spinal level. More information is needed to determine what specific stimuli activate the receptors from which these fibers arise. Such information may clarify their role in conditions that may predispose to ventilatory failure. This information should also allow the specific reflex ventilatory effects produced by activation of these receptors to be defined.

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Inspiratory Reflexes in Diabetes Mellitus*


Diabetic neuropathy may involve both autonomic and sensory nerves. To determine whether diabetic patients have deficient respiratory system proprioception, we administered inspiratory resistive loads (ΔR) to 7 diabetic (D) and 3 normal (N) subjects and measured ventilation (Ve) before and after loading. All D and N subjects had normal spirometry and inspiratory muscle strength.

While D and N responded similarly to ΔR, of 13.6 cm

*From the Department of Medicine, New Jersey Medical School, Newark.
Table 1—Inspiratory Data for Diabetic and Nondiabetic Patients

<table>
<thead>
<tr>
<th>Factor</th>
<th>Baseline</th>
<th>Breath #1</th>
<th>Breath 3 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>D</td>
<td>N</td>
<td>D</td>
</tr>
<tr>
<td>Ve(L/m⁻¹)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>5.8</td>
<td>7.6</td>
<td>7.7</td>
</tr>
<tr>
<td>SD</td>
<td>2.4</td>
<td>3.6</td>
<td>3.2</td>
</tr>
<tr>
<td>PetCO₂</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>38</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>5</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

*Different from Ve (D) baseline, p<0.002.

H₂O L⁻¹S⁻¹ given over 3 minutes, D failed to compensate for ΔR, during the first loaded breath (Table 1).

Deficient load compensation in D did not correlate with vagal neuropathy (as quantified during Valsalva), peripheral neuropathy (electromyogram), or duration of diabetes. Neither Ve, or f alone could account for failure to compensate for loading in D.

CO₂ stimulation showed no difference between N and D subjects during unloaded or loaded breathing. We conclude that subtle abnormality in proprioception may be identified in D during loaded breathing.

Mechanism of Detection of Added Respiratory Loads*

A. Puddy, M.D.; D. Jung, M.D.; G. Giesbrecht, Ph.D.; R. Sanii, Ph.D.; and M. Younes, M.D.

Changes in respiratory mechanical loads are readily detected by humans. Although it is widely believed that respiratory muscle afferents serve as the primary source of information for load detection, there is, in fact, no convincing evidence to support this belief. To assess the validity of this assumption, we developed a shell that encloses the body, excluding head and neck, and where the seal between neck and shell is sufficiently stable to preclude extraneous clues at the neck as a result of seal motion when shell pressure is changed. A special loading apparatus altered pressure in proportion to expired volume (elastic load) in 1 of 3 ways: (1) at the mouth only (C), producing a conventional load in which respiratory muscles are loaded and airway and intrathoracic pressures are made negative in proportion to volume; (2) both at mouth and in the shell. (AW); here, the same pattern of airway and intrathoracic pressure occurs, but the muscles are not loaded since Pm (ie, Pm-Paw) is unchanged; (3) positive pressure in proportion to volume at the shell only, loading the chest wall but with no change in airway or thoracic pressures (CW). The threshold for detection (δEₘₙ) with the 3 types of application was determined in 7 normal subjects. δEₘₙ (mean ± SE) was 2.16 ± 22, 2.65 ± .54, and 6.21 ± .85 cm H₂O/L for C, AW, and CW, respectively. Therefore, the active chest wall, including muscles, is a much less potent source of information than structures affected by negative airway and intrathoracic pressure. The latter account for the very low threshold for load detection.

The Respiratory and Vascular Responses Evoked by Chemical Activation of Small Fiber Phrenic Nerve Afferents in Dogs*

S. Hussain, M.D.; A. Chatillon, M.D.; S. Magder, M.D.; and Ch. Roussos, M.D.

In chloralose-anesthetized, mechanically ventilated and vagotomized dogs, we assessed the influence of small fiber (type III and IV) phrenic afferents on the inspiratory efferent drive, arterial pressure, and heart rate. These afferents were selectively activated by intra-arterial (phrenic artery) infusion of capsaicin (1 ml bolus). We used an in situ isolated and innervated left diaphragm preparation in which the left costal and crural attachments to the chest wall were severed and the free diaphragmatic margin was then secured to 3 metal bars which in turn were connected to 3 force transducers. The arterial (left phrenic artery) and venous (phrenic vein) drainage of the left diaphragm were catheterized and isolated from the rest of the systemic circulation. Inspiratory neural drive was assessed by recording the integrated electromyelogram (EMG) activities of left hypoglossus, left parasternal, right and left diaphragm. Mean arterial pressure, heart rate, and phrenic arterial pressure were also monitored. Spontaneous breathing attempts were evoked by reducing the tidal volume and frequency of the ventilator.

In the first group of dogs (n=8), capsaicin was infused every 30 minutes at doses of 1, 10, 50 μg/ml. In the second group (n=5), capsaicin concentrations of 100 and 500 μg/ml were injected. Doses higher than 10 μg/ml elicited a significant rise in the peak activities of the 4 inspiratory muscles, shortened expiratory time, and increased breathing frequency. Mean arterial pressure and heart rate also rose, whereas phrenic arterial flow declined significantly. These changes (except that of phrenic arterial flow) subsided within 2 to 3 minutes. Repeated injection of capsaicin resulted in the blunting of the respiratory and vascular responses. The rise in the inspiratory neural drive, heart rate and arterial pressure were not observed when capsaicin was infused after sectioning of the left phrenic nerve. To compare the responses evoked by small fiber phrenic afferent to those of limb muscles, we infused capsaicin (50 μg/ml) into the arterial supply of an isolated, innervated gastrocnemius preparation. Capsaicin infusion in this muscle evoked changes similar to those evoked by diaphragmatic injection.

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Supported by National Heart, Lung and Blood Institute grant No. 1R01-HL33369-01A1.