Nasal wall compliance in vasomotor rhinitis

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1Physiopathologie et Thérapeutique Respiratoires INSERM UMR 651; 2Service de Physiologie-Explorations Fonctionnelles, Hôpital Henri Mondor (Assistance Publique-Hôpitaux de Paris); and 3Service d’ORL et de Chirurgie Cervico-Faciale des hôpitaux Intercommunals et Henri Mondor (Assistance Publique-Hôpitaux de Paris), Créteil, France

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Papon, Jean-François, Lydia Brugel-Ribere, Redouane Fodil, Céline Croce, Christian Larger, Michel Rugina, André Coste, Daniel Isabey, Françoise Zerah-Lancner, and Bruno Louis. Nasal wall compliance in vasomotor rhinitis. J Appl Physiol 100: 107–111, 2006. First published September 1, 2005; doi:10.1152/japplphysiol.00575.2005.—Nasal compliance is a measure related to the blood volume in the nasal mucosa. The objective of this study was to better understand the vascular response in vasomotor rhinitis by measuring nasal cross-sectional area and nasal compliance before and after mucosal decongestion in 10 patients with vasomotor rhinitis compared with 10 healthy subjects. Nasal compliance was inferred by measuring nasal area by acoustic rhinometry at pressures ranging from atmospheric pressure to a negative pressure of − 10 cmH2O. Mucosal decongestion was obtained with one puff per nostril of 0.05% oxymetazoline. At atmospheric pressure, nasal cross-sectional areas were similar in the vasomotor rhinitis group and the healthy subject group. Mucosal decongestion did not induce any decrease of nasal compliance in patients with vasomotor rhinitis in contrast with healthy subjects. Our results support the hypothesis, already proposed, of an autonomic dysfunction based on a paradoxical response of the nasal mucosa in vasomotor rhinitis. Moreover, the clearly different behavior between healthy subjects and vasomotor rhinitis subjects suggests that nasal compliance measurement may therefore represent a potential line of research to develop a diagnostic tool for vasomotor rhinitis, which remains a diagnosis of exclusion.

acoustic rhinometry; nasal physiology; oxymetazoline

VASOMOTOR RHINITIS, ALSO CALLED noninfectious nonallergic rhinitis (1) or idiopathic rhinitis (23), is a chronic nasal dysfunction characterized by a nasal hyperreactivity, i.e., nasal blockage, rhinorhoea, and sneezing in response to nonallergic stimuli such as emotional factors, exposure to cold air, sudden temperature change, humidity, tobacco smoke, or irritating body sprays and cosmetics (7, 15). Vasomotor rhinitis remains a diagnosis of exclusion (19), i.e., absence of infection or exposure to drugs, absence of significant anatomical disorder of the nose or hormonal change, negative response to skin prick test, and normal serum specific IgE. Although rhinometric studies are useful to evaluate nasal hyperreactivity, no single diagnostic test, symptom, or clinical feature appears to be sufficiently relevant to demonstrate the presence of vasomotor rhinitis.

Nasal compliance is a dynamic measurement recently developed to evaluate inspiratory collapse of the nasal valve region, especially in the nasal valve syndrome observed in forced inspiration during exercise (2). This recent study also demonstrated that inspiratory collapse of the inferior and middle turbinate regions can also be observed in healthy subjects and that local compliance is related to mucosal blood volume.

As the pathophysiology of vasomotor rhinitis could involve an abnormality of nasal blood flow regulation (15, 20), the aim of this study was to investigate a possible alteration of nasal compliance in patients with vasomotor rhinitis.

SUBJECTS AND METHODS

Subjects. Ten patients with vasomotor rhinitis were included in the study. Table 1 lists the morphological characteristics for each subject. All patients were not treated at the time of inclusion. Ten healthy subjects were studied as controls (see Table 2 for morphological details). Pregnant women and previously treated patients were excluded from the study. Both vasomotor rhinitis and control patients were nonsmoking adults, with no history of atopic disease, chronic sinusitis, or endocrinopathy, in whom a nasal examination (anterior rhinoscopy for healthy subjects and nasal endoscopy for vasomotor rhinitis patients) was performed. Vasomotor rhinitis patients had negative skin prick tests and RAST responses to a panel of common allergens.

Each nasal fossa was examined separately and the vasomotor rhinitis group was compared with the healthy subject group. Each subject was evaluated in a comfortable upright seated position before and 10 min after nasal mucosa decongestion (MD) (0.05% oxymetazoline; one puff/nostril). In accordance with the guideline of local ethics committee that approved the study, informed consent was obtained from all subjects after complete information.

Nasal resistance. Posterior rhinomanometry was used to determine nasal resistance according to international recommendations (5, 8). A differential pressure transducer (Validyne MP 45, Northridge, CA; +/− 2 cmH2O) connected to a catheter inserted through a hole drilled in a stopcock obstructing the cylindrical part of a modified mouthpiece and connected to nasal face mask fitted with a Fleisch no. 1 pneumotachograph (Lausanne, Switzerland) was used to measure transnasal pressure and flow during breathing. Nasal resistance was defined as the ratio between transnasal pressure and flow when the transnasal pressure reached 1 cmH2O.

Nasal area and compliance. The longitudinal area profiles of the nasal cavity, A(x), were inferred by the two-microphone acoustic reflection method (21, 22). The acoustic reflection method is based on analysis of a planar wave propagating in a duct. Briefly, A(x) from the tip of the nostril to the middle meatus region (12) can be inferred from the relation between the incident and reflected waves at the airway entry under a set of restrictive but workable conditions (26). The set-up used was derived from a two-microphone device provided by Benson Hood Laboratories (Pembroke, MA) with two microphones and a horn driver mounted on a home-made wave tube (inner diameter: 12 mm, overall length: 220 mm). This wave tube was connected at one end to the subject’s nostril with a nosepiece (Benson Hood

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Laboratories), allowing tight closure of the nasal aperture without deformation of the nasal valve. The nosepiece was not inserted into the nostril. The other end of the wave tube was connected to a steady negative pressure generator (see Fig. 1). An acoustic wave was generated by the horn driver, and the resulting wave pressures were recorded by the two microphones. These digitized data were analyzed to obtain \( A(x) \) with a spatial step increment of \( \Delta L = 0.41 \) cm. The wall compliances of the nasal cavity were estimated as previously described (2, 9, 17) from the measurements of \( A(x) \) 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 cmH\(_2\)O.

During acoustic measurement, the steady pressure in the nasal cavity and in the wave tube was checked to ensure that there was no leak with a U-tube. If this pressure was not stabilized at the predetermined negative value, the acquisition sequence was rejected.

Compliance per unit length was defined as the ratio between the variation of area \( \Delta A(x) \) and the variation of steady pressure \( \Delta P \) applied to the nasal cavity. Compliance was computed as the slope of the line \( \Delta A(x) = C(x) \cdot \Delta P \), where \( C \) is the compliance) fitting the set of data using a least square error method.

For each patient the acoustic measurement procedure took \(-2\) min. Steady pressures were successively applied during 20 s. During these 20 s, 30 acoustic waves were generated and the corresponding areas distance curves were inferred. The first wave was generated 5 s after the desired pressure level was reached. For each pressure level the area-distance curves were averaged.

Data analysis. To facilitate interpretation of compliance and area, we divided the nasal cavity into three distinct physiological regions, as previously described by our group (2, 9). The three 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 \( -\Delta L \) to MCA + \( \Delta L \), the inferior turbinate region was defined as the segment lying from MCA + 2\( \Delta L \) to MCA + 5\( \Delta L \), and the middle meatus region was defined as the segment lying from MCA + 6\( \Delta L \) to MCA + 9\( \Delta L \). In each patient with vasomotor rhinitis, we distinguished between the results for the more resistive nostril (MRN) and the less resistive nostril (LRN). The average of these two groups was compared with the average obtained with healthy nostrils (HN) of the control group.

Statistical analysis. Comparisons among the three groups (HN vs. MRN vs. LRN) were performed using the Kruskal-Wallis test (Software: Statistica v6, Statsoft France). The limit of significance was defined as \( P = 0.05 \).

RESULTS

Resistance measurements. Before MD, the MRN group was significantly more resistive than the LRN and HN groups. No differences were observed between LRN and HN (Fig. 2A). After MD, no significant differences were observed among the three groups (Fig. 2B). Moreover, the MD-induced variation of resistance was not significantly different between groups (Fig. 2C).

Area measurements. No significant differences were observed between groups for any of the test conditions, i.e., before or after MD. Similarly, no difference in the area variation induced by MD was observed (Fig. 3A, C, and E).

Compliance measurements. No significant differences were observed between groups before MD (Fig. 3B). After MD, the compliances of the inferior turbinates and middle meatus regions were significantly higher in the vasomotor rhinitis groups than in the control group, whereas compliance of the valve region was similar in all three groups (Fig. 3D). Moreover, MD induced a negative variation of compliance in all regions in the HN group that was significantly different from the positive variation of compliance observed in the two vasomotor rhinitis groups (Fig. 3F).

DISCUSSION

To our knowledge, this is the first study of nasal compliance in vasomotor rhinitis patients. Our main finding was that the local vasoconstrictor did not induce a decrease of nasal compliance in patients with vasomotor rhinitis, in contrast with its action in healthy subjects.

Our results confirm the previous hypothesis (2) that nasal compliance in healthy subjects is related to mucosal blood volume and the quantity of vascular tissue in the turbinates. As MD reduces blood volume by acting on capacitance vessels of the cavernous plexus located in the vascular tissues of the turbinates, the volume of vasoerectile tissue is considerably reduced and no longer influences nasal compliance.

Table 1. Morphological characteristics of patients with vasomotor rhinitis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Gender</th>
<th>Weight, kg</th>
<th>Size, m</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43/M</td>
<td>72</td>
<td>1.82</td>
</tr>
<tr>
<td>2</td>
<td>33/M</td>
<td>63</td>
<td>1.72</td>
</tr>
<tr>
<td>3</td>
<td>68/M</td>
<td>69</td>
<td>1.75</td>
</tr>
<tr>
<td>4</td>
<td>55/M</td>
<td>85</td>
<td>1.78</td>
</tr>
<tr>
<td>5</td>
<td>31/M</td>
<td>81</td>
<td>1.76</td>
</tr>
<tr>
<td>6</td>
<td>48/F</td>
<td>67</td>
<td>1.7</td>
</tr>
<tr>
<td>7</td>
<td>52/F</td>
<td>70</td>
<td>1.7</td>
</tr>
<tr>
<td>8</td>
<td>39/M</td>
<td>87</td>
<td>1.87</td>
</tr>
<tr>
<td>9</td>
<td>39/F</td>
<td>74</td>
<td>1.63</td>
</tr>
<tr>
<td>10</td>
<td>59/F</td>
<td>110</td>
<td>1.75</td>
</tr>
</tbody>
</table>

M, male; F, female.

Fig. 1. Diagram of the set-up used to infer area and compliance by the acoustic reflection method.
property is important, because it indicates that this characteristic of vasomotor rhinitis is present independently of the side of the nasal cycle.

The pathophysiology of vasomotor rhinitis has not been clearly elucidated but an autonomic dysfunction has been proposed, on the basis of a paradoxical response of the nasal mucosa, i.e., increase of nasal resistance and/or reduction of nasal volume in response to peripheral sympathetic stimulations, e.g., exercise (16) or cold stimulation of the feet (23). Other studies have highlighted nasal epithelium alterations and a local increase of nitric oxide synthase in vasomotor rhinitis (10) as well as neuropeptide (substance P) release (18). Strongly sympathetic-dependent vasoconstriction was observed in our patients with vasomotor rhinitis, as nasal areas were increased after MD. However, even after this marked sympathetic-dependent vasoconstriction, the capacitance vessels were still able to refill in response to mechanical stimulation, i.e., negative pressure, as nasal compliance did not decrease in these patients after MD. In other words, this could mean that the constriction of nasal blood vessels induced by sympathetic stimulation of α-adrenergic receptors is less efficient in vasomotor rhinitis patients than in healthy subjects, as mechanical stimulation still inflates blood vessels. Our results therefore support the hypothesis of autonomic dysfunction already proposed in vasomotor rhinitis (15, 20). There are many α-adrenoceptor subtypes (α1 and its subtypes, and α2 and its subtypes) (4). At this time the repartition and the function of each α-adrenoceptor subtype are unknown in the human nasal mucosa. Further studies focused on the autonomic system in the nasal mucosa are required to describe the role of these receptors in the vasomotor rhinitis. For example, it is possible to perform functional studies by using selective stimulation and/or inhibition of α-adrenoceptor subtypes on isolated arteries from nasal mucosa. Moreover, binding studies of each α-adrenoceptor subtype and analysis of respective mRNA could be performed to obtain a better knowledge of the α-adrenoceptor subtype repartition in the normal and pathological nasal mucosa. Nevertheless, such studies are far beyond the scope of the present work.

These results may have an important impact on the diagnosis of vasomotor rhinitis, because this diagnosis remains a diagnosis established by exclusion mainly of allergic rhinitis. The variation of compliance induced by mucosal decongestion seems already allow discrimination between healthy subjects and vasomotor rhinitis patients with relatively high value of specificity sensitivity (see Table 3). If the absence of variation of compliance induced by MD is specific to vasomotor rhinitis, compliance measurement may represent a positive diagnostic tool for this disease. Because we know that the nasal mucosal reaction to peripheral sympathetic stimulation is less pronounced in vasomotor rhinitis patients than in allergic rhinitis patients (23), we can hypothesize that nasal compliance after MD should be decreased in allergic patients, whereas an increase was observed in vasomotor rhinitis patients. This hypothesis is purely speculative at the present time and needs to be investigated by further studies.

Before MD, nasal resistance was significantly increased in the MRN group of vasomotor rhinitis patients compared with healthy controls, whereas the areas were similar among the three groups. It is difficult to pinpoint the exact reason for this discrepancy between areas and resistances. From a mechanical
point of view, it is known that nasal resistance is related to the nasal valve (11) and to the MCA (13). In our study, the MCA in the MRN group was lower (results not shown) than in the healthy group, although the difference was not significant. Moreover, in fluid mechanics it is well known that for steady developed incompressible flow the pressure loss in a pipe depends on the shape of this pipe among others (14). Generally, pressure drop is inferred from the formula used for circular duct by replacing the diameter with the hydraulic diameter Dh (Dh = 4A/P where A is the pipe cross-sectional area and P is the perimeter). In other words, changing the shape of a duct by increasing the perimeter without changing the cross-sectional area will increase the pressure loss and the resistance. This result applied to the nasal cavity suggests that a narrow nostril with a crevice shape will be more resistive than a circular nostril. Finally, in the biomechanical literature (3), various studies of the velocity flow field in the nose indicate that inspiratory flow is mostly directed toward the lower part of the nose, more or less corresponding to the floor of the nasal fossa. This result suggests that resistance could be related to the part of the area corresponding to the lower part of the nose. Acoustic rhinometry gives a representation of nasal geometry (6, 22) by estimating the cross-sectional areas. This information is only partially representative of resistance because it was showed that acoustic rhinometry and rhinometry did not necessarily present correlation (24).

Published reports have shown that the physical characteristics of the nasal mucosa in vasomotor rhinitis may be highly variable (25). Our study confirms this observation, because we did not observe systematic swelling of the nasal mucosa of the inferior turbinate on either acoustic rhinometry or physical examination.

Table 3. Specificity and sensitivity analysis

<table>
<thead>
<tr>
<th></th>
<th>HN vs. MRN</th>
<th>HN vs. LRN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve region</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior turbinate region</td>
<td>80%</td>
<td>90%</td>
</tr>
<tr>
<td>Middle meatus region</td>
<td>80%</td>
<td>90%</td>
</tr>
<tr>
<td>specificity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sensitivity</td>
<td>100%</td>
<td>90%</td>
</tr>
</tbody>
</table>

The variation of compliance induced by mucosal decongestion may be used as criterion to discriminate between the healthy nostril (HN) group and the most resistive nostril (MRN) group of vasomotor rhinitis patient and between the healthy nostril group and the less resistive nostril (LRN). This table indicates for the different regions the specificity and the sensitivity of such a discrimination when a −0.0033 cm²/cmH₂O threshold was used.
In conclusion, acoustic rhinometry provides a noninvasive, convenient, and accurate method to measure the dimensions of the nasal airways and nasal wall compliance in patients with vasomotor rhinitis. This study also improves our understanding of the complex pathophysiology of vasomotor rhinitis. Because nasal compliance is not reduced after MD, vasomotor rhinitis patients present a very different behavior from that of healthy volunteers. Measurement of nasal compliance may therefore represent a potential line of research to develop a diagnostic tool for vasomotor rhinitis.

REFERENCES