DISCUSSION

The normal occlusion pressure response to elastic loads in COPD patients in whom the response to flow loads is impaired indicates that the diminished response to flow loads cannot be explained by decreased muscle performance or fatigue. Rather, this phenomenon appears to represent a specific defect perhaps in the recognition of the resistive load by mechanoreceptors or in the interpretation of the sensory input in patients with COPD.

This notion is consistent with the magnitude estimation studies of Gottfried et al6 in which the ability of COPD patients to consciously detect and quantify changes in airflow resistance was found to be impaired.

Zechman and Davenport5 pointed out that detection of flow-resistive loads occurs at midinspiration and corresponds to peak flow, while elastic loads are perceived towards the end of inspiration corresponding to maximal change in lung volume.6 He predicted that a resistive load should not interfere with the detection of elastic loads since at end inspiration air flow is zero. Our findings of a normal response to elastic loads in patients with chronic airways obstruction is consistent with their predictions.

The relationship between the respiratory sensations elicited by external loads and the magnitude of the neuromuscular response is still unclear. It seems reasonable to believe that the intensity of the sensation elicited by a load might influence the motor respiratory response.

REFERENCES


Q. (Cherniack): What happens to the FRC during CO2 rebreathing?

A. (Nuchomovitz): We measured the FRC in 3 patients and there was no significant change from baseline.

Q. (Cherniack): What does an increased FRC do to the perception of external loading?

A. (Nuchomovitz): I don't know.

C. (Altose): We have studied a group of patients who had recovered from acute respiratory failure. After recovery, one-half of them returned to normocapnia while approximately one-half remained hypercapnic. Although all patients had clear abnormalities of hypercapnic and hypoxic respiratory responses, abnormalities in respiratory sensation were noted only in those patients who remained in chronic respiratory failure.

Q. (Zwilich): Does abnormal load response predict which COPD patient will develop high Pco2?

A. (Nuchomovitz): We don't know.

Interaction Between Chemical Ventilatory Drive and Respiratory Compensation for Flow-Resistive Loading*


Normal individuals increase inspiratory effort during inspiratory flow-resistive loading. Several conditions have been shown to impair this response. These include anesthesia, brain hypoxia, sleep and chronic airways obstruction. Since all of these may be associated with blunting of chemical drive, and load compensation is tested during progressive hypercapnia, we determined whether alterations in ventilatory response to CO2 per se, could influence the respiratory compensation for flow-resistive loading. Ten goats were prepared with chronic tracheostomies and indwelling arterial and venous cannulae. Ventilatory and occlusion pressure (P100, as an index of inspiratory drive) responses to rebreathing CO2 in O2 were assessed before and after imposition of an inspiratory flow-resistive load of 25 cm H2O/L/sec. In five goats, studies were done before and after infusion of doxapram (3 mg/min); in the other five, studies were repeated 30 min after infusion of Tris buffer (15 mM/kg).

As anticipated, doxapram increased and Tris buffer decreased ventilatory responses to CO2. However, occlusion pressure responses to CO2 were enhanced by flow-resistive loading to an approximately equivalent degree before and after either drug. We thus conclude that the

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<table>
<thead>
<tr>
<th>Pre-infusion</th>
<th>Post-infusion</th>
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<tr>
<td></td>
<td>Unloaded</td>
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<tr>
<td>TRIS</td>
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<tr>
<td>ΔV/ΔPco2 L/min/mm Hg</td>
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<td>ΔV/ΔPco2 ml/mm Hg</td>
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<td>ΔPmin/ΔPco2 × mm Hg</td>
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<td>ΔPmin/ΔPco2 cm H2O/min/mm Hg</td>
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</tr>
<tr>
<td>DOXAPRAM</td>
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<td>ΔV/ΔPco2 L/min/mm Hg</td>
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</tr>
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<td>ΔV/ΔPco2 ml/mm Hg</td>
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<tr>
<td>ΔPmin/ΔPco2 × mm Hg</td>
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<tr>
<td>ΔPmin/ΔPco2 cm H2O/min/mm Hg</td>
<td>.38±.09</td>
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</table>
compensation for an inspiratory flow-resistive load, when assessed as relative change in the occlusion pressure response to CO₂, is uninfluenced by acute changes in CO₂ ventilatory drive. An additional finding of interest was that upon analysis of unloaded frequency and occlusion pressure responses to CO₂ it appeared that the effect of Tris buffer on chemical ventilatory drive was primarily mediated by the rate-generating mechanism.

Q. (Flenley): Does external resistive loading simulate upper airway obstruction during sleep?
A. (Edelman): It might, but we have no data to prove it.

Dyspnea in Chronic Airways Obstruction

N. K. Burki, M.B., F.C.C.P.*

The sensation of breathlessness or dyspnea is one of the cardinal symptoms of lung disease, but an understanding of the mechanism of production of this sensation remains elusive. Campbell and co-workers¹ ² have postulated that this sensation is a product of “length-tension inappropriateness,” and the present study was undertaken to assess the validity of this concept in terms of the relationship of ventilation to mouth occlusion pressure.

METHODS

Twenty-four male patients with chronic asthma or chronic Airways obstruction were divided into two groups on the basis of the presence or absence of subjective and objective dyspnea at rest. The assignment of individuals to each group was made at the beginning of the study without foreknowledge of the pulmonary function tests or other measurements.

Spirometric tests and body plethysmography were performed in each patient. Minute ventilation (Ve), breathing frequency and the breathing pattern were measured in the seated position in each subject while he breathed via a mouthpiece through a Collins J valve. Fifty consecutive breaths were recorded and analyzed. The inspiratory side of the Collins J valve was completely occluded after every four to six breaths so that the next breath was occluded at FRC for 0.25 to 0.30 sec. The mouth pressure developed 0.1 sec after the start of inspiration (Pp,e) and the maximum rate of rise of this pressure within the first 0.2 sec (dP/dt max) were measured³ ⁴ a minimum of eight times in each subject. The expired gas was collected and analyzed for calculation of Ve, the respiratory exchange ratio (R) and the O₂ uptake (Vo₂).

The sequence of measurements consisted of spirometry and body plethysmography, followed by simultaneous measurements of ventilatory pattern, mouth occlusion pressure and collection of expired gas over a three-five minute period. Toward the end of the collection of expired gas, an arterial blood sample was obtained for measurement of arterial Po₂, Paco₂, and pH.

RESULTS AND DISCUSSION

The breathless patients had a greater degree of airway obstruction (Table 1), though mean FRC was not significantly different; these results are in accordance with previous studies. However, there was considerable overlap in the FEV₁, sGaw and FEV₁/FVC values, and it would seem, therefore, that the degree of airway obstruction is unlikely to be the sole determinant of dyspnea in these patients. Chemoreceptor respiratory drive is unlikely to have been a significant factor since Paco₂ did not differ significantly (Table 1), and the slightly but significantly lower mean PaO₂ level in the breathless group is unlikely to result in a major chemo- receptor drive. Mean Vo₂ was also not significantly different between the groups; nor were Ve or respiratory frequency or the ratio T₁/T₁₀₁, which is an index of central breath timing.⁵ Thus, breathlessness does not appear to be associated with differences in total O₂ consumption, Ve or central breath timing. Mouth occlusion pressure was increased above normal levels in both groups of patients (Table 2), but it was significantly higher in the breathless compared to the nonbreathless group. This would indicate an increased inspiratory neuromuscular drive in breathless patients.

Campbell and co-workers¹ ² suggested that if the

<table>
<thead>
<tr>
<th>Group</th>
<th>FRC Liters</th>
<th>FEV₁/FVC, %</th>
<th>PaO₂ mm Hg</th>
<th>PaCO₂ mm Hg</th>
<th>R</th>
<th>Vo₂ L/min</th>
<th>BTPS</th>
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</thead>
<tbody>
<tr>
<td>Breathless (age 54 ± 6 yrs) n = 14</td>
<td>5.8</td>
<td>±1.4</td>
<td>±1.5</td>
<td>±0.9</td>
<td>0.36</td>
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<tr>
<td>Non-breathless (age 51 ± 9 yrs) n = 10</td>
<td>4.8</td>
<td>±1.1</td>
<td>±1.3</td>
<td>±0.3</td>
<td>0.31</td>
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Significance of difference, P > 0.05 < 0.01 < 0.05 > 0.5 > 0.05 > 0.1

FRC = functional residual capacity; FEV₁ = forced expired volume in 1 sec; FVC = forced vital capacity; PaO₂, PaCO₂ = arterial Po₂ and Pco₂ respectively; R = respiratory exchange ratio; Vo₂ = O₂ uptake.

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