direction of change in volume history ratios reverses concomitant with a decrease in parenchymal hysteresis.13

These acute changes in parenchymal hysteresis with both induced obstruction and bronchodilation support the appropriateness of the relative hysteresis analysis in explaining volume history responses in asthma. Yet to be done are assessments of volume-pressure hysteresis changes and differences in inflammatory asthma with different degrees of obstruction. Also yet to be done is a direct examination of parenchymal hysteresis in relation to volume history effects in animal equivalents of asthma.

To date, diminished volume history responses have been shown in antigen-induced obstruction (in comparison with methacholine) in asthmatic humans who develop increased responsiveness18 and in guinea pigs that have peripheral inflammation histopathologically and by BAL analysis.19 To my knowledge, no direct assessments of parenchymal hysteresis in these circumstances have been made thus far.

CONCLUSIONS

The relationships among lung responsiveness, sites and mechanisms of responses, and effects of a DI on airway caliber could make volume history ratio assessment an interesting and perhaps valuable physiologic tool in both clinical and epidemiologic studies of asthma. In addition, increased insight into the phlogistic and physiologic aspects of the phenomena described should enhance our understanding of airway-parenchymal interactions in health and disease.

REFERENCES

5 Ingram RH Jr. Site and mechanism of obstruction and hyperresponsive asthma. Am Rev Respir Dis 1987; 136:S62-S64

Peripheral Lung Mechanics May Account for the Rise in the Maximal:Partial Ratio Which Follows Hyperpnea-Induced Bronchospasm*

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A deep inhalation (DI) increases airflow following hyperpnea-induced bronchospasm in asthmatics.1 This effect is thought to be due to an increase in airways hysteresis relative to parenchymal hysteresis, brought about by large airway constriction induced by hyperpnea. To investigate this hypothesis, we used a wedged bronchoscope technique to measure peripheral airway resistance (Rp) following simulated, local hyperpnea with a segmental cold, dry air challenge. Eight asthmatic and eight normal subjects initially underwent a whole-lung hyperpnea challenge by breathing −15°C, 5%CO2/air at a rate of 80% maximal voluntary ventilation for 5 min; the change in FEV1 following challenge was −7.6 ± 2.8% in normal subjects and −28.4 ± 3.8% (mean ± SEM) in asthmatics (p<0.01). To assess the effects of DI on airflow both before and after the hyperpnea challenge, maximal (M) to partial (P) flow rate ratios were measured at 30% vital capacity on consecutive M and P flow volume maneuvers.

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Baseline M:P was similar between normal subjects (0.96) and asthmatics (0.94 ±0.06), but M:P rose significantly more over baseline in asthmatics than in normal subjects (absolute change = 1.24±0.25 vs 0.30±0.13, p<0.01). Baseline Rp, determined by the bronchoscopic technique, was significantly higher in asthmatics than normal subjects (0.06 (0.05 to 0.23) vs 0.05 (0.03 to 0.07) cm H2O/mL/min; median interquartile range; p=0.04). Following a 5-min segmental challenge with 22°C, dry air at 500 to 1000 mL/min, Rp increased significantly more over baseline in asthmatics than normal subjects (0.10 (0.03 to 0.15) vs 0.02 (0.0 to 0.03), p=0.02). By analyzing stop-flow pressure decay curves in five of the asthmatics and six of the normal subjects, we found no difference between the pressure at the tip of the bronchoscope and segmental pressure, suggesting that the change in Rp did not occur in the proximal, large airways. From the decay time constant, we calculated peripheral lung compliance (Cp), which fell below baseline in many individuals after the challenge.

We conclude that following cold, dry air challenge, Rp rises more in asthmatics than in normal patients, and the increase in Rp is primarily due to increased resistance in distal (collateral), rather than proximal airways. The changes in Rp and Cp may be due to recruitment of lung units during the challenge, followed by subsequent derecruitment, with less recruitment occurring in asthmatics. To the extent that proximal, large airway resistance and peripheral compliance relate to airways and parenchymal hysteresis, respectively, we speculate that the rise in the M:P ratio which follows hyperpnea might be due, in part, to a fall in parenchymal hysteresis, and not just a rise in airways hysteresis.

REFERENCE