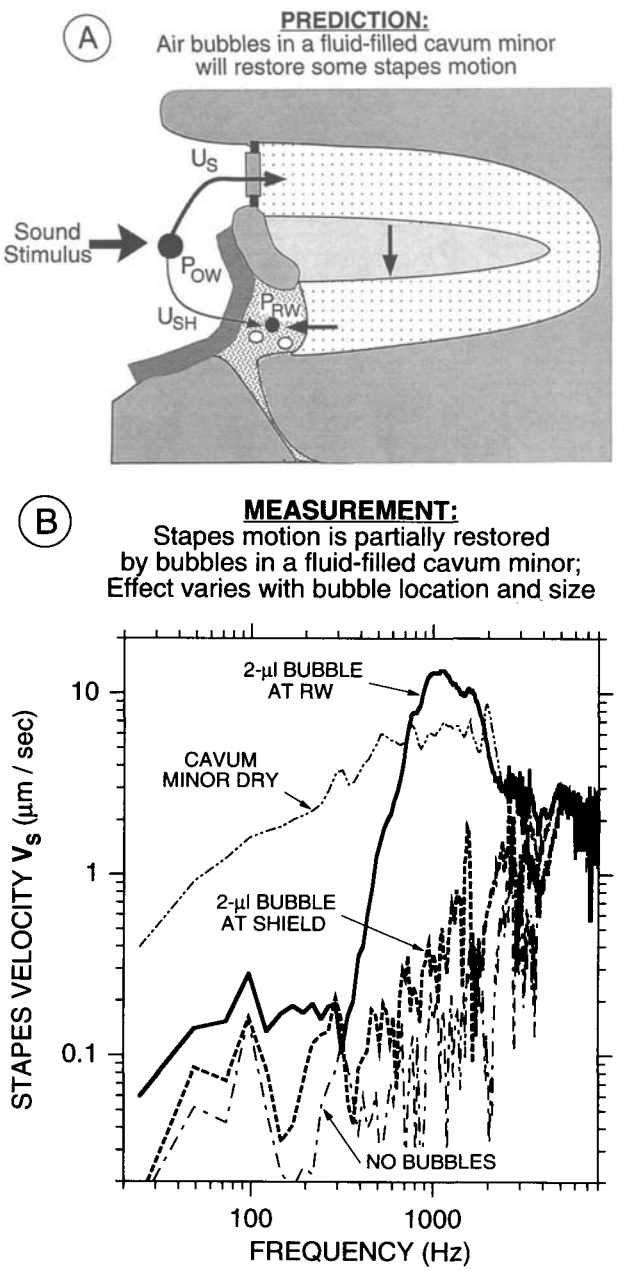

FIG. 11

Type IV tympanoplasty:

A. A schematic showing the prediction that adding air bubbles to a fluid-filled cavum minor will restore some of its compressibility, and lower its impedance. The reduced impedance will lead to a decrease in the sound pressure at the round window and make it easier to move the stapes.

B. Measurements in the temporal-bone preparation demonstrating that adding air bubbles to a fluid-filled cavum minor restores some of the lost stapes velocity. The effect of the bubble varies with bubble size and location. The y-axis shows magnitude of stapes velocity produced by an oval window sound pressure of 1 pascal (94 dB SPL). (Modified from Merchant *et al.*, 1997a.)

insufficiently stiff shield will displace more with sound and will produce larger sound pressures in the cavum minor such that the round-window sound pressure will become more comparable to the sound pressure at the oval window, and the sound-pressure difference that drives the stapes and cochlea will be decreased. This prediction was tested in the temporal bone preparation by using shields of different impedances. The largest stapes velocities were achieved with highly impedant shields made of cartilage or Silastic™, 1 mm thick. Shields made of one or two layers of temporalis fascia were not as effective at frequencies below 1000 Hz, and the measured stapes velocities with

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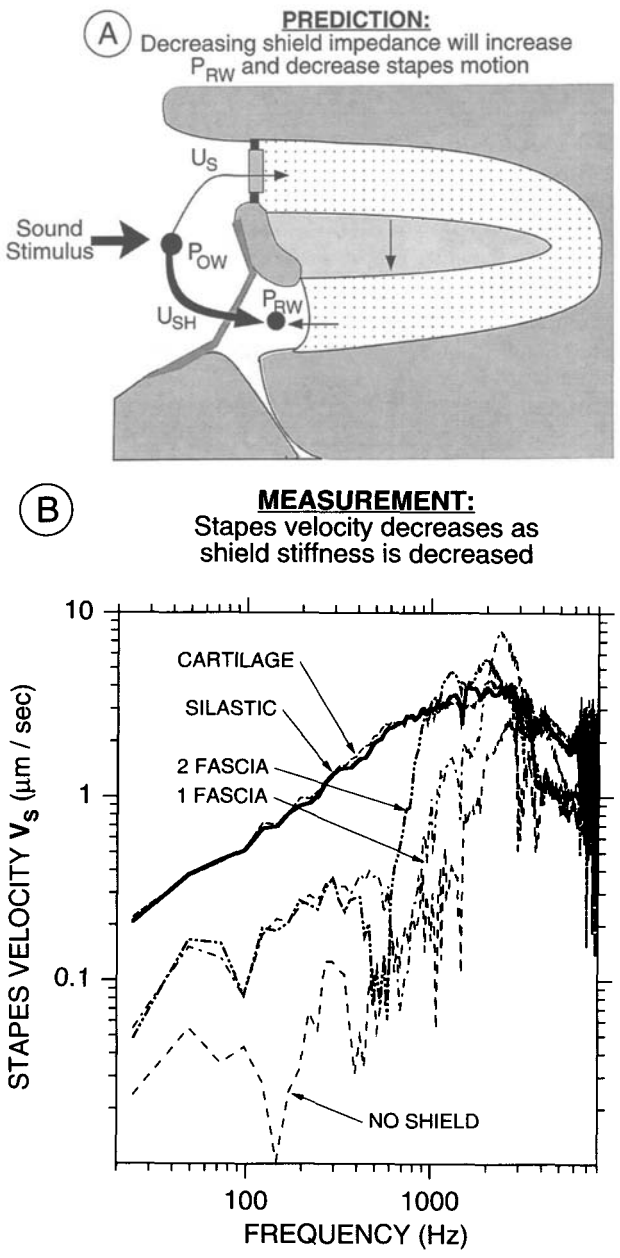

FIG. 12

Type IV tympanoplasty – The effect of varying shield impedance: A. A schematic showing the prediction that despite a moderately large cavum minor volume, a flaccid acoustic shield will allow enough volume velocity into the cavum minor to increase round window sound pressure, thereby decreasing the window pressure difference, and leading to a decrease in stapes volume velocity U_S.

B. Measurements in the temporal-bone preparation demonstrating that stapes velocity is directly related to the impedance of the shield. The y-axis shows magnitude of stapes velocity produced by an oval window sound pressure of 1 pascal (94 dB SPL). (Modified from Merchant *et al.*, 1997a.)

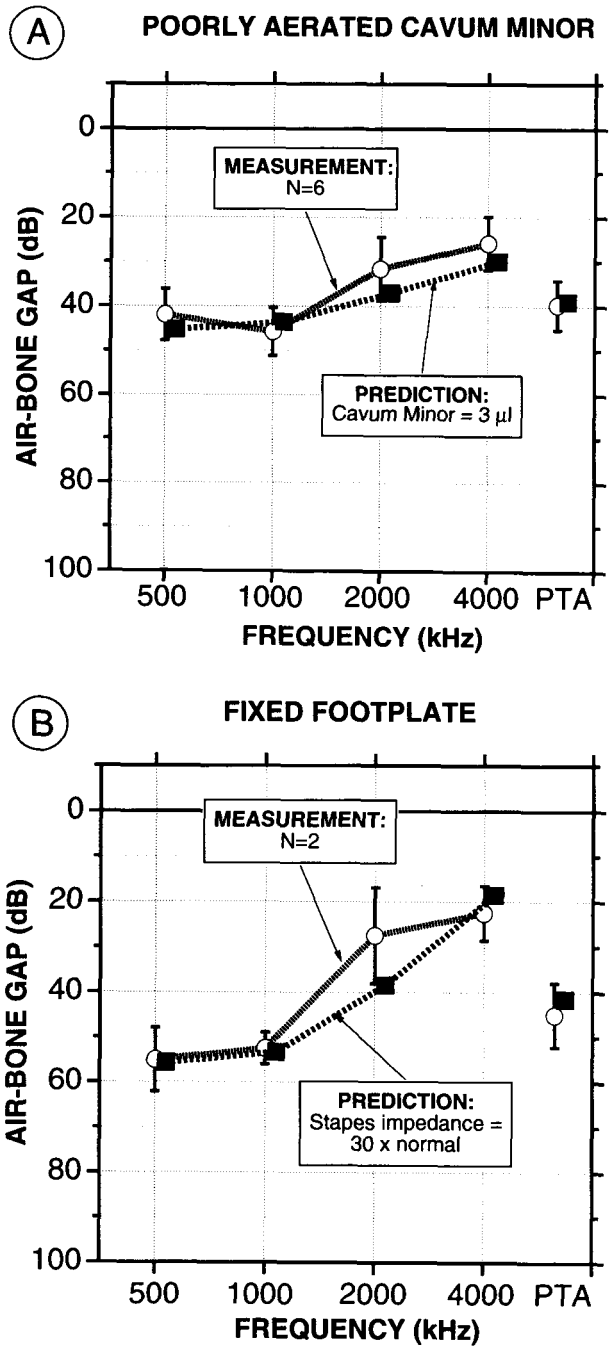

FIG. 13

A. Type IV tympanoplasty: hearing results of six clinical cases with documented poor aeration of cavum minor (mean ± one standard error of air-bone gaps are plotted) are compared to model prediction for a poorly-aerated cavum minor containing only 3 μl of air. PTA = pure tone average. (Modified from Merchant *et al.*, 1995.)

FIG. B. Type IV tympanoplasty: hearing results of two cases with fixed footplate (mean ± one standard error of air-bone gaps are plotted) are compared to model prediction for stapes impedance 30 times greater than normal. PTA = pure tone average. (Modified from Merchant *et al.*, 1995.)

fascia shields were 10–15 dB lower for frequencies below 1000 Hz when compared to shields made of cartilage or Silastic (Figure 12B).

As a test of the relevance of the model to clinical experience, we compared its predictions against clinical audiometric data in the literature and gathered from our hospital (Merchant *et al.*, 1995; Rosowski *et al.*, 1995). As shown in Figure 7, the model predictions under optimal conditions match the best published hearing results. These optimum conditions include a mobile footplate, an aerated cavum minor and a sufficiently stiff shield (Peake *et al.*, 1992; Rosowski *et al.*, 1995). We also evaluated the hearing results of 27 type IV procedures

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performed in our institution (Merchant *et al.*, 1995; this retrospective review comprised all those type IV cases that had post-operative audiograms *and* had clinical data documenting the status of the cavum minor and stapes footplate). Within this group of 27, six ears had documented non-aeration or poor aeration of the cavum minor, as determined by otoscopic examination. The mean hearing level of these six cases matches the model prediction for a very small cavum minor air space of only 3 μl (Figure 13A). We also had two cases where the footplate had bony ankylosis. These two cases had a large (55 dB) low frequency conductive loss. The model prediction for a stapes impedance 30 times greater than normal matches the observed loss (Figure 13B).

The practical application of our analytical model is the development of simple surgical guidelines to help clinicians improve post-operative hearing results in type IV tympanoplasties (Merchant *et al.*, 1995; Rosowski *et al.*, 1995; Merchant *et al.*, 1997a). The first guideline is to *promote aeration of the cavum minor and preserve round window mobility.* Every effort should be made to avoid occlusion of the tympanic orifice of the Eustachian tube by fibrous tissue and to prevent formation of fibrous adhesions between the shield and the medial wall of the hypotympanum by saving all healthy mucosa lining the protympanum, hypotympanum and round window. However, one should remove mucosa and expose bone in the region surrounding the oval window in order to allow the shield graft to adhere to the margins of the oval window niche, thereby acoustically isolating the round window from the oval window. The second guideline is to *make the round window graft-shield as stiff as possible.* Temporalis fascia alone is not sufficient. We recommend that a crescent-shaped piece of tragal or meatal cartilage be used to shield the round window. It is helpful to elevate the perichondrium from the medial surface of such a cartilage graft and turn it as a flap onto the inferior canal wall in order to stabilize the cartilage graft. A large piece of temporalis fascia is then placed superficial to the cartilage graft and a U-shaped aperture is cut in the fascia to keep the stapes footplate exposed. A crescent shaped piece of 1 mm thick Silastic can be used instead of cartilage. The third guideline is that *the footplate should not be covered by the fascia* since fascia is thick and will increase the impedance of the annular ligament. Instead, a *very thin split-thickness skin graft* taken from the post-auricular or inner arm skin should be invaginated into the oval window niche. The skin graft is held in place by a small cotton ball soaked in an oil-based antibiotic solution. The cotton ball is left in place for three weeks post-operatively.

An additional interesting aspect of the type IV analysis is that our temporal bone measurements have shown that the cochlear impedance varies by ±12 dB among normal individuals (Merchant *et al.*, 1996). These natural variations can explain small air-bone gaps <20 dB that occur occasionally in patients who have undergone type IV tympanoplasty (Merchant *et al.*, 1995). For example, provided the other parameters are optimal, a low value of cochlear impedance will make it easier for sound at the oval window to move the stapes footplate and to elicit a cochlear response.

A modified type V tympanoplasty procedure is similar to a type IV and is performed when the stapes footplate is ankylosed (Gacek, 1973). The footplate is removed and is usually replaced by a fat graft. It is reasonable to assume that the impedance of the fat graft will be less than that of the normal footplate. Hence, we would predict that the average hearing results for a type V would be better than those for a type IV, especially for the low frequencies. This prediction is supported by clinical evidence. Two reports summarizing results of the type V procedure (Gacek, 1973; Montandon and Chatelain, 1991) show better hearing results on average than reports dealing with type IV results (Wullstein, 1960; Proctor, 1964; Lee and Schuknecht, 1971; Gotay-Rodriguez and Schuknecht, 1977; Merchant *et al.*, 1995). In Montandon and Chatelain's series of 64 cases, 51 per cent had post-surgical air-bone gaps less than 20 dB. Furthermore, of their 22 cases with conditions favourable for aeration of the cavum minor, 86 per cent had an air-bone gap less than 20 dB.

Type I, II and III tympanoplasty

Types I, II and III tympanoplasty procedures involve reconstruction of the tympanic membrane and/or the ossicles (Wullstein, 1956; Nadol and Schuknecht, 1993). We wish to generalize the framework of Equation 1 to these tympanoplasty procedures as well, but our understanding of these more complex mechanical reconstructions is much less complete. However, one can state that, in general, the hearing result will depend on the adequacy of *middle-ear aeration* (including the *static air pressure* in the middle ear), the efficacy of the reconstructed *tympanic membrane* and the efficacy of the reconstructed *ossicular chain.*

Aeration of the middle ear and round window is critical to the success of any tympanoplasty procedure. Clinical experience has shown that non-aerated ears often demonstrate 40–60 dB air-bone gaps (Nadol and Schuknecht, 1993; Merchant *et al.*, 1997b). Figure 14 (A–D) is a case report where a partial ossicular replacement prosthesis (PORP) was placed between a reconstructed tympanic membrane and the stapes head. Hearing results are shown for two conditions (aerated and non-aerated middle ear) with the same tympanic membrane-PORP-stapes linkage in the two conditions. The air-bone gap is 20–25 dB in the aerated condition (Figure 14D) and 40–55 dB in the non-aerated condition (Figure 14A). These results can be explained in the context of the framework of Equation 1: in the aerated middle-ear, acoustic coupling and cochlear impedance are assumed to be normal, and the tympanic membrane-PORP-stapes coupling is assumed to be 20–25 dB less than normal ossicular coupling. In the non-aerated middle ear, ossicular coupling is

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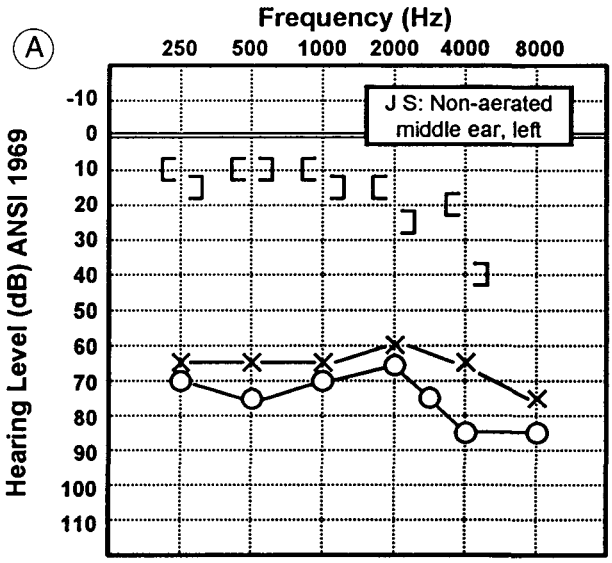
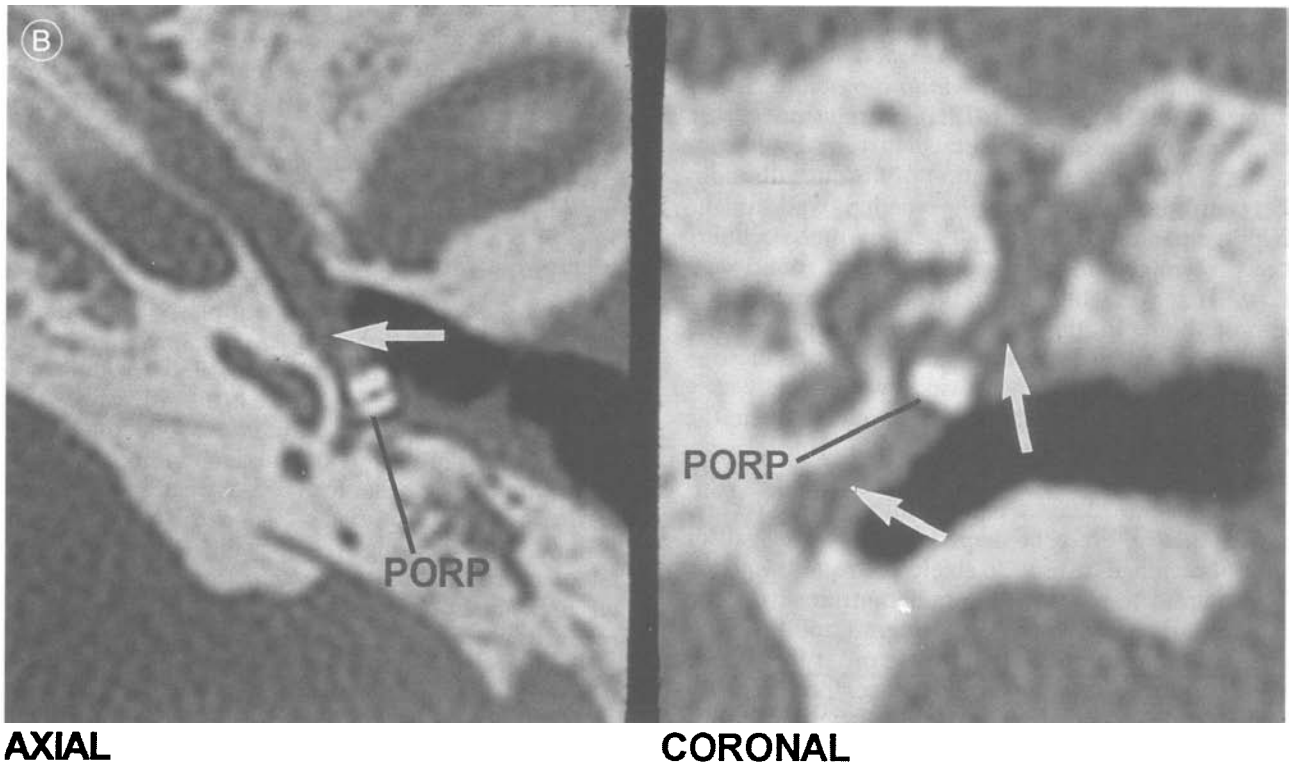


FIG. 14 A and B

Case report of a 47-year-old woman with bilateral chronic otitis media who underwent bilateral ear operations with successful control of her infections. However, she was left with bilateral, large, 45–60 dB conductive losses (A). The left ear had undergone placement of a hydroxyapatite PORP between the stapes and the tympanic membrane (TM) graft. Her CT scan in axial and coronal cuts (B) shows non-aeration (indicated by arrows) of the bony eustachian tube, middle ear and mastoid. The PORP is in good position. A tympanomeatal flap was elevated, fibrous adhesions were lysed between the TM-graft and the promontory and a Goode T-tube was placed. The attachment of the TM-graft to the PORP was not disturbed.



greatly reduced and stapes-cochlear impedance is increased because the round window membrane cannot move freely. Clinical observations such as this case report suggest that if aeration is adequate, then less-than-optimal tympanic membrane-ossicle configurations can still result in air-bone gaps $\leq$20–25 dB as long as the tympanic membrane-ossicle linkage to the footplate is preserved and stapes mobility is normal.

In the case report of Figure 14, non-aeration of the middle ear (manifested as fibrous tissue in the middle-ear space) was felt to be the result of a non-functioning Eustachian tube. Aeration was restored by elevating a tympanomeatal flap, lysing fibrous adhesions between the tympanic membrane and the promontory, and placing a Goode T-tube in the tympanic membrane. In contrast, some non-aerated, post-tympanoplasty ears exhibiting large 45–50 dB air-bone gaps have fibrous tissue filling the mesotympanum, while the Eustachian tube is apparently functional as indicated by aeration of the protympanum. The hearing in such ears can be improved without using a ventilation tube if aeration of the mesotympanum and round window (via the Eustachian tube) can be re-established by elevating a tympanomeatal flap, lysing adhesions between the flap and promontory and placing a piece of Silastic or Teflon sheeting in the middle ear to promote mucosalization and aeration of the mesotympanum and round window. (For a case report showing such a situation, the reader is referred to Merchant *et al.*, 1997b.)

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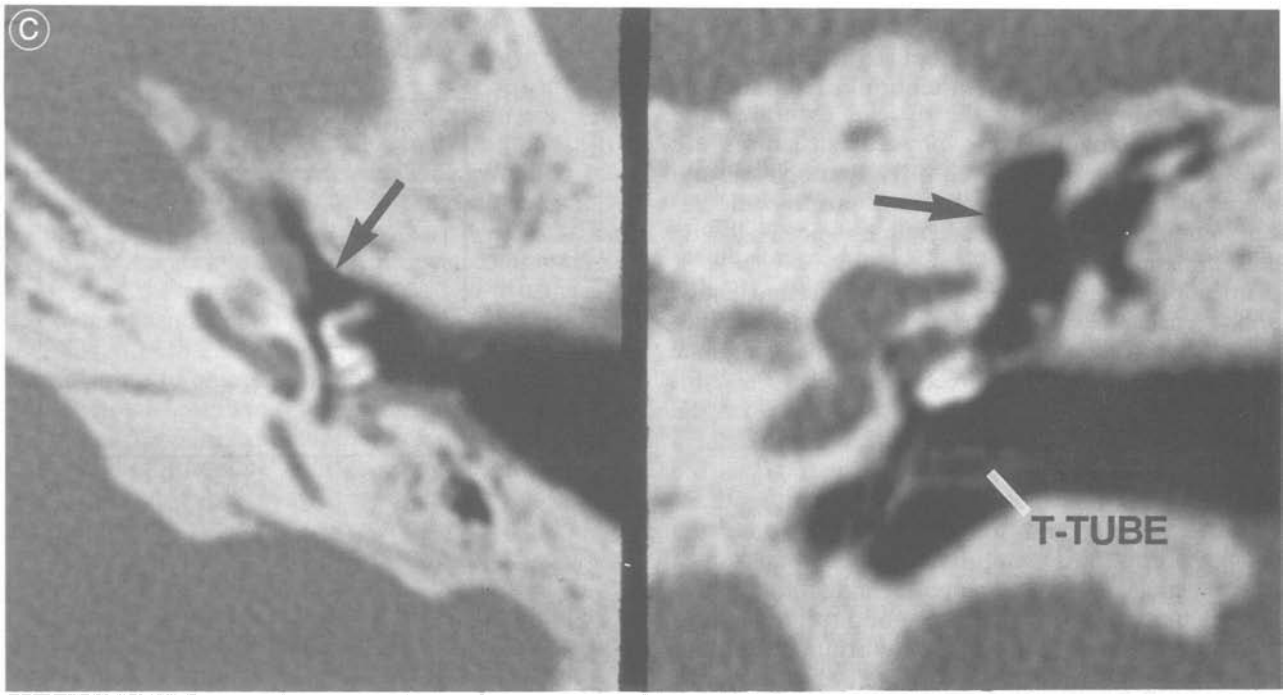

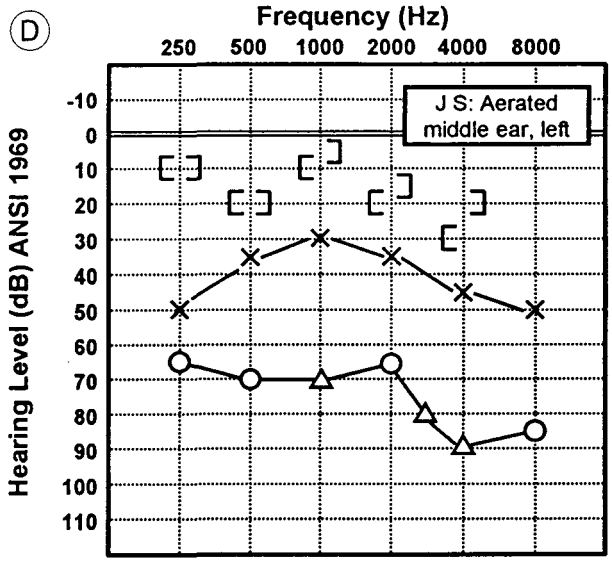

FIG. 14 C and D

Her post-operative CT scan (C) shows establishment of middle-ear aeration (indicated by arrows) and there was closure of her air-bone gap to within 20 to 25 dB for most frequencies (D).

How much aeration of the middle ear is necessary? Shown in Figure 15A are predictions of the effects of varying the air volume of the middle ear and mastoid. The baseline is taken to be the normal combined middle-ear and mastoid volume of 6 ml (Molvaer *et al.*, 1978). As the volume is decreased, a low frequency hearing loss is predicted (Rosowski and Merchant, 1995). A combined middle ear and mastoid volume of 0.5 ml is predicted to cause about a 10 dB air-bone gap. Volumes less than 0.5 ml are predicted to result in progressively larger gaps, while volumes greater than 1 ml provide little additional acoustic benefit. The clinical implication is that in a canal wall down tympanomastoidectomy, the lack of a mastoid air space will not be detrimental from an acoustical perspective, provided there is at least 0.5 ml of air behind the drum graft. An interesting but yet unresolved question is, 'where should the air be?' Must the air be at the round window, at the oval window, behind the tympanic membrane or all three? A compressible air cushion at the cochlear windows has been demonstrated to be important for stapes and cochlear fluid motion (Figure 11B). It is also clear that air is necessary behind the tympanic membrane to enable ossicularly coupled sound to reach the cochlea. We are currently investigating the issue of influence of location of the air space.

Another parameter that can influence the hearing result is the *static air pressure in the middle-ear space.* The mechanisms and structural alterations by which static-pressure changes reduce middle-ear sound transmissions are not well defined, and possible

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sites of pressure sensitivity include the tympanic membrane, the annular ligament (Lynch *et al.*, 1982), the incudo-malleal joint (Hüttenbrink, 1988) and the suspensory ligaments of the ossicles (Murakami *et al.*, 1997). Some of these physical structures are drastically altered as a result of tympanoplasty, and the acoustic effects of negative and positive middle-ear static pressure in such ears need to be determined.

In addition to middle-ear aeration and static pressure, the success of type I, II and III tympanoplasty depends on the efficacy of the tympanic membrane and ossicular reconstruction. *The tympanic membrane* is the major transducer of the middle ear. Our knowledge of the mechanics of a reconstructed drum is somewhat rudimentary at the present time. Clinical observations suggest that those techniques that tend to preserve or restore the drum's normal anatomy give good hearing results. This is especially true when performing a total drum replacement, in which case, one good technique is to place the graft medial to the manubrium and to use skin grafting plus packing to restore a sharp anterior tympano-meatal angle (Nadol and Schuknecht, 1993).

Issues relevant to the mechanics of *ossicular reconstruction* include *stiffness* and *mass* of the reconstructed ossicle, its *position, tension, length* and *coupling*. In general, the stiffness of an ossicle strut will not be a significant factor as long as the strut stiffness is much greater than that of the stapes-cochlear impedance (Merchant *et al.*, 1997b). For clinical purposes, implants and struts made of ossicles, cortical bone and synthetic materials generally meet this requirement.

Analysis of our middle-ear model (Rosowski and Merchant, 1995) suggests that middle-ear sound transmission should not be significantly affected by substantial increases in ossicular mass. Shown in Figure 15B are model predictions of air-bone gaps resulting from increasing the mass of an ossicle strut, relative to the stapes mass which is 3 mg. Increases up to 16 times are predicted to cause less than 10 dB conductive loss and only at frequencies >1000 Hz. In contrast to this result, Nishihara *et al.* (1993) found using a temporal bone preparation, that in two bones, a mass of 5 mg added to the stapes head led to a 13–15 dB loss at high frequencies. Thus, the acoustic effects of variations in the mass of an ossicular strut need to be clarified.

Where should an ossicular prosthesis be positioned in relation to the tympanic membrane and the manubrium? Because the tympanic membrane couples sound to the manubrium, it makes intuitive sense to place the prosthesis at the manubrium. However, in most chronic ears, the anatomy is such that the manubrium is unfavourable, and it is more stable to place the prosthesis from the stapes head to the postero-superior quadrant of the tympanic membrane. Experimental temporal bone data (Goode and Nishihara, 1994) suggest that positioning a prothesis against the drum will give acceptable results, provided the diameter of the prosthesis in

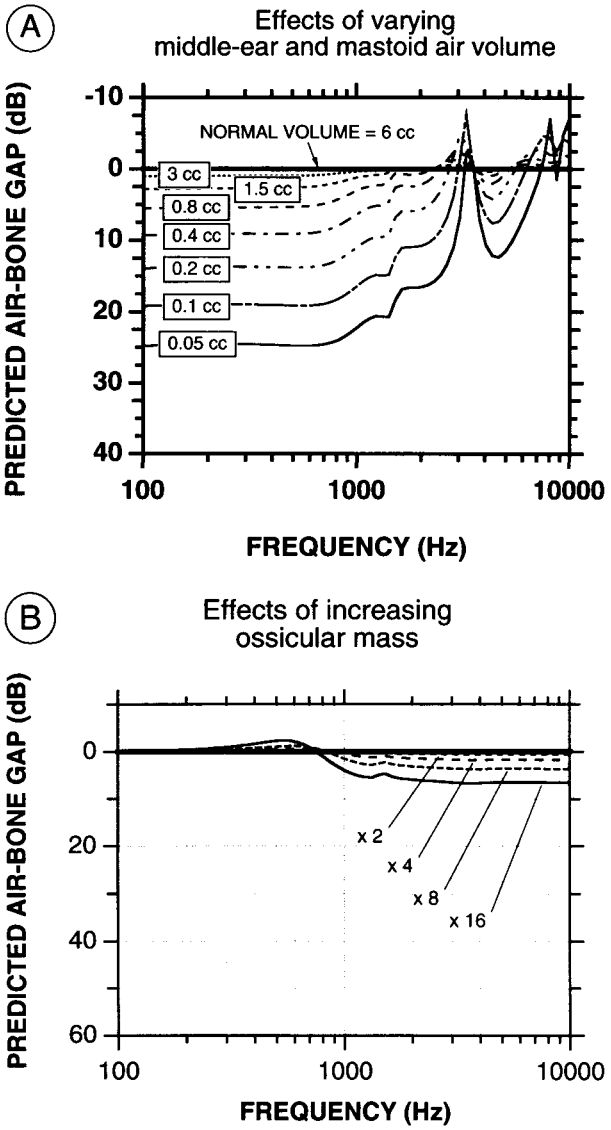

FIG. 15

A. Model prediction of the effects of decreasing the volume of the middle ear and mastoid. The baseline is taken to be the normal volume of 6 ml. (Modified from Rosowski and Merchant, 1995; the reader is referred to this paper for a discussion of the model analysis used to make the predictions shown in A and B.)

B. Model prediction of the effect of increasing the ossicular mass. The mass of an ossicular strut is increased as shown. These increases are relative to the stapes mass which is 3 mg. (Modified from Rosowski and Merchant, 1995.)

contact with the tympanic membrane is at least 3–4 mm. Human temporal bone measurements have also indicated that the angle between the stapes and a prosthesis should be less than 45 degrees for optimal sound transmission (Vlaming and Feenstra, 1986; Nishihara and Goode, 1994).

Tension and length of an ossicular prosthesis are important in determining the hearing result (Nishihara and Goode, 1994). Too long a prosthesis causes undue tension within the tympanic membrane and annular ligament and will lead to a significant air-bone gap. Currently, the assessment of tension cannot be made intra-operatively in an objective

fashion, and a reliable method would be useful to the otosurgeon.

Coupling refers to how well the motion of the footplate 'follows' the motion of the prosthesis and the tympanic membrane. The degree of coupling can be measured by ascertaining the relative motion of the tympanic membrane, prosthesis and stapes; poor coupling is indicated when large motions of the tympanic membrane fail to produce comparable motions of the prosthesis or footplate. A prosthesis will only transmit sound effectively if there is 'good' coupling at both ends. Clinical observations indicate that it is rare to obtain a firm bony union between a TORP (total ossicular replacement prosthesis) and the stapes footplate, and hence, inadequate coupling at the TORP-footplate interface may be an important cause of a persistent post-operative air-bone gap. The physical factors that control coupling have not been determined in a quantitative manner, and further study of this parameter is warranted. To summarize, it would be safe to say that we have only a limited understanding of the mechanics of ossicular reconstructions and much work remains to be done.

Canal wall down mastoidectomy

A canal wall down mastoidectomy poses three acoustical considerations. First, the residual middle-ear air space is significantly reduced. However, as long as this air space is more than 0.5 ml, the resulting hearing loss should be less than 10 dB (see above). Second, a canal wall down procedure creates a relatively large air space lateral to the tympanic membrane, i.e. the air space within the mastoid bowl including the external auditory canal. This mastoid bowl and ear canal air space generates resonances which can influence middle-ear sound transmission favourably or unfavourably (Goode *et al.*, 1977). For example, Goode *et al.* (1977) described a patient with a 4.1 ml radical mastoid cavity that had a 25–30 dB resonance for frequencies between 2500 and 4000 Hz which was felt to contribute to the patient's good hearing at these higher frequencies. The structure-function relationships between the size and shape of a given mastoid cavity and its resonances have not been well defined. An improved understanding of this issue should help to develop guidelines for an otologist in order to configure mastoid cavities in ways that could be acoustically beneficial. Third, after a canal wall down procedure, the tympanic membrane graft comes to lie in a more medial position compared to normal, and the graft usually couples to the stapes head or to a TORP. The mechanics of such a graft and its coupling to the stapes/TORP are likely to be different from normal and need to be characterized. For example, when placed against the stapes head, such a graft is often in contact with the promontory and tympanic segment of the facial canal; such contact may limit the functional surface area of the graft. When a TORP is considered, if the top of the TORP rises much above the level of the oval window niche, the TORP tends to extrude, whereas, if the TORP is kept low, the prosthesis often settles against the margins of the niche.

Stapedotomy

Finally, a brief comment on the mechanics after a stapedotomy. As previously noted, the output of the middle ear can be quantified by the volume-velocity of the stapes, $\mathbf{U_S}$. $\mathbf{U_S}$ is equal to the stapes (linear) velocity multiplied by the area of the stapes footplate. After a stapedotomy, the effective area of the footplate is reduced to the area of the prosthesis, thereby reducing the volume velocity produced by a given stapes (linear) velocity. The reduction in effective footplate area also reduces the area of the cochlear fluid over which the force generated by the stapes is applied. While the reduced footplate area leads to a local increase in pressure over the surface of the prosthesis area, the average pressure at the cochlear entrance is reduced. The reduction in stapes volume-velocity and cochlear sound pressure lead to a decrease in ossicular coupling and the development of an air-bone gap (Rosowski and Merchant, 1995). The smaller the area of the stapes prosthesis, the greater the air-bone gap. Using such analysis, we have made model predictions of the relationship between piston diameter and residual air-bone gap after a stapedectomy (Rosowski and Merchant, 1995). These predictions apply mainly to the lower frequencies below 1000 Hz. A 0.8 mm piston is predicted to cause a 5 dB gap, a 0.6 mm piston is predicted to cause a 10 dB gap and 0.4 mm piston is predicted to cause a 15 dB gap. These predictions assume that the effective vibrating 'footplate' surface area after a stapedotomy is no more than the area of the lower end of the prosthesis. In cases of partial stapedectomy with placement of a vein graft and a stapes prosthesis, the effective vibrating surface may be greater than the area of the prosthesis alone, and our predictions may overestimate the air-bone gap.

Conclusions

The following conclusions are relevant to a clinician performing reconstructive middle-ear surgery:

(1) For a type IV tympanoplasty, one should use cartilage or Silastic to reinforce the fascia graft used to shield the round window, cover the footplate with a thin skin graft, promote aeration of the cavum minor, and preserve mobility of the round window membrane.

(2) For a type V tympanoplasty, which is an option if the footplate is ankylosed, the footplate should be removed and replaced by a compliant tissue graft such as fat.

(3) Aeration is critical to the success of types I, II and III tympanoplasty, and the air volume behind the tympanic membrane should be at least 0.5 ml.

(4) The stiffness of an ossicular strut or prosthesis is probably of little clinical concern because most strut materials are as stiff as the ossicles that they replace.

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(5) The effect of mass of an ossicular strut or prosthesis is not clear. Our model analyses suggest that ossicular prostheses of masses as large as 16 times the stapes have little effect on ossicular function, while the experimental work of Nishihara *et al.* (1993) showed 10–15 dB losses in high frequency middle-ear function when mass was added to the ossicular system.

(6) The positioning of ossicular replacement prostheses is an important factor. Struts should make an angle of less than 45 degrees with the stapes. The strut should be placed against the manubrium, though a connection to the tympanic membrane is acceptable as long as the contact surface is at least 3–4 mm. Tension and coupling are critical parameters, but their mechanics are not well understood at the present time.

(7) The diameter of a stapes replacement prosthesis should be at least 0.6 mm for optimal results.

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