Tracking Procedure to Assess Load Detection Threshold

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Essentially all of the past work on load detection, beginning with the classic studies of Campbell’s groups, has focused on subjects with normal pulmonary mechanics. As a result, there is very little known about how alterations in natural loads (resistive and/or elastic) influence load perception. We are currently initiating investigations to examine the ability of asthmatic patients to perceive added resistive loads. Prior to initiating the patient studies, we felt it was necessary to develop procedures for defining load perception thresholds that are simpler and less time-consuming than those we and others have used in the past. This report describes such a procedure and presents some of our initial experiences.

This effort began with extended conversations with Dr. R. L. Wiley and his psychologist collaborator, Dr. Zechman.

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Table 1—Carbon Dioxide Responses

<table>
<thead>
<tr>
<th>Subject</th>
<th>V_e/P_Co_2</th>
<th>P_H_2O/P_Co_2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.71</td>
<td>0.97</td>
</tr>
<tr>
<td>2</td>
<td>3.48</td>
<td>0.59</td>
</tr>
<tr>
<td>3</td>
<td>2.88</td>
<td>0.56</td>
</tr>
<tr>
<td>4</td>
<td>1.55</td>
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<td>5</td>
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<td>0.53</td>
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<td>6</td>
<td>5.34</td>
<td>0.63</td>
</tr>
<tr>
<td>7</td>
<td>3.65</td>
<td>0.81</td>
</tr>
</tbody>
</table>

Mean ± SD 3.36 ± 1.15 0.64 ± 0.20

Preliminaries

Pulmonary function tests are performed on each subject consisting of spirometry and body plethysmography.

Each subject is given a brief standardized training/orientation run on the apparatus. Prior to (and following) each tracking test, the subject’s pulmonary resistance is estimated at rest using the interrupter technique according to the recent report by Jackson, Milhorn and Norman.

Tracking Procedure

The subject, seated comfortably in a dental chair, is connected to the mouth piece and three to five minutes are allowed to adjust to the apparatus and its minimal resistance.

An inspiratory resistance is added (∆R) to the minimal resistance of the apparatus for a single inspiration approximately every third breath. This is called a trial. The subject presses a button if the added resistance is detected.

If the subject’s response indicates that he or she detected the added resistance on the preceding trial, the ∆R added on the subsequent trial is lowered one level; when the subject fails to detect the ∆R of the preceding trial, the resistance is increased one level on the next trial. This simple procedure is continued for 30 trials and requires 5-10 minutes depending upon the subject’s respiratory rate.

The tracking procedure outlined above was carried out in two modes: 1) with a signal light indicating to the subject the presence of an added inspiratory load, and

D. E. Parker, at Miami University, who have been developing scaling techniques for evaluating respiratory load perception. As a result of this consultation, we settled upon using a simple tracking test to estimate thresholds for detection of added inspiratory airflow resistance. For these tracking observations, our resistance manifold, described previously, was modified to provide 11 discrete, approximately equal, levels of resistance giving a resistance range from 0.16 to 1.89 cm H_2O/L/sec measured at 0.5 L/sec. The manifold was connected to the inspiratory side of a Collins J-valve (Fig 1). The minimal resistance (inspiratory) of the apparatus was 0.30 cm H_2O/L/sec.

The basic features and steps of the tracking test are outlined below.

REFERENCES

6 Whitelaw WA, Derenne J-Ph, Millet-Emili J: Occlusion pressure as a measure of respiratory center output in conscious man. Resp Physiol 23:181-190, 1975

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2) without such a signal light.

Examples of 30-trial tracking plots are shown in Figure 2. The added resistance values plotted against trial number, are expressed as fractional changes in resistance, added resistance/total initial resistance ($\Delta R/R_{\text{total}}$). The total initial resistance is the sum of the subject's pulmonary resistance and the minimal resistance of the apparatus during inspiration. Fractional, rather than absolute changes in load are used since previous studies demonstrated that detection of resistive loads generally obeyed the Weber-Fechner Law. We have arbitrarily used the last ten trial points to calculate mean perception threshold values for each subject.

Figure 1. Experimental apparatus showing resistance manifold connected to the inspiratory side of a Collins J valve. The resistance elements are encapsulated porous metal discs 2% inches in diameter arranged in series. The operator selects the desired inspiratory resistance level by appropriate placement of rubber stoppers in the manifold inlet ports.

Figure 2. Examples of tracking plots for two subjects. Approximately every third breath, resistance was added for a single inspiration (a trial). When the subject detected the $\Delta R$ (trials designated by $x$ symbols), the next load was decreased one level and when not detected (trials designated by solid dots), the load was increased one level. In the tracking plots labeled "light on," a light came on to identify the loaded inspiration. Resistance values on the ordinate are expressed as fractional changes in resistive load ($\Delta R/R_{\text{total}}$).
Thus far, we have estimated threshold for detection of inspiratory resistive loads in seven normal young adults. The individual and group performances are shown in Figure 3. With one exception, light presentation during the tracking procedure lowered mean detection threshold values. Without the light signal, the group mean \( \Delta R/R_T \) detectable was 0.45 ± 0.20, whereas with the light the corresponding value was 0.27 ± 0.16. The rationale for using the light presentation was to maintain a relatively high, constant level of attentiveness by the subject. The light was expected to yield lower threshold values with less variability. This was the trend observed but the differences are not statistically significant.

At this point we decided to explore the question of whether or not an individual's ability to detect mechanical loads varied in some positive manner with sensitivity or responsiveness to chemical stimuli. Howell\(^5\) reported at the McMaster University Symposium on Loaded Breathing that two patients with raised Pco\(_2\), in whom they suspected reduced CO\(_2\) sensitivity, had impaired ability to detect added mechanical loads. Since the load threshold values observed within our normal group varied three to four fold between subjects, we decided to measure and compare their individual CO\(_2\) responses. Both ventilatory responses and the mouth pressure generated during a 100 msec occlusion of the airway at the onset of inspiration were measured about every 20 seconds during the course of CO\(_2\) rebreathing using the method of Read.\(^6\) The slopes derived from the individual CO\(_2\) response curves are presented in Table 1.

To examine the possible relationship between CO\(_2\) responsiveness and detection threshold, slope data characterizing each subjects' CO\(_2\) responses were plotted against their load detection threshold values. The results are presented in Figure 4. It is obvious that there is no apparent correlation between levels of CO\(_2\) sensitivities and load detection thresholds exhibited by our group of normal subjects. The interesting and important suggestion that patients with low CO\(_2\) sensitivity may have high thresholds for perceiving mechanical loads will be
examined in subsequent patient studies.

REFERENCES


Some Steady-State Effects of Respiratory Loads*

N. R. Anthonisen, M.D.

The subject of ventilatory loading recently has been reviewed very thoroughly1 and I will not presume to improve this. I will simply point out what seems to be some general truths, and illustrate these with our experience from our laboratory.

Loads stress the ventilatory control system in a way which may be similar to the stresses imposed by disease and may therefore afford insight into problems of clinical relevance. Loading allows each subject to be his own control; analysis is much simpler than when the investigator compares normals to subjects with chronic bronchitis. On the other hand, loading may not be equivalent to disease. Subjects who detect external respiratory loads may in fact be detecting abnormalities of mouth pressure. I will show evidence that application of acute loads do not produce the same results as do chronic disease.

Investigators without clinical interests use ventilatory loading to discern specific mechanisms of ventilatory control. In general to avoid dealing with a lumped system with nonspecific outputs such as ventilation, these experiments have been conducted in anesthetized animals. Species differences and anesthesia lead to serious difficulties in interpretation of these results in terms of intact awake humans.

A final general issue in ventilatory loading is that of quantitation of the applied load. It is relatively easy to apply known, linear elastances, resistances and thresholds and most workers indicate exactly what they have done. Comparing different kinds of loads is quite another thing, however. At present there is no universal standard by which elastic and resistive loads, for example, can be compared.

We have been interested in the effects of loads in conscious humans in steady-state situations but we have not looked at elastic loading. Suffice it to say that in contrast to results in anesthetized humans and animals, in awake men, elastic loads effect ventilation very little. Ventilatory pattern is changed, but total output remains constant.2

Effect of resistive loading in conscious humans is very dependent upon the circumstances. Milic-Emili and Tyler3 indicated that during CO2 breathing, inspiratory work was uniquely related to PaCO2 and independent of resistance. The ventilation achieved with a given resistive load was simply the result of the fraction of the total inspiratory work which was not done on the external resistance. This appears not to be the case during hypoxic breathing, however. Given the same control stimulus intensity, hypercapnic ventilation is more depressed by a resistive load than hypoxic ventilation.4,5 This would appear to imply some kind of stimulus differentiation not apparent in the unloaded system. During resistive loading, exercise ventilation is less dependent upon inspiratory work rate than at rest. As the exercise level increases, ventilation is less influenced by loading6 and if the resistance is of the same order as that of the lungs, readily detectable at high levels of exercise, it has very little influence on ventilation.

The studies quoted above have the obvious drawback that ventilatory control was assessed by measuring ventilation. When ventilatory mechanics are altered, as is the case in loading or disease, ventilation is not the same as ventilatory effort, and it is the latter we wish to measure. The most practical method of assessing ventilatory effort which is currently available is the measurement of P0.1, the pressure measured at the mouth during the first 0.1 sec of inspiration against a closed airway.7 We have measured P0.1 both with and without resistive loading at rest and have found that both during hypoxia and hypercapnia, P0.1 increased with addition of inspiratory resistance.8 We interpret this as indicating that acute increase of inspiratory resistance caused increases in inspiratory drive. In our series of nine subjects we were unable to relate the degree of augmentation of P0.1 to control measurements of ventilatory sensitivity such as the slope of the unloaded CO2 response curve. This might indicate that the response to resistive loads is under different control than nonloaded ventilatory sensitivity, but also might indicate that our assessments of inspiratory drive were inadequate. Similar interpretations could be made of measurements of P0.1 after meperidine (1 mg/kg) administration. Ventilatory and P0.1 responses to both hypoxia and hypercapnia were depressed. Of greater interest was the fact that meperidine appeared to decrease selectively the resistance-induced augmentation of P0.1. For a given stimulus which produced the same P0.1 in a subject before and after administration of meperidine, addition of resistance resulted in a smaller