Commentary on “The role of the large airways on smooth muscle contraction in asthma”

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TO THE EDITOR: In view of the Viewpoint by Solbert Permutt (3) in this issue of the Journal of Applied Physiology, it is becoming clear that the extent of decline in pulmonary function in asthmatics is not a simple function of the degree of smooth muscle shortening, but is the manifestation of complicated relationships among baseline airway structure, the degree of airway smooth muscle shortening, and compensatory changes in lung volumes. In this case, increases in total lung capacity and functional residual capacity minimized the impact of the observed increase in residual volume on the decline in pulmonary function (1).

We showed that in a mouse model of asthma, acute increases in airway wall thickness due to airway inflammatory effects leading to airway closure (and presumably change in lung volume) is the cause of airways hyperresponsiveness (AHR), not increased contractility of airway smooth muscle (2, 5). The study by Brown et al. (1) shows that airway structure and lung volumes are important determinants of AHR and pulmonary function in humans as well. Furthermore, Venegas et al. (4) showed that asthmatics have disturbed ventilation distribution that can be understood in terms of airway closure. These findings suggest a link between airway structure and its function that manifests as a change in lung volumes, irrespective of the model system used. Hence, we concur with Dr. Permutt that lung volumes are highly related to airway function in asthmatics and we believe an important determinant of AHR in both mice and humans.

REFERENCES

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