Barotrauma during air travel: predictions of a mathematical model

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Kanick, Stephen Chad, and William J. Doyle. Barotrauma during air travel: predictions of a mathematical model. J Appl Physiol 98: 1592–1602, 2005. First published December 17, 2004; doi:10.1152/japplphysiol.00974.2004.—Middle ear barotrauma during flight is a painful disorder experienced by passengers who cannot properly regulate their middle ear pressure in response to the changing cabin pressures during ascent and descent. Previous reports emphasized the important role of poor eustachian tube function in disease pathogenesis but paid little attention to other moderating factors. Here we describe a mathematical model of middle ear pressure regulation and simulate the pressure response to the changes in cabin pressure experienced over typical flights. The results document buffering mechanisms that decrease the requisite efficiency of active, muscle-assisted eustachian tube opening for disease-free flight. These include the relative difference between destination and departure elevations and the ratio of maximum tympanic membrane volume displacement to middle ear volume, where greater absolute values require lesser efficiencies for disease-free flight. Also, the specific type of functional deficit is important since ears with a completely obstructed eustachian tube can be less susceptible to barotrauma than those with a eustachian tube that passively opens but fails to dilate in response to muscle activity. These buffering systems can explain why some children and adults with poor eustachian tube function do not experience middle ear barotrauma.

ME Pressure Regulation

Barotrauma is caused by an inability to maintain near pressure equivalence between the ME (PME) and airplane cabin (PCabin) as the latter is changed rapidly during ascent and descent. Normally, the pressure of the fluid-free ME is near ambient (Pam) (PME ≈ Pam ≈ PCabin), which ensures free vibration of the TM and efficient transduction of sound energy to the inner ear. Because the ME is usually a closed, relatively noncollapsible, temperature-stable, mucosal-lined bony cavity, its pressure is a direct function of the contained gas volume, and gas transfers to or from the ME change its pressure.

The ME consists of two functionally discrete but continuous air spaces: the anterior tympanum, which contains the ossicles, ligaments, and muscles of the sound transducer mechanism; and the posterior mastoid cavity, which is subdivided into numerous intercommunicating air cells (5). While the variance among individuals and age groups in tympanum volume (Vtym) is low (Vtym ≈ 1 ml), that of the mastoid (Vmax) is large (Vmax ≈ 0–15 ml) due to contributions of age, gender, and disease history effects (37, 46). The anterior wall of the tympanum is continuous with the osseous portion of the ET, the lateral wall includes the TM, the medial wall includes the round window membrane, and the posterior wall opens to the mastoid air space by way of a large air cell, the antrum (5).

Figure 1A shows the various gas exchange pathways for the ME when isolated within an airplane cabin. The tympanum can exchange gas with the external environment via the TM and with the inner ear via the round window, but experimental measurements show that transfers across these pathways are negligible (18, 21). Therefore, in describing PME regulation, the physiologically relevant pathways are as follows: tympanum-antrum-mastoid, ME-MEM-blood, and tympanum-ET-nasopharynx (NP). Because the tympanum and mastoid are continuous in the air phase, total pressure differentials are rapidly equilibrated, and established gas partial-pressure differentials decay quickly (24). ME-MEM-blood-gas exchange is a diffusive process whose rate depends on the extant partial-pressure gradients and gas-specific exchange constants (20, 22, 23).

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and venous blood (VB), gas exchange across this path is primarily attributable to the relatively slow exchange of N₂, and, consequently, this exchange is expected to have a minimal effect on P_ME over most flight durations. In contrast, gas exchange across the ET is a rapid, gradient-dependent bolus exchange of mixed gases between NP and tympanum. Under normal physiological conditions, this is the only direct, potential communication between ME and ambient environment and the only exchange pathway capable of reducing established, positive Pam-P_ME gradients.

The functional anatomy of the ET has been described in many publications (2, 5, 17, 52). Briefly, the posterior portion of the ET is a mucosa-lined, bony tube continuous with the anterior tympanum, whereas the anterior portion is cartilaginous medially and membranous laterally (Fig. 2). The cartilaginous portion is usually closed by a tissue pressure, ET pressure (P_ET), that equals the sum of the Pam (a consequence of the incompressibility of body fluids) and a vascular pressure (P_VAS) (17, 27). A muscle, the tensor veli palatini (mTVP), takes origin from the membranous wall of the ET and terminates on the hamular process and within the palatine aponeurosis (5). Activation of the muscle during swallowing exerts an anterior-lateral-inferior vector force (F_TVP) on the membranous wall of the ET (52).

Figure 1B depicts these functional relationships. There, the ET is represented as a balloon pressure valve that is normally closed by the pressure difference between P_ET and both NP pressure (P_NP) and P_ME. ET opening can be affected by passive, pressure-driven processes or by active, pressure-driven or muscle-assisted mechanisms (36, 51). Passive, pressure-driven ET opening occurs when the force, F(P), associated with either P_ME or P_NP exceeds that of P_ET. Active, pressure-driven ET opening occurs when P_NP is increased by Valsalva or other maneuvers so that the applied F(P_NP) exceeds F(P_ET), or, for some individuals, when F(P_ET) is reduced by yawning and mandibular repositioning (5, 13, 17, 36). Active, muscle-assisted ET opening occurs when the mTVP contracts with sufficient force (F_TVP) to overcome the F(P_ET) (5, 27, 28). The teleological effect of these “normal” ET openings is to allow NP-ME gas exchange so as to maintain an approximate equilibrium between P_ME and Pam as P_ME is decreased by transmucosal gas exchange and Pam fluctuates with barometric conditions, i.e., normal P_ME regulation (5, 31, 43).

Movements of the TM in response to P_ME-Pam differentials are an important exception to the assumed fixed ME volume (V_ME). There, small fluctuations in that pressure gradient can be absorbed by V_ME changes in response to pressure-driven TM movements (46, 47). This is illustrated in Fig. 1C, which shows the TM response to a P_ME-P_Cabin gradient. As given by Boyle’s law, the magnitude of this pressure buffering effect is a function of the ratio of TM volume displacement to V_ME. In healthy ears, the maximum TM displacement volume is ~1% of the ME (i.e., tympanum + mastoid) volume (46), and the buffering effect of TM displacement on P_ME is limited. However, persistent ME disease causes a significantly reduced V_max and can cause a hypercompliant TM (19, 26), changes that will increase the determinate ratio for TM buffering and may reduce the affected ME’s susceptibility to barotrauma.

Normal P_ME Regulation During Flight

During airplane ascent, P_Cabin (= Pam) decreases, which causes decreasing P_M, P_ET, and MEM pressure (P_MEM), whereas P_ME is relatively unchanged (with the exception of a minor decrease associated with TM bulging) vis-à-vis takeoff. This results in the development of positive P_ME-Pam, P_ME-P_NP, and P_ME-P_ET gradients. At times when F(P_ME) exceeds F(P_ET), the ET passively opens, gas of ME composition flows from the ME to NP, and P_ME is reset to the extant value of P_ET. The residual P_ME-P_Cabin gradient representing P_C (i.e., P_ET – Pam), as well as any gradients that develop by trans-MEM gas exchange (Pam – P_ME – δP_ME, where δ is change) or by minor changes in elevation during flight (Pam ± δPam – P_ME)
are reduced by directional gas flows when the ET is actively opened by the mTVP.

On descent, Pam increases, causing increases in P_{NP}, P_{ET}, and P_{MEM}, whereas P_{ME} is relatively unchanged vis-à-vis cruising altitude. This causes a rapidly developing, positive Pam-P_{ME} (and P_{ET}-P_{ME}) gradient, and a relative P_{MEM} over-pressure with respect to P_{ME}. Under such conditions, neither F(P_{NP}) or F(P_{ME}) will exceed F(P_{ET}), and passive ET openings are not possible. Consequently, during descent, the passenger must periodically open the ET actively by swallowing to inducing mTVP activity or by other maneuvers that cause F(P_{NP}) to transiently exceed the extant F(P_{ET}) or cause F(P_{ET}) to decrease to less than F(P_{NP}). Of the latter, Valsalva is the most commonly used wherein air is forcibly expelled from the lungs while keeping the mouth closed and pinching the nose (6, 17, 33, 36, 45). This greatly increases the P_{NP} and can passively open the ET to allow for NP gas transfer to the ME.

Pathogenesis of Barotrauma

The rapid changes in P_{Cabin} (Pam) during airplane ascent and descent can overtax the P_{ME}-regulating system and provoke barotrauma. For passengers with excellent active ET opening function, P_{ME} regulation during flight is a nominal task, but for those with less efficient ET function, infants and children, and those with concurrent nasal inflammation caused by colds or allergy, the task may be impossible (6, 7, 33). If trans-ET gas flow does not reestablish a near zero P_{ME}-Pam gradient during descent, P_{ET} will exert its force over a larger collapsible section of the ET lumen, which can exceed the maximal force exerted by either the mTVP or active NP pressurization (17). This phenomenon, known as ET “locking,” occurs at an individual-specific P_{ME}-Pam gradient and effectively obstructs the ET to any further gas flow.

In the absence of adequate pressure regulation, the large P_{ME}-Pam gradients that develop during ascent and descent cause maximal extension of the TM with stretching and tearing of its structural elements. The TM can develop focal hemorrhages or local pocket formation and may perforate (17, 45). At submaximal extension, this is perceived as a feeling of “fullness” in the ear and at maximal extension as severe pain (16, 55). These are signs and symptoms of barotitis media. Alternatively, at a specified value of ~200–300 mmH_{2}O, the positive Pam-P_{ME} gradient that develops during descent will cause a larger P_{MEM}-P_{ME} gradient, resulting in MEM swelling, capillary dilatation, transudative leakage, and accumulation of fluid in the ME via “hydrops ex vacuo” (50). This set of signs presents as barotitis media.

An issue often faced by otolaryngologists is the assignment of individual patients to risk groups for barotrauma, i.e., which patients can fly safely and which should take precautions before air flight (54). Currently, such assignments are based on history, clinical observations, and, in some centers, ET function test. We believe that these assessments may not account for all influential factors that determine barotrauma risk. Here, we take a unique approach to addressing this issue by first formulating a mathematical model of P_{ME} regulation during flight based on the physiological considerations outlined above and then studying the effects on barotrauma risk of varying physiological parameters included within the model.

### Glossary

**Pressures**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>P_{ME}</td>
<td>Total ME pressure</td>
</tr>
<tr>
<td>P'_{ME}</td>
<td>ME partial pressure (O_{2}, CO_{2}, N_{2}, H_{2}O)</td>
</tr>
<tr>
<td>P_{Cabin}</td>
<td>Total cabin pressure</td>
</tr>
<tr>
<td>P'_{Cabin}</td>
<td>Cabin partial pressure (O_{2}, CO_{2}, N_{2}, H_{2}O)</td>
</tr>
<tr>
<td>P_{NP}</td>
<td>Total NP pressure</td>
</tr>
<tr>
<td>P'_{NP}</td>
<td>NP partial pressure (O_{2}, CO_{2}, N_{2}, H_{2}O)</td>
</tr>
<tr>
<td>P_{ET}</td>
<td>Tissue pressure surrounding ET lumen</td>
</tr>
<tr>
<td>P_{ET}</td>
<td>ET intraluminal pressure</td>
</tr>
<tr>
<td>Pam</td>
<td>Ambient pressure</td>
</tr>
<tr>
<td>P_{vas}</td>
<td>Vascular pressure</td>
</tr>
<tr>
<td>P_{MEM-Cabin}</td>
<td>ME-cabin pressure differential</td>
</tr>
<tr>
<td>P_{MEM-NP}</td>
<td>ME-NP pressure differential</td>
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**Volumes**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>V_{ME}</td>
<td>ME air space volume</td>
</tr>
<tr>
<td>V_{mas}</td>
<td>Mastoid volume</td>
</tr>
<tr>
<td>V_{NP}</td>
<td>Tympanum volume</td>
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</table>

**ET Passive Opening**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>P_{O'}</td>
<td>ET opening pressure (reference ambient)</td>
</tr>
<tr>
<td>P'_{ME-ET}</td>
<td>ME-side opening pressure (absolute)</td>
</tr>
<tr>
<td>P_{ME-ET}</td>
<td>ME-side opening pressure (reference ambient)</td>
</tr>
<tr>
<td>P_{NP-ET}</td>
<td>NP-side opening pressure (absolute)</td>
</tr>
<tr>
<td>P_{NP-ET}</td>
<td>ME-side opening pressure (reference ambient)</td>
</tr>
<tr>
<td>P_{ET}</td>
<td>ET closing pressure (reference ambient)</td>
</tr>
<tr>
<td>A_{ME'}</td>
<td>ET contact area from ME</td>
</tr>
<tr>
<td>A_{NP'}</td>
<td>ET contact area from NP</td>
</tr>
</tbody>
</table>

**TM Displacement**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_{TM}</td>
<td>TM cross-sectional area</td>
</tr>
<tr>
<td>C_{TM}</td>
<td>TM compliance</td>
</tr>
<tr>
<td>X_{TM}</td>
<td>TM linear displacement</td>
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</table>

**ET Active Opening**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>F_{ET}</td>
<td>ET “closing” force</td>
</tr>
<tr>
<td>F_{ST}</td>
<td>ET intraluminal surface tension (ST) force</td>
</tr>
<tr>
<td>F_{TVP}</td>
<td>Force exerted by mTVP on ET lumen</td>
</tr>
<tr>
<td>C_{ET}</td>
<td>ET compliance</td>
</tr>
<tr>
<td>X_{ET}</td>
<td>ET mediolateral lumen width</td>
</tr>
<tr>
<td>Q_{ET}</td>
<td>Trans-ET volume gas flow</td>
</tr>
<tr>
<td>R_{A}</td>
<td>ET active resistance</td>
</tr>
<tr>
<td>T_{A}</td>
<td>ET opening time</td>
</tr>
<tr>
<td>S_{1}</td>
<td>Swallowing frequency</td>
</tr>
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**Miscellaneous**

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<th>Description</th>
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<tbody>
<tr>
<td>k_{i}</td>
<td>Species-specific trans-MEM exchange time constants (O_{2}, CO_{2}, N_{2}, H_{2}O)</td>
</tr>
</tbody>
</table>

### METHODS

**Definition of Disease States**

We use the P_{ME}-P_{Cabin} gradient (ΔP_{ME-Cabin}) as an index measure of barotrauma, or
\[ \Delta P_{\text{ME-Cabin}}(t) = P_{\text{ME}}(t) - P_{\text{Cabin}}(t) \]  

(1)

where \( P_{\text{ME}} \) and \( P_{\text{Cabin}} \) are absolute pressures within the ME and cabin, respectively, at a time step \( t \). Based on the results of previous studies, we assigned \( \Delta P_{\text{ME-Cabin}} \leq -250 \text{ mmH}_{2}\text{O} \) as the threshold for onset of barotitis media \( (50) \) and \( \Delta P_{\text{ME-Cabin}} \geq 1,300 \text{ mmH}_{2}\text{O} \) as the threshold for onset of baromyringitis with severe pain \( (6) \).

**Gas Exchange Model**

The model compartments and linkages shown in Fig. 1A depict the gas-exchange components of the ME system. All compartments are assumed to be well mixed and isothermal with intercompartmental communication defined as the transfer of gas moles down pressure gradients along the linkages. Model compartments include the ME (tympanum + mastoid), MEM, NP, VB, and cabin. The ME is linked periodically to the NP during ET openings and continuously with the VB via the MEM. The cabin acts as the ambient environment for the system, directly affects \( P_{\text{ET}} \) and \( P_{\text{MEM}} \) [assumed to be nearly instantaneous] and linear, based on the results of pressure chamber experiments \( (30) \), exerts a mechanical force on the TM, and exchanges gas with the NP. The TM is assumed to be an infinite gas source/sink, and the volumes of the NP and VP are assumed to be finite but much greater than that of the ME. Consequently, species gas exchange between the ME and larger compartments does not affect the partial and total pressures of those compartments, but does have a significant effect on ME partial and total pressures.

**Cabin Pressurization**

During ascent, the airplane rises to a cruising altitude of \( \sim 30,000 \text{ ft} \) above sea level. To protect passengers from the adverse effects of these extreme low pressures, the cabin is pressurized to an effective cruising altitude of \( \sim 8,000 \text{ ft} \) \( (35, 45, 55) \). Cabin pressurization was modeled by increasing cabin altitude at a constant rate of 90 m/min (approximately that of a Boeing 747) from departure elevation to the effective cruising altitude \( (45) \). \( P_{\text{Cabin}} \) is a function of cabin elevation and, assuming ideal compressible gas behavior, is given by:

\[ P_{\text{Cabin}}^{\text{tot}}(t) = \text{Pam} \left( \frac{mgz(t)}{s} \right) \]

(2)

where \( P_{\text{Cabin}}^{\text{tot}} \) is total \( P_{\text{Cabin}} \), \( t \) is time, \( g \) is acceleration due to gravity, \( m \) is the average mass of an air molecule, \( B \) is Boltzman’s constant, \( T \) is the cabin temperature, \( \text{Pam} \) is referenced to sea level, and \( z(t) \) is the effective altitude of cabin pressurization (reference sea level). Because gas species mole fractions are constant during flight, cabin \( \text{N}_{2} \) \( (P_{\text{Cabin}}^{\text{N}_{2}}) \) and \( \text{O}_{2} \) partial pressures \( (P_{\text{Cabin}}^{\text{O}_{2}}) \) are calculated using:

\[ P_{\text{Cabin}}^{\text{N}_{2}}(t) = 0.79 P_{\text{Cabin}}^{\text{tot}}(t) \]

(3)

\[ P_{\text{Cabin}}^{\text{O}_{2}}(t) = 0.21 P_{\text{Cabin}}^{\text{tot}}(t) \]

(4)

Similarly, airplane descent was modeled as a linear decrease from effective cruising to destination altitudes at \( \sim 90 \text{ m/min} \), while calculating the increases in \( P_{\text{Cabin}} \).

**Pulmonary Exchange**

Total \( P_{\text{NP}} \) is assumed to be equal to that of the cabin or,

\[ P_{\text{NP}}(t) = P_{\text{Cabin}}(t) \]

(5)

while NP gas species pressures are assumed to be an average of the respective cabin (experienced during inhalation) and alveolar (experienced during exhalation) values \( (34) \). Total VB pressure \( (P_{\text{VB}}) \) is linked to total \( P_{\text{Cabin}} \) via nasopharyngeal-pulmonary gas exchange as, under the assumptions that the pulmonary-blood-gas exchange is very rapid and blood gases stored in fatty tissues would contribute minimally to the ME arterial supply. Throughout flight, VB partial pressures of \( \text{O}_{2} \) \( (P_{\text{VB}}^{\text{O}_{2}}) \) and \( \text{CO}_{2} \) \( (P_{\text{VB}}^{\text{CO}_{2}}) \) are assumed to be buffered at constant values by hemoglobin and bicarbonate reactions, the VB remains saturated at a constant \( \text{H}_{2}\text{O} \) pressure \( (P_{\text{VB}}^{\text{H}_{2}\text{O}}) \), and VB \( \text{N}_{2} \) pressure \( (P_{\text{VB}}^{\text{N}_{2}}) \) is a function of nasopharyngeal \( \text{N}_{2} \) pressure, calculated as:

\[ P_{\text{VB}}^{\text{N}_{2}}(t) = P_{\text{Cabin}}^{\text{N}_{2}}(t) - P_{\text{VB}}^{\text{O}_{2}}(t) - P_{\text{VB}}^{\text{CO}_{2}}(t) \]

(7)

**PME Dynamics During Flight**

The driving mechanisms included in the model that affect \( P_{\text{ME}} \) dynamics during flight are trans-ET and trans-MEM gas exchanges and the pressure effects of \( V_{\text{ME}} \) changes due to TM displacement.

**ET opening.** During ET openings, gas flows between the ME and NP in response to the total extant pressure gradient. The ET opens when a force applied to the ET lumen \( (F_{\text{ET}}) \) equals the ET closing force equal to the sum of the force of the mucosal tissue pressure \( (P_{\text{ET}} A_{\text{ET}}) \) and that attributable to intraluminal surface tension \( (F_{\text{ST}}) \) or:

\[ F_{\text{ET}}(t) = P_{\text{ET}}(t) A_{\text{ET}} + F_{\text{ST}} \]

(8)

where \( A_{\text{ET}} \) is the surface area of mucosal contact.

Pressure-driven ET opening occurs when \( P_{\text{ME}} \) (passive) or \( P_{\text{NP}} \) (active or passive) exerts a force \( (P_{\text{ME}} A_{\text{ME}} \text{ or } P_{\text{NP}} A_{\text{NP}}) \) on the ET lumen greater than \( F_{\text{ET}} \) such that,

\[ P_{\text{ME}}(t) > \frac{F_{\text{ET}}(t)}{A_{\text{ME}}} \]

(9)

where \( A_{\text{ME}} \text{ and } A_{\text{NP}} \text{ are the effective ET surface areas exposed to the ME and NP, respectively, and } P_{\text{ME-ET}} \text{ and } P_{\text{NP-ET}} \text{ are the ME and NP opening pressures of the ET, respectively. These opening pressures have been measured empirically and were reported as pressure differentials referenced to ambient \text{ i.e., } P_{\text{ME-ET}} = P_{\text{ME}} - P_{\text{ET}}(t) \text{ and } P_{\text{NP-ET}} = P_{\text{NP}}(t) - P_{\text{ET}}(t) \text{.} \}

We used representative values from those data sets in this model (see Table 1).

When relative ME overpressures cause the ET to passively open, gas exchange continues until the intraluminal air phase pressure of the ET \( (P_{\text{ET}}) \) equals the tissue pressure \( (P_{\text{ET}}) \text{ of the ET, where } P_{\text{ET}} = P_{\text{ME}} \text{. This results in a residual ME overpressure with respect to the NP}

Table 1. Average values of model parameters for “normal” MEs used in simulation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Mean</th>
<th>Units</th>
<th>Reference No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_{\text{ME}} )</td>
<td>ME volume</td>
<td>8.75</td>
<td>ml</td>
<td>14, 38</td>
</tr>
<tr>
<td>( \Delta V_{\text{TM}} )</td>
<td>TM displacement volume</td>
<td>0.025</td>
<td>ml</td>
<td>14, 46</td>
</tr>
<tr>
<td>( A_{\text{TM}} )</td>
<td>TM surface area</td>
<td>0.6</td>
<td>cm²</td>
<td>15</td>
</tr>
<tr>
<td>( s_{\text{TM}} )</td>
<td>TM stiffness coefficient</td>
<td>170</td>
<td>mmH_{2}O/ml</td>
<td>26</td>
</tr>
<tr>
<td>( R_{\text{A}} )</td>
<td>ET active resistance</td>
<td>2</td>
<td>mmHg·ml⁻¹·min⁻¹</td>
<td>11</td>
</tr>
<tr>
<td>( T_{\text{A}} )</td>
<td>ET active opening duration</td>
<td>0.25</td>
<td>s</td>
<td>11</td>
</tr>
<tr>
<td>( P_{\text{ME-ET}} )</td>
<td>ME opening pressure</td>
<td>350</td>
<td>mmH_{2}O</td>
<td>51</td>
</tr>
<tr>
<td>( P_{\text{NP-ET}} )</td>
<td>NP opening pressure</td>
<td>600</td>
<td>mmH_{2}O</td>
<td>13</td>
</tr>
<tr>
<td>( P_{\text{C}} )</td>
<td>Closing pressure</td>
<td>100</td>
<td>mmH_{2}O</td>
<td>51</td>
</tr>
</tbody>
</table>

ME, middle ear; TM, tympanic membrane; ET, eustachian tube; NP, nasopharynx.
(PCE) that is usually referred to as the ET closing pressure and can be written expressed as:

\[ P_{CE} = P_{ET}(t) - P_{ME}(t) = P_{gas} \]  

(10)

Because gas flows from ME to NP, ME species gas fractions are not affected by this transfer, and these were calculated by multiplying the preexisting gas fractions by the revised total PME. As with PME_{ET}, PCE has been measured empirically (51), and representative values are used in this model (see Table 1).

For ET openings caused by relative NP overpressures, gas exchange first occurs between NP and ME, wherein those pressures are equilibrated, and then between ME and NP as PME is reduced to the ET closing pressure. The effect of the NP-to-ME gas transfers on ME partial pressures at a time step (dt) was modeled as the weighted average of NP and ME species pressures as given by:

\[ \frac{dP_{ME}(t)}{dt} = \gamma_{NP}[P_{NP}(t) - P_{ME}(t)] \]  

(11)

where \( \gamma_{NP} \) is the species mole fraction in the NP, \( P_{NP} \) is total NP, and PME_{ME} is total PME. These partial pressures were then adjusted for the ME-to-NP gas exchange as described above.

Active muscle-assisted ET opening occurs when the force of mTVP contraction (F_{mTVP}) surpasses F_{ET}(t), where

\[ F_{mTVP}(t) > F_{ET}(t) \]  

(12)

For all F_{mTVP} satisfying this condition, the magnitude of that muscular force determines the ET lateral wall displacement as described by Hooke's law:

\[ X_{ET}(t) = F_{mTVP}(t)C_{ET} \]  

(13)

where \( C_{ET} \) is the compliance of the ET lumen, and \( X_{ET} \) is the lumen wall displacement distance. Figure 2 provides a detailed representation of the forces acting on the ET during mTVP activity. Assuming that trans-ET gas exchange follows Hagen-Poiseuille flow between two parallel plates (11), then

\[ Q_{ET}(t) = \frac{2}{5} \frac{\Delta P_{ME-NP}(t)X_{ET}(t)L}{\mu L} \]  

(14)

where \( Q_{ET} \) is the volume of gas transferred, L is the ET length, \( \mu \) is the viscosity of air, and \( \Delta P_{ME-NP} \) is the driving force for transfer. Because \( \mu \) and \( L \) are constants for a given ET and \( X_{ET} \) is a defined function of F_{mTVP}, we can extract from this equation an analytical expression for the active gas flow rate (R_{A}) that is conditioned on F_{mTVP}, or:

\[ R_{A}(t) = \frac{\Delta P_{ME-NP}(t)}{Q_{ET}(t)} = \frac{3}{5} \frac{\mu L}{W[F_{mTVP}(t)C_{ET}]} \]  

(15)

While F_{mTVP} is not measurable in vivo, \( R_{A} \) is an outcome measure of the forced-response test of ET function, which has been used in both clinical evaluations and experimental studies in humans (11, 12, 23, 51). In the model, \( R_{A} \) is an input parameter used to describe mTVP effectiveness with respect to active ET openings with representative values selected from existing data sets (see Table 1). Lacking measured values of F_{mTVP}, we did not include ET "locking" in the model description.

Using the empirical measures of \( R_{A} \) and ET opening time (T_{A}) reported by Cantekin and colleagues (10–12), trans-ET volume gas exchange can be then be described as follows:

\[ Q_{ET}(t) = \frac{\Delta P_{ME-NP}(t)T_{A}}{R_{A}} \]  

(16)

Regular tubal opening by mTVP activity occurs during rhythmic swallowing, as reported by Tideholm et al. (53). In this model, we used a normal swallowing frequency (S_{f}) of 5.2 openings/h during cruising and an increased value of 3.1 openings/h during descent.

Volume gas flow during mTVP-induced tubal openings (at time-step \( \delta t \)) represents the directional movement of a proportional number of gas moles (N) between compartments, with the relationship formalized as:

\[ \Delta N_{ET}(\delta t) = \frac{P_{ME}(\delta t)Q_{ET}(\delta t)}{K} \]  

(17)

where \( \Delta N_{ET} \) is the change in number of ET gas moles, and \( K \) is the product of ME temperature and the gas constant. Assuming an ideal gas, \( P_{ME} \) after the swallow is calculated from the sum of \( \Delta N_{ET}(\delta t) \) and the number of ME moles (N_{ME}(t)) before the swallow. This value is then used to calculate a new V_{ME}, V_{ME}(t + \delta t), and P_{ME}, P_{ME}(t + \delta t) (see TM displacement section below). The effect of these transfers on ME gas species pressures was modeled as described above for the directional transfers caused by passive ET openings.

\textit{MEM gas exchange.} The ME exchanges gas with the local VB by diffusion across the MEM. Here, the MEM was modeled as the VB source/sink for this exchange, such that ME gas species pressures, \( P_{ME} \), are calculated as

\[ \frac{dP_{ME}(t)}{dt} = k_{i}[P_{ME}(t) - P_{VB}(t)] \]  

(18)

where \( k_{i} \) is an empirical species exchange constant, and \( P_{VB} \) is VB species pressure. Equation 18 was applied for N_{2}, O_{2}, CO_{2}, and H_{2}O, and total PME was equal to the summation:

\[ P_{MME}(t) = \sum P_{ME}(t) \]  

(19)

Table 2 lists the initial gas-species pressures for these compartments and the trans-MEM time constants measured by experiment (22). The resultant P_{ME}(t + \delta t) value following trans-MEM exchange is calculated after the V_{ME}, V_{ME}(t + \delta t), is adjusted for \( \Delta V(t + \delta t) \) (see TM displacement section below).

\textit{TM displacement.} Figure 1C illustrates TM displacements in response to a pressure gradient across the membrane, \( \Delta P_{ME-cabin} \). TM deformation is a function of its compliance and the force applied to the TM (equal to trans-TM pressure gradient multiplied by TM surface area). The deformation is governed by Hooke’s law:

\[ X_{TM}(t + \delta t) = \Delta P_{ME-cabin}(t + \delta t)A_{TM}C_{TM} \]  

(20)

where \( X_{TM} \) is the TM displacement distance, \( A_{TM} \) is the TM surface area, and \( C_{TM} \) is the TM compliance. TM volume displacement (\( \Delta V_{TM} \)) is calculated as:

\[ \Delta V_{TM} = X_{TM}(t + \delta t)A_{TM} \]  

(21)

with displacements constrained to the range,

\[ -\Delta V_{TM}^{\text{max}} < \Delta V_{TM}(t + \delta t) < \Delta V_{TM}^{\text{max}} \]  

(22)

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|}
\hline
\textbf{Gas Species} & \textbf{ME} & \textbf{NP} & \textbf{VB} \\
\hline
\textbf{Partial Pressure, mmHg} & \textbf{Partial Pressure, mmHg} & \textbf{Partial Pressure, mmHg} & \textbf{Partial Pressure, mmHg} & \\
\hline
\textbf{Oxygen} & 40 & 112 & 45 & 0.0008 \\
\textbf{Carbon dioxide} & 46 & 32 & 46 & 0.16 \\
\textbf{Water vapor} & 47 & 47 & 47 & 0.32 \\
\textbf{Nitrogen} & Balance & Balance & Balance & 0.00008 \\
\hline
\end{tabular}
\caption{Initial gas species partial pressure in the ME (24), NP (34), VB, and trans-ME mucosal exchange constants (21).}
\end{table}
compartment ($P_{\text{cabin}}$, $P_{\text{NP}}$, and $P_{\text{VB}}$); gas species pressures and total pressure ($P_{\text{ME}}$ adjusted for $\Delta V_{\text{TM}}$) for the ME after trans-MEM exchange; and gas species pressures and total pressure for the ME after conditional gas transfers through the ET based on inputted swallowing rhythm ($Q_{\text{ET}}$ adjusted for $\Delta V_{\text{TM}}$) and/or passive openings ($P_{\text{ME}}$ adjusted for $\Delta V_{\text{TM}}$).

**RESULTS**

**Model Validation**

To evaluate the predictive accuracy of the model, we simulated the $P_{\text{ME}}$ dynamics for a pressure chamber experiment by Groth and colleagues (29), who described $P_{\text{ME}}$ change (measured as TM volume displacements) in pilots exposed to high rates of pressurization (1,920 ft/min) over short time periods (25 s). Model parameters were estimated from the experimental data ($P^{\text{ET}} = 292$ mmHg, $P^{\text{C}} = 136$ mmHg, $R_A = 7.5$ mmHg·ml$^{-1}$·min$^{-1}$, $C_{\text{TM}} = 425$ mmHg/ml, $T_A = 250$ ms, and $S_1 = 33$ swallows/min). A comparison of model and experimental results is shown in Fig. 3. During ascent, $P_{\text{ME}}$ decreased, and the resulting ME overpressures caused outward TM displacement. At a relative ME overpressure of 292 mmHg, the ET passively opened, and the $P_{\text{ME}}$-Pam gradient was partly dissipated as gas was transferred from ME to NP, a process interrupted when the ET passively closed at $P_{\text{ME}} = P_{\text{ET}}$. This was associated with TM repositioning to a lesser volume displacement. During simulated descent, $P_{\text{ME}}$ increased, causing inward displacement of the TM. At all times, $P_{\text{ET}}$ exceeded $P_{\text{ME}}$ and $P_{\text{NP}}$, and passive ET openings did not occur. Rather, at semiregular intervals, swelling caused mTVP contraction and active ET openings. Each opening was associated with a transfer of gas from NP to ME, a consequent reduction in the $P_{\text{ME}}$-Pam gradient and reduced TM volume displacement. Sequential swallows caused a progressive lessening of the residual ME underpressure. This comparison shows that our model can accurately reproduce experimental data for $P_{\text{ME}}$ behavior during simulated flights.

**Flight Simulations**

Figure 4 shows $P_{\text{cabin}}$ as a function of time during three simulated 170-min “flights,” each departing from Pittsburgh, PA (PIT) and arriving at PIT, Denver, CO (DEN), and Miami, FL (MIA). For all “flights,” $P_{\text{cabin}}$ decreased during airplane ascent, remained relatively constant during cruising, and in-
creased on descent. The magnitude of pressure change experienced by passengers depends on the relative pressure differences between departure, cruising, and destination elevations. Table 3 lists the elevation and Pam values for these airports and for the airplane cabin at the effective cruising altitude. Using these three flight paths, we simulated the $P_{ME}$ dynamics for a “normal” ME (see Table 1) and for ears with “abnormal” structural (e.g., $V_{ME}$, TM displacement) or functional (e.g., $P_{ME-ET}$, $R_A$) parameters.

ET function measurements in “normal,” disease-free ears document passive ET openings at moderate ME-ambient overpressures (300–500 mmH$_2$O), passive ET closing at above-ambient $P_{ME}$ (100–200 mmH$_2$O), and the ability of the mTVP to open the ET over a large range of applied $P_{ME}$-Pam gradients (12). The $P_{ME}$ changes during a simulated flight for such an ear (all parameters equal to normal) are shown in Fig. 5A, which demonstrates the development of relatively low-magnitude $P_{ME}$-Pam gradients throughout the duration of the flight, with none of those gradients exceeding the threshold for either expression of ME barotrauma.

A common treatment for otitis media is the insertion of tympanostomy tubes, small tubes placed within the TM that allow for constant communication between ME and ambient environment (42). An abnormal physiological condition referred to as a “patulous” ET also allows for constant NP-ME communication (4). There, function tests show that the $P_{ET}$ is less than Pam, resulting in a continuously open ET (5). Simulated flights for ears with either of these conditions yield the trivial result of $2,020$ mmH$_2$O $P_{ME}$-Pam gradient throughout flight and, consequently, protection from barotrauma.

Rarely, clinical tests document an ET that is physically obstructed by enlarged adenoids or by nasopharyngeal carcinomaa (40, 44). More frequently, the ET is intrinsically blocked by intraluminal swelling and venous engorgement caused by posterior extension of NP inflammation that accompanies viral infections or allergy (5). ET function tests for both conditions document a failure of applied ME overpressures to passively open the ET and an inability of the mTVP to affect ME-NP gas transfers (5). We modeled this condition by inputting high $P^O$ values ($P^O_{ME-ET} = 2,500$ mmH$_2$O, $P^O_{NP-ET} > 2,500$ mmH$_2$O) and a high $R_A$ ($1/R_A \approx 0$) value (other parameters equal normal). The results for the three simulated flights are shown in Fig. 5B. During ascent, the lack of passive ET openings leads to a positive ME-cabin gradient of 2,020 mmH$_2$O, a pressure that exceeds the threshold for pain and barommyringitis. During cruising, that gradient is slightly reduced by the slow, trans-MEM N$_2$ exchange, and, during descent, the gradient is decreased as $P_{Cabin}$ increases. On landing, the ME-cabin gradient [terminal pressure gradient (TPG)] depends almost exclusively on the difference in elevation between departure and arrival; the TPG for a flight departing and arriving at PIT was $-202$ mmH$_2$O, for a flight arriving in DEN was $1,070$ mmH$_2$O, and for a flight arriving in MIA was $-612$ mmH$_2$O. Only the MIA destination was associated with the expression of barotitis media.

The most common cause of ET dysfunction is a constitutively impaired, active ET opening mechanism. There, function tests document “normal” passive ET opening and closing pressures, but an inability of the mTVP muscle to dilate the ET during swallowing (5). To model these ears, we inputted normal values for the opening and closing pressures (and other variables) but constrained the activity of the mTVP muscle by inputting a high $R_A$ value ($1/R_A \approx 0$). Note that $1/R_A$ is the airflow conductance of the ET during a swallow (i.e., the extent to which the ET dilates during mTVP contraction) and does not necessarily reflect the airflow conductance resulting from applied pressure differentials or the other passive properties of the ET. Figure 5C shows the dynamics of the $P_{ME}$-$P_{Cabin}$ gradient for the three simulated flights. During ascent, the developing positive $P_{ME}$-$P_{Cabin}$ gradient is repeatedly reduced to the value of $P^C$ as the ET is passively opened at $P^O$. No barotrauma is experienced during this phase of flight.

Table 3. Elevations and ambient pressures for airports and airplane cabin (35)

<table>
<thead>
<tr>
<th>Location</th>
<th>Elevation, ft</th>
<th>Ambient Pressure, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pittsburgh, PA</td>
<td>1,204</td>
<td>730</td>
</tr>
<tr>
<td>Miami, FL</td>
<td>8</td>
<td>760</td>
</tr>
<tr>
<td>Denver, CO</td>
<td>5,431</td>
<td>630</td>
</tr>
<tr>
<td>London, UK</td>
<td>80</td>
<td>758</td>
</tr>
<tr>
<td>Airplane cabin</td>
<td>8,000 (equivalent elevation)</td>
<td>577</td>
</tr>
</tbody>
</table>

Fig. 5. Predicted change in middle ear-cabin pressure gradient ($\Delta P_{ME-Cabin}$) for a “normal” ME with parameters listed in Table 2 (A), for a ME with an obstructed ET ($P^O_{ME-ET} = 2,500$, $P^O_{NP-ET} > 2,500$ mmH$_2$O) (B), and for a ME with poor mTVP function ($1/R_A \approx 0$) (C). Barotrauma onset is specified by the dotted-dashed indicator lines.
residual gradient ($\Delta P_{\text{ME-Cabin}} = P^C$) is slowly reduced during flight by trans-MEM N2 exchange. However, the developing negative ME-cabin gradient during descent cannot be alleviated by muscle-assisted ET openings, leading to TPGs of $-1,731$, $-2,226$, and $-486$ mmHg for landings at PIT, MIA, and DEN, respectively. All underpressures are of sufficient magnitude to provoke barotitis media, and the former two are expected to provoke baromyringitis.

The results of this simulation are not applicable to ears that test positive for the Valsalva maneuver, wherein large PNP gradients are generated by closed nose/mouth forced exhalations. If the generated PNP-PET gradient is sufficient to passively open the ET, NP gas is transferred to the ME, and the former two are expected to provoke barotitis media, and the former two are expected to provoke baromyringitis.

The majority of persons who fly do not exhibit these extreme forms of ET dysfunction but rather exhibit a graded series of active ET opening efficiencies. For example, studies comparing children with adults or persons with and without a history of otitis media document similar passive ET properties among all groups, but less efficient active ET openings in the former groups (5, 8, 12). In our model, this variability in active opening efficiency can be represented by varying $R_A$. Figure 6A shows the simulated $P_{\text{ME}}-P_{\text{Cabin}}$ gradient during the course of a PIT-MIA flight for an ear with normal and one with compromised mTVP-induced ET openings ($R_A = 2$ and 20 mmHg·ml$^{-1}$·min$^{-1}$; other parameters = “normal” values). The larger $R_A$ value limits trans-ET flow at each opening, compromises the ability of the ET to regulate $P_{\text{ME}}$, and leads to a negative TPG sufficient to precipitate barotitis on landing.

$V_{\text{ME}}$ shows a growth-related increase (attributable primarily to expansion of $\text{V}_{\text{mas}}$), an effect that is stunted or delayed in ears with poor ET function and/or a history of otitis media (14, 37, 38). This observation causally links poor ET function to both $V_{\text{ME}}$ values. There, low $R_A$ ($V_{\text{ME}}$ = 4.4 ml) buffers the effect of the compromised mTVP function on $P_{\text{ME}}-P_{\text{Cabin}}$ deviations and prevented the barotitis documented for the larger $V_{\text{ME}}$ ($V_{\text{ME}}$ = 13.1 ml).

From these observations, the ability to maintain a near-0 mmHg $P_{\text{ME}}-P_{\text{Cabin}}$ gradient depends on the relative magnitudes of volume gas supply and demand. In the absence of active, pressure-driven ET openings (e.g., Valsalva maneuver), supply is a function of mTVP ET opening efficiency (proportional to $S_f/T_{\text{TA}}/R_A$), while demand is a function of both the difference in $P_{\text{Cabin}}$ at effective cruising and landing altitudes (maximum $\Delta P$ to be equilibrated) and $V_{\text{ME}}$ (moles of gas required to equilibrate that $\Delta P$). Figure 7A summarizes this relationship for simulated PIT-MIA flights by plotting the TPGs for ears with constant $S_f$ and $T_{\text{TA}}$ but different $R_A$ and $V_{\text{ME}}$ values. There, low $R_A$ ($\leq$4 mmHg·ml$^{-1}$·min$^{-1}$) allows for the exchange of sufficient gas volumes to prevent both expressions of barotrauma over all reasonable $V_{\text{ME}}$ (<16 ml).
In contrast, buffering against barotrauma for increasing $V_{\text{ME}}$ was decreased with increasing values of $R_A$.

In ears with a history of disease, changes in the TM are observed frequently (16). These include increases in TM compliance, which is termed atelectasis. In the extreme, TM retraction can displace the total $V_{\text{sym}}$, resulting in a $V_{\text{ME}}$ restricted to that of the mastoid. As noted, the magnitude of $P_{\text{MIE}}$-Pam deviations in ears with compromised mTVP function can be buffered by TM displacement volume (see Eq. 24). Figure 7B shows the simulated TPG values for a ME with compromised mTVP function ($R_A = 8$, other parameter values = "normal") as a function of both TM stiffness ($k_{\text{TM}} = 1/C_{\text{TM}}$) and $V_{\text{ME}}$ (with $\Delta V_{\text{TM}} \leq V_{\text{sym}} = 1$ ml) after a PIT-MIA flight. The plot demonstrates the expected effect of changing the $\Delta V_{\text{TM}}$-to-$V_{\text{ME}}$ ratio on $P_{\text{MIE}}$-Pcabin gradients. Specifically, greater $k_{\text{TM}}$ values are associated with lesser TPG values, and the magnitude of this effect is greater for larger $V_{\text{ME}}$. Conversely, hypercompliant TMs ($k_{\text{TM}} < 0.14$ “normal” $k_{\text{TM}}$) protected the ME from barotitis media over all reasonable $V_{\text{ME}}$.

Finally, we examined the effect of flight duration on TPG by comparing the predicted TPG values for PIT-MIA (170 min) and PIT to London, UK (533 min), destinations with similar elevations (Table 3). For all ME function and structure configurations, the TPGs for the two flights were similar. Because the major difference between these flights is the duration of cruising at fixed altitude, any effect of flight duration will be driven by the rate of trans-MEM N$_2$ exchange, a process that was previously measured to be extremely slow (20, 22).

DISCUSSION

Unlike previous descriptions that focused only on a categorical representation of ET function (poor/good ET opening), our model of $P_{\text{MIE}}$ regulation during flight is founded on mathematical descriptions of the physiology underlying gas transfers between the ME and all adjacent compartments. Calibration of the model parameters was done using published data for disease-free MEs, and thus this description is not applicable to the ME with extant otitis media or MEM inflammation. Specifically, those conditions 1) introduce additional system compartments (e.g., effusion), 2) change the capacitances of existing compartments (e.g., increase MEM volume at the expense of $V_{\text{ME}}$), and 3) affect the exchange parameters for trans-MEM gas transfers (e.g., increase MEM blood flow) (1). Nonetheless, our model does have broad applicability to the “disease-free” ME and to MEs expressing the predispositions (e.g., poor mTVP function) and/or sequelae (e.g., altered TM compliance, reduced $V_{\text{max}}$) of those conditions. Moreover, by including continuous measures of relevant parameters, our model realistically maps disease expression onto the known continuum of underlying ET dysfunctions.

An important test of any model is its predictive accuracy with respect to describing and explaining well-established observations. For ME barotrauma, these include the previously documented increased risk associated with young age and nasal inflammation (concurrent colds or nasal allergy). Our model is capable of representing and explaining these effects by incorporating the changes in the contributing parameters measured by experiment. For example, the age effect is explicable by the established improvement in mTVP functional efficiency (modeled as progressively decreasing $R_A$) with advancing age (8, 9), and the effect of nasal inflammation is mediated by intraluminal venous engorgement (modeled as a greater $P_{\text{ET}}$) (17). These explanatory analyses can be extended to include the effects of preventative treatments, such as nasal decongestants (17, 39), that act by decreasing tissue inflammation (decreased $P_{\text{ET}}$) or of less well-established interventions, such as bottle-feeding of infants during descent (7), where the associated jaw movements initiate mTVP activity (greater $S_T$) and/or reduce ET tissue pressure (lesser $P_{\text{ET}}$).

Earlier descriptions of barotrauma during airflight usually did not discriminate between barotitis media and baromyringitis in reporting results. As discussed above, these expressions have different underlying causes with the former, resulting from a moderate, positive MEM-$P_{\text{ME}}$ gradient, and the latter resulting from large positive or negative MEM-$P_{\text{Cabin}}$ gradients. Consequently, baromyringitis can be experienced throughout flight and is usually associated with signs of TM damage and symptoms of ear-fullness and pain, but barotitis media develops during descent and, in the absence of baromyringitis, is often unrecognized by the traveler. By considering both expressions, our model predicts postflight ME barotrauma that is and is not perceived by the traveler and, by consequence, recorded as an event in the compilation of prevalence reports (49).

Perhaps the most important feature of our model is the demonstration of potential buffering mechanisms that modify or prevent disease expression in ears with constitutively or situationally impaired ET function. For example, we showed that, for ears with a blocked ET (by enlarged adenoids, nasopharyngeal carcinoma, nasal inflammation due to a cold/allergy, or other conditions), high positive pressures and baromyringitis will develop on ascent to cruising altitude for all flights, but the development of barotitis media on descent will depend on the difference between departure and destination altitudes. Likewise, for ears with poor ET function, a protective effect is provided by high TM volume displacement-to-$V_{\text{ME}}$ ratio. Support for the physiological relevance of these buffering mechanisms was provided in a recent paper (48) that reported a low frequency of barotrauma in ears that were expected to have poor ET function but also had preexisting conditions that favored a hypermobile TM and small (mastoid) $V_{\text{ME}}$.

Earlier descriptions of the pathogenesis of barotrauma focused primarily on ET function and did not include these nuances. In that regard, tests of ET function were used to screen candidates for service as pilots (29), and attempts have been made by industry to extend these tests to the professional flight crews of commercial airlines. Our results suggest that, while good ET function is highly predictive of disease-free flight, poor function only defines an increased risk of flight-induced barotrauma. This distinction has important implications to interpreting the results of ET function screening where failure to repeatedly open the ET during swallowing or to transfer NP gas to the ME during Valsalva can be career limiting.

In conclusion, we present a physiological model of barotrauma development for “normal” MEs during flight. The presented model simulates the empirical data for experiments conducted on pilots in a pressure chamber and explains past observations with respect to risk assessments. Also, our results identified diverse physiological and anatomical parameters that interact in affecting adequate and abnormal $P_{\text{MIE}}$ regulation during flight. This underscores the importance of considering contextual relationships in predicting the susceptibility of a given ME to barotrauma.
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