Inspiratory flow in the nose: a model coupling flow and vasoerectile tissue distensibility

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Physiopathologie et Thérapeutique Respiratoires INSERM UMR 492, and Service d’ORL et de Chirurgie Cervico-Faciale, Centre Hospitalier Inter-Communal de Créteil, Créteil; Centre de Recherche Claude Delorme, Air Liquide, Jouy en Josas; and Service de Physiologie-Explorations Fonctionnelles, Hôpital Henri Mondor Assistance Publique Hôpitaux de Paris, Créteil, France

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Fodil, Redouane, Lydia Brugel-Ribière, Céline Croce, Gabrielle Sbirlea-Apiou, Christian Larger, Jean-François Papon, Christophe Delclaux, André Coste, Daniel Isabey, and Bruno Louis. Inspiratory flow in the nose: a model coupling flow and vasoerectile tissue distensibility. J Appl Physiol 98: 288–295, 2005. First published August 27, 2004; doi:10.1152/japplphysiol.00625.2004.—We have developed a discrete multisegmental model describing the coupling between inspiratory flow and nasal wall distensibility. This model is composed of 14 individualized compliant elements, each with its own relationship between cross-sectional area and transmural pressure. Conceptually, this model is based on flow limitation induced by the narrowing of duct due to collapsing pressure. For a given inspiratory pressure and for a given compliance distribution, this model predicts the area profile and inspiratory flow. Acoustic rhinometry and posterior rhinomanometry were used to determine the initial geometric area and mechanical characteristics of each element. The proposed model, used under steady-state conditions, is able to simulate the pressure-flow relationship observed in vivo under normal conditions (4 subjects) and under pathological conditions (4 vasomotor rhinitis and 3 valve syndrome subjects). Our results suggest that nasal wall compliance is an essential parameter to understand the nasal inspiratory flow limitation phenomenon and the associated increase of resistance that is well known to physiologists. By predicting the functional pressure-flow relationship, this model could be a useful tool for the clinician to evaluate the potential effects of treatments.

acoustic rhinometry; nasal physiology; nasal wall compliance; fluid-structure coupling

THE INSPIRATORY FLOW LIMITATION PHENOMENON in the nose has been reported by clinicians since the 1970s (1, 18). This phenomenon can occur during hyperventilation in normal subjects and at rest in pathological situations. This phenomenon has been previously explained by collapse of the nasal valve due to wall compliance, which induces an increase in nasal resistance associated with marked nasal obstruction. To our knowledge, this phenomenon has not been studied from a mechanical point of view, as the majority of physical models and numerical models of the biomechanical literature describing pressure-flow relationships in the nose have used rigid wall properties. However, rigid wall properties are unsuitable to describe flow limitation.

In this study, we developed a discrete multisegmental model describing the coupling between flow and nasal wall distensibility that can occur in any part of the nose. The concept behind

this model was initially developed to describe flow-structure coupling in pharyngeal airways (8).

This model was developed by using acoustic rhinometry, posterior rhinomanometry, and pressure-drop measurement in a plastinated nose model. Acoustic rhinometry is a noninvasive method providing measurements of the nasal cross-sectional area and nasal volume as a function of the axial distance along the nasal passage (9). Associated with negative pressure applied to the nostril, this method has been shown (2, 14) to be a sensitive method to describe the mechanical properties of the various structures of the anterior part of the nasal cavities (i.e., nasal valve, anterior and medial parts of the inferior turbinate, and the middle meatus region). Posterior rhinomanometry provides an in vivo measurement of nasal resistance (3–5). The plastination technique consists of preserving the geometry of an anatomic specimen (in this case, a cadaver nasal cavity) by substituting polymers (silicone) for water and lipids (6), which has the advantage of accurately reproducing the complexity of nasal geometry.

MATERIALS AND METHODS

Nose area and compliance (wall state law). The compliances of the wall of the nasal cavity were estimated from measurement of longitudinal area profiles of the nasal cavity, A(x), obtained by the two-microphone acoustic reflection method (15, 16), when different steady pressures were applied to the distal end of the wave tube connected to the nostril, i.e., atmospheric pressure (Patm), Patm – 2, Patm – 4, Patm – 6, Patm – 8, and Patm – 10 cmH2O (2). Briefly, the device consisted of two microphones (piezoresistive pressure transducers 8510-B; Endevco France, Le Pré Saint-Gervais, France) and a horn driver mounted on a wave tube (inner diameter of 1.2 cm and overall length of 22 cm) connected at one end to the subject’s nostril with a nosepiece, allowing tight closure of the nasal aperture without deformation of the nasal valve. The other end of the wave tube was connected to a steady negative pressure generator. An acoustic wave was generated by the horn driver, and the resulting pressures were recorded by the two microphones. These digitized data were analyzed to obtain the cross-sectional area of the nasal airway as a function of the distance along the longitudinal axis, with a spatial step increment (ΔL) of ≈0.41 cm. Compliance per unit length was defined for each segment as the ratio between the variation of area (ΔA) and the variation of steady pressure (ΔP) applied to the nasal cavity. Compliance (C) was computed as the slope of the line (ΔA = C·ΔP) by fitting the set of data using a least squares error method.

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In vivo nasal resistance. Posterior rhinomanometry was used to determine nasal resistance. Briefly, while the subject was breathing through the nose, flow measurements were performed using a transparent nasal face mask fitted with a Fleisch no. 1 pneumotachograph (Lausanne, Switzerland) connected to a pressure transducer (Validyne MP 45, Northridge, CA; ±2 cm H2O). Oropharyngeal pressure was recorded via a catheter inserted through a hole drilled in a stopcock obstructing the cylindrical part of a modified mouthpiece placed between the lower lip and the protruding tongue. One port of a differential pressure transducer (Validyne MP 45; ±14 cm H2O) was connected to the catheter of the mouthpiece, whereas the second port was connected to the nasal mask to allow transnasal pressure measurement. Nasal resistance was defined as the ratio between transnasal pressure and flow when the transnasal pressure reached 1 cm H2O.

In vitro pressure drop measurement. Flow and pressure measurements in a plastinated nose model were performed by using transducers located at the nasopharyngeal extremity of the physical nasal model. The pneumotachograph was connected to a pressure transducer (Validyne MP 45; ±2 cm H2O). A differential pressure transducer (Validyne MP 45; ±14 cm H2O) was used to measure the pressure drop between the two ends of the plastinated nose model. In accordance with the local ethical committee guidelines, informed consent was obtained for all measured subjects after full information on the experiments was disclosed.

Model. The proposed model is a discrete multicomponent model including 14 compliant elements in series (see Fig. 1). Each element represents an individual segment of the anterior part of the nose. Each element is independent of the other elements, with its own length representing an individual segment of the anterior part of the nose. Each element includes 14 compliant elements in series (see Fig. 1). Each element corresponding to the upstream segment representing the nostril extremity connected to the atmospheric pressure (P atm), whereas element 14 corresponds to the downstream segment. $V$, flow.

Energy. The term $\Delta P_n$ depends on the velocity profile via the kinetic energy coefficient $\alpha_n$, where $\alpha_n = \int\int_A [u/(V/A_n)]^2 1/A_n dA_n$, where $u$ is the velocity and $V$ is flow.

Under steady-state conditions, i.e., for a given steady pressure variation between the two ends of the model ($\Delta P$), numerical determination of flow throughout the model and the cross-sectional areas ($A_n$) may be numerically inferred from the 14 couples of Eqs. 1 and 2 written for each segment, in which the compliance ($C_n$), the viscous dissipation ($\Delta P_v$), and the kinetic energy coefficient ($\alpha_n$) are known. This numerical resolution uses a dichotomy procedure similar to that described by Fodil et al. (8). This resolution was implemented in a FORTRAN language program and computed on a personal computer.

Evaluation of parameters of the model. The length of each model segment was chosen according to the available acoustic spatial step increment $\Delta L$. The simulation was also limited to the anterior part of the model, i.e., from the nostril to the end of the meatus region, due to artifacts generated by the paranasal sinuses when acoustic rhinometry was performed (10). This limitation defined the total length of our model and indirectly the number of segments. Fortunately for our model, it has already been shown that the anterior part of the normal nasal cavity is responsible for the majority of nasal resistance (11). As a first approximation, we can, therefore, consider that a model of the anterior part of the nose takes into account the effects of the entire normal nasal cavity in terms of the pressure-flow relationship. The initial area and the segment wall state law of each element were computed as described above from acoustic data. Resolution of the couples of Eqs. 1 and 2 required determination of the pressure-drop laws (viscous and kinetic) that depend, in each segment, on both the cross-sectional area pattern and the velocity profile in the cross-sectional area considered. To determine the pressure drop due to changes in kinetic energy, we arbitrarily fixed the value of the kinetic energy coefficient at 2. The viscous pressure-drop law was deduced from a Moody diagram derived from the measurement performed in the plastinated nose model. In this diagram, we fitted the relationship between nondimensional pressure drop and the Reynolds number by the following equations.

\[
\Delta P_r/(1/2\rho u^2) = 1.39(Re)^{-1.57} \text{ if } Re < 390
\]

\[
\Delta P_r/(1/2\rho u^2) = 0.130(Re)^{-0.19} \text{ if } Re > 390
\]

where $u$ is the mean velocity and $Re$ is the Reynolds number calculated from the effective diameter corresponding to the minimal cross-sectional area of the plastinated model measured by acoustic rhinometry. The aim of these equations was simply to compute a realistic nondimensional relationship between flow and pressure drop, despite the fact that no real physical significance can be attributed to the parameters of these fitting equations. To determine the viscous pressure drop in any segment of our model, we used these fitted equations into which the same multiplication coefficient was introduced for each segment and which were adjusted for each subject to obtain the resistance obtained by rhinomanometry, i.e., resistance for a pressure drop of 1 cm H2O.

RESULTS

We simulated the pressure-flow curve in 11 subjects: 4 “healthy” subjects and two “pathological” conditions with 4 vasomotor rhinitis subjects and 3 valve syndrome subjects. Table 1 lists the main clinical and mechanical features for each subject. In this table, to facilitate interpretation of compliance, we have divided the nasal cavity into three distinct physiological regions, as previously described by our group (2). We also computed the closing pressure defined as the pressure where the area of the region is zero: $Pc = -A(P = P_{atm})/C$, where $Pc$...
is closing pressure, A is cross-sectional area of the region at atmospheric pressure, and C is compliance. From a mechanical point of view, the patients with valve syndrome were characterized by higher compliances and lower closing pressures than healthy subjects. To a certain extent, the same tendency was observed for compliance and closing pressure of inferior and middle meatus regions between healthy and vasomotor rhinitis subjects.

Our simulations were inferred in two ways. First, the model was resolved with the compliance values obtained by acoustic rhinometry. In the second case, the model was computed with rigid wall conditions (all parameters of the model remained unchanged, except for compliance values that were set at zero). These simulations (Figs. 2–4) were compared with the pressure-flow measurement obtained during rhinomanometry. For all subjects, we found a good agreement between the results of the compliant model simulation and the measurement data, at least while the pressure variation between the two ends of the nose increased (increased negative pressure in the figure). The pressure-flow curves recorded by rhinomanometry sometimes served in pathological subjects. The rigid model tends to underestimate nostril resistance (Fig. 2). The simulation results obtained with the rigid model are clearly unable to describe the flow limitation observed in pathological subjects.

A physiological and mechanical characteristic of the subjects

Table 1. Biometric and mechanical characteristics of the subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age/Gender</th>
<th>Weight, kg</th>
<th>BMI, kg/m²</th>
<th>Valve region</th>
<th>Inferior turbinate region</th>
<th>Middle meatus region</th>
<th>Valve region</th>
<th>Inferior turbinate region</th>
<th>Middle meatus region</th>
</tr>
</thead>
<tbody>
<tr>
<td>H1</td>
<td>47/M</td>
<td>72</td>
<td>22.2</td>
<td>0.04</td>
<td>0.04</td>
<td>0.05</td>
<td>-26</td>
<td>-29</td>
<td>-37</td>
</tr>
<tr>
<td>H2</td>
<td>42/M</td>
<td>63</td>
<td>21.5</td>
<td>0.01</td>
<td>0.01</td>
<td>0.03</td>
<td>-53</td>
<td>-64</td>
<td>-56</td>
</tr>
<tr>
<td>H3</td>
<td>29/F</td>
<td>59</td>
<td>20.4</td>
<td>0.03</td>
<td>0.04</td>
<td>0.06</td>
<td>-19</td>
<td>-23</td>
<td>-23</td>
</tr>
<tr>
<td>H4</td>
<td>25/F</td>
<td>55</td>
<td>24.4</td>
<td>0.01</td>
<td>0.02</td>
<td>0.03</td>
<td>-53</td>
<td>-38</td>
<td>-59</td>
</tr>
<tr>
<td>R1</td>
<td>43/M</td>
<td>85</td>
<td>25.4</td>
<td>0.04</td>
<td>0.12</td>
<td>0.24</td>
<td>-22</td>
<td>-11</td>
<td>-10</td>
</tr>
<tr>
<td>R2</td>
<td>39/F</td>
<td>74</td>
<td>27.9</td>
<td>0.03</td>
<td>0.06</td>
<td>0.07</td>
<td>-26</td>
<td>-17</td>
<td>-19</td>
</tr>
<tr>
<td>R3</td>
<td>73/M</td>
<td>95</td>
<td>28.1</td>
<td>0.03</td>
<td>0.05</td>
<td>0.08</td>
<td>-25</td>
<td>-22</td>
<td>-23</td>
</tr>
<tr>
<td>R4</td>
<td>33/M</td>
<td>63</td>
<td>21.3</td>
<td>0.03</td>
<td>0.07</td>
<td>0.15</td>
<td>-25</td>
<td>-15</td>
<td>-14</td>
</tr>
<tr>
<td>V1</td>
<td>66/M</td>
<td>73</td>
<td>26.8</td>
<td>0.09</td>
<td>0.18</td>
<td>0.31</td>
<td>-5</td>
<td>-3</td>
<td>-3</td>
</tr>
<tr>
<td>V2</td>
<td>55/F</td>
<td>56</td>
<td>21.9</td>
<td>0.04</td>
<td>0.12</td>
<td>0.25</td>
<td>-11</td>
<td>-7</td>
<td>-5</td>
</tr>
<tr>
<td>V3</td>
<td>44/M</td>
<td>92</td>
<td>29.7</td>
<td>0.04</td>
<td>0.09</td>
<td>0.15</td>
<td>-14</td>
<td>-12</td>
<td>-11</td>
</tr>
</tbody>
</table>

BMI, body mass index; H, healthy subject; R, vasomotor rhinitis subject; V, valve syndrome subject. To facilitate interpretation of compliance, we have divided the nasal cavity into 3 distinct physiological regions, as previously described by our group (2). The 3 segments were defined for each subject from the minimum cross-sectional area (MCA) obtained with decongestant (0.05% oxymetazoline). The valve region was defined as the segment lying from a length of ΔL to MCA + 2ΔL (0.4 cm). The inferior turbinate region was the segment lying from MCA + 2ΔL to MCA + 5ΔL. The middle meatus region was the segment lying from MCA + 6ΔL to MCA + 9ΔL. The area profiles presented here are those obtained before application of decongestant.

Although the model proposed in this study is clearly an oversimplification of anatomic reality (independent element, pressure-drop law, flow distribution, steady-state conditions, etc.), it is the first model allowing an accurate description of the pressure inspiratory flow limitation phenomenon observed by physiologists in the nasal cavity. Physiological implications. Comparison of the compliant model and the rigid model simulations (Figs. 2–4) clearly indicates that nose-wall distensibility must be taken into account to understand inspiratory flow limitation in the nose. Moreover, under some pathological conditions (Figs. 3 and 4), wall distensibility must be taken into account even during quiet breathing. The simulation also indicates the site of the collapsed segment in the model by showing that the narrowest cross section of the collapsed segment occurs concomitantly with flow limitation. Surprisingly for ear, nose, and throat surgeons and physiologists, our model predicts that the greatest area variations are observed in the middle meatus and inferior turbinate regions and not in the valve region.

DISCUSSION

In clinical practice, it is well known that the nasal valve wall, composed of distensible structures (elastic tissue structures of the alae nasi and facial muscles of the nose), is a collapsible structure. This collapse of the nasal valve is believed to be a common cause of nasal obstruction, especially during exercise when a high negative pressure is generated by forced inspiration at the
Fig. 2. Simulations obtained with 1) the proposed compliant model and 2) the model with rigid wall conditions in 4 healthy subjects (subjects H1–H4; A–D, respectively). These simulations were compared with the measurement obtained by rhinomanometry. Left: inspiratory flow vs. pressure variation between the 2 ends of the model. Right: area profile curve vs. pressure variation between the 2 ends of the model.
Fig. 3. Simulations obtained with 1) the proposed compliant model and 2) the model with rigid wall conditions in 4 vasomotor rhinitis subjects (subjects R1–R4; A–D, respectively). These simulations were compared with the measurement obtained by rhinomanometry. Left: inspiratory flow vs. pressure variation between the 2 ends of the model. Right: area profile curve vs. pressure variation between the 2 ends of the model.
entry of the nasal cavity (17). In contrast, collapse of the deeper regions (inferior turbinate and middle meatus regions) has rarely been reported. However, a simple endoscopic examination easily illustrates collapse of some of these regions, confirming the prediction of the proposed model. Figure 5 presents two pictures obtained during such an examination. The first picture was obtained during deep inspiration, whereas the second picture was obtained during the subsequent expiration. The first picture clearly shows collapse of the inferior turbinate. It is interesting to note that our model predicts that the narrowest cross-sectional area of the nasal cavity is observed in the valve region before collapse and in the first part of the inferior turbinate region during collapse, except during the second step of the area profile curve as a function of $\Delta P$, whose validity is open to question (see Physical implications).

Fig. 4. Simulations obtained with 1) the proposed compliant model and 2) the model with rigid wall conditions in 3 valve syndrome subjects (subjects V1–V3; A–C, respectively). These simulations were compared with the measurement obtained by rhinomanometry. Left: inspiratory flow vs. pressure variation between the 2 ends of the model. Right: area profile curve vs. pressure variation between the 2 ends of the model.

Fig. 5. Endoscopic views of a left nasal cavity. Left: image taken during deep inspiration. Right: image taken during the following expiration.
first step of the area profile curve predicted by our model is, therefore, more or less in agreement with the clinical view that the nasal valve region is responsible for one-half of the airflow resistance of the entire respiratory tract (19).

Clearly, the flow limitation concept included in the proposed model is identical to the one used to describe the pharynx narrowing and its associated flow limitation encountered with the obstructive sleep apnea (12). In both cases, the narrowing of the duct due to collapsing pressure induces flow limitation. Such a phenomenon is favored by the couple large compliance and small area (large viscous pressure drop). However, the intraluminal pressure ranges concerned by flow limitation phenomena are somewhat different in the two cases. Indeed, in the nasal case, the estimated closing pressures were always negative (see Table 1), whereas the pharynx closing pressures were reported positive with obstructive sleep apnea subjects and slightly negative with normal subjects (13). In the pharynx, the presence of surrounding tissue induces “tissue pressure” that may tend to more easily collapse the pharyngeal airway (12).

In nasal conduct, due to the anatomy, this tissue pressure is probably less effective. Due to this closing pressure difference, flow limitation in the nose should not mask obstructive sleep apnea syndrome. Nevertheless, the nasal compliance, because it augments the nasal resistance, tends to drop off the intraluminal pressure at the end of the nose at the level of the pharynx. Consequently, it may emphasize or initiate the collapse of the pharyngeal region as observed with the obstructive sleep apnea syndrome. This phenomenon will depend on the balance between nasal pressure drop and pharyngeal closing pressure.

Nasal obstruction is a complex phenomenon that is not always easy to evaluate and quantify. The various methods of investigation used in routine practice generally provide fairly limited information about nasal function. X-rays, computer tomography scan, endoscopic examination, and acoustic rhinometry are performed under conditions of apnea. Rhinomanometry is performed during quiet breathing and indicates the tissue pressure in the nasal cavity is submitted to a variable pressure. Figure 6 shows the cross-sectional area variations of the nose when the nasal cavity is submitted to a variable pressure. Figure 6 presents an example of these area variations obtained by acoustic reflection. For clarity, this figure only shows the area of the three physiological regions (2), i.e., nasal valve, inferior turbinate, and middle meatus regions. As shown in Fig. 6, the cross-sectional areas of the three regions decrease extremely rapidly as the negative steady pressure is applied. Area variations and pressure variations appear to be concomitant. In contrast, when the applied pressure is switched to zero, the cross-sectional areas of the three regions returned much more slowly to their baseline values. This return to baseline values took ~15 s in the subject shown in Fig. 6.

Physical implications. Simulation of the proposed model was essentially inferred with three main assumptions: 1) steady-state conditions, 2) independent elements, and 3) specific pressure-drop law and flow distribution.

Resolution of the couples of Eqs. 1 and 2 using steady-state conditions implies that respiration consists of a series of equilibrium points, depending on the pressure variation between the two ends of the model. In other words, the area variations are considered to be almost instantaneous, at least compared with the time scale of the pressure variation, with no inertia phenomenon. We also assume that the wall behavior is purely elastic. The microvasculature of the nose is composed of a system of arterioles, a system of capacitance vessels, and arteriovenous anastomoses (7, 20). Blood flow in these microvascular systems is known to regulate nasal airway pattern, but the relationships between these various systems and their various pathways of control (parasympathetic, sympathetic nerves, etc.) are not well understood. The effects of external and mechanical forces, such as the pressure in the nasal airway, are unknown. In particular, there is no reason to believe that inflation and deflation of these microvascular systems induced by a sudden pressure variation in the nasal passages occur at the same rate. Such a rate discrepancy between inflation and deflation of vasoerectile tissue can be easily demonstrated by following the temporal area variations of the nose when the nasal cavity is submitted to a variable pressure. Figure 6 presents an example of these area variations obtained by acoustic reflection. For clarity, this figure only shows the area of the three physiological regions (2), i.e., nasal valve, inferior turbinate, and middle meatus regions. As shown in Fig. 6, the cross-sectional areas of the three regions decrease extremely rapidly as the negative steady pressure is applied. Area variations and pressure variations appear to be concomitant. In contrast, when the applied pressure is switched to zero, the cross-sectional areas of the three regions returned much more slowly to their baseline values. This return to baseline values took ~15 s in the subject shown in Fig. 6. This behavior...
introduces a hysteresis phenomenon into the pressure-flow curve, suggesting that the wall-state behavior differs according to whether pressure increases or decreases. In the case of increasing values of $\Delta P$, an elastic law appears to be sufficient to describe the wall behavior, whereas a more complex law, such as a viscous-elastic law, appears to be required to describe the wall behavior in the case of decreasing values of $\Delta P$. The importance of this hysteresis phenomenon probably depends on the subject, the breathing level, and the underlying disease. It seems reasonable to suppose that a disease affecting vasoreactive tissues, such as vasomotor rhinitis, may introduce a more marked hysteresis for the same breathing level than in the healthy conditions, as a difference in the vascular innervation has already been demonstrated between nonrhinitic and rhinitic patients (7). Further studies are needed to confirm this hypothesis. In any case, this discrepancy between our model and the measurement only concerns a very brief period at the end of the inspiratory phase. Underestimation of flow resistance during this period would, therefore, not really modify the estimation of the inspiratory volume and/or inspiratory work induced by the nose during a breathing cycle.

The second oversimplification of this model concerns the assumption of the segment independence. This oversimplification concerns both the compliance measurement procedure and the model simulation. Due to the continuity of tissues, displacement of one point of the nasal walls will clearly induce displacement of the wall close to this point. Unfortunately, there is no way to estimate this phenomenon in vivo. We, therefore, ignored this effect in this model. For this reason, the second step of the area profile curve vs. $\Delta P$, i.e., the step at which the increasing values of $\Delta P$ introduce a brutal collapse of a segment associated with an increase in cross-sectional area of all upstream segments, which is characterized by a very large area variation between the collapsed segment and the surrounding segments, does not appear to be physiologically valid. It probably explains the discrepancies observed between the model and the measurement observed in Figs. 2–4 (subjects H4, R3, R4, and V2) for the large value of $\Delta P$.

The third major oversimplification concerned the pressure-drop laws (viscous and kinetic), because there is no reason to suppose that these laws remain the same over the entire length of the nasal cavity. In the future, further studies based on three-dimensional reconstructions and numerical computations could improve our knowledge, but this is well beyond the scope of the present study. The good agreement observed between measurements and simulations computed with such a simplified law can probably be explained by the fact that our procedure tends to emphasize the most depressive segments that are the most critical segments in terms of pressure drop. Due to the uncertainty of the pressure law, it is difficult to define the relative importance of viscous pressure drop vs. the pressure drop due to the kinetic energy variation during the flow limitation phenomenon. Nevertheless, in the two pathological conditions, we found that the viscous pressure drop was much higher (more than one order of magnitude) than the pressure drop due to the kinetic energy variation. In these cases, and even if we underestimate the value of $\alpha$, we can probably conclude that the pressure drop due to the viscous loss is the main parameter.

In summary, the 14-element model proposed in this study is able to describe the inspiratory flow in the nasal cavity and to predict the flow limitation phenomenon. Our results suggest that it is essential to take into account the wall compliance linked to vasoreactive tissues (2) to understand the pressure-flow relationship in the nose. This is especially important in pathological situations such as vasomotor rhinitis or valve syndrome. The proposed model may also represent a useful tool for the clinician to reach a more accurate diagnosis and to more accurately predict the functional effects of treatment.

GRANTS
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REFERENCES
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