Expiratory flow limitation during exercise in competition cyclists

SUSANA MOTA, PERE CASAN, FRANCHEK DROBNIC, J ORDI GINER, OLGA RUIZ, JAOUQUIN SANCHIS, AND JOSEPH MILIC-EMILI

Expiratory flow limitation during exercise in competition cyclists. J. Appl. Physiol. 86(2): 611–616, 1999.—In some trained athletes, maximal exercise ventilation is believed to be constrained by expiratory flow limitation (FL). Using the negative expiratory pressure method, we assessed whether FL was reached during a progressive maximal exercise test in 10 male competition cyclists. The cyclists reached an average maximal O2 consumption of 72 ml·kg\(^{-1}\)-min\(^{-1}\) (range: 67–82 ml·kg\(^{-1}\)-min\(^{-1}\)) and ventilation of 147 l/min (range: 122–180 l/min) (88% of preexercise maximal voluntary ventilation in 15 s). In nine subjects, FL was absent at all levels of exercise (i.e., expiratory flow increased with negative expiratory pressure over the entire tidal volume range). One subject, the oldest in the group, exhibited FL during peak exercise. The group end-expiratory lung volume (EELV) decreased during light-to-moderate exercise by 13% (range: 5–33%) of forced vital capacity but increased as maximal exercise was approached. EELV at peak exercise and at rest were not significantly different. The end-inspiratory lung volume increased progressively throughout the exercise test. The conclusions reached are as follows: 1) most well-trained young cyclists do not reach FL even during maximal exercise, and, hence, mechanical ventilatory constraint does not limit their aerobic exercise capacity, and 2) in absence of FL, EELV decreases initially but increases during heavy exercise.

VENTILATION (Ve) is not believed to limit the exercise capacity of healthy, untrained young individuals (4, 21, 25, 28). At maximal exercise in such subjects, there is a considerable ventilatory reserve, with Ve amounting to 60–70% of maximum voluntary ventilation in 15 s (MVV\(_{15}\)), and expiratory flow limitation (FL) is seldom detected (21). Electromyographic signs of fatigue have been reported for peripheral but not respiratory muscles (28). However, for endurance-trained athletes who reach very high Ve levels during exercise, ventilatory capacity may be a limiting factor. There is evidence in the literature for development of both FL (2, 8, 14) and respiratory muscle fatigue (2, 10, 29) in well-trained subjects, although its relevance has not been clearly established. Tidal FL constitutes a mechanical constraint for Ve, and its presence implies that any further increase in expiratory flow must take place at increased lung volume (14, 17, 25). This implies dynamic hyperinflation with a concomitant increase in inspiratory work and impaired inspiratory muscle function. Moreover, in the presence of FL, the intrathoracic expiratory pressures may surpass the maximal effective values, i.e., the critical pressures (Pcrit) that allow maximal expiratory flow (V\(_{max}\)) to be reached at each lung volume. This situation implies an unwarranted increase in expiratory muscle work as well as the possibility that expiratory flow will decrease paradoxically as a result of airway compression. Thus the presence of tidal FL during exercise may herald compromised Ve.

In the past, detection of FL was difficult. Until recently, the method commonly used during exercise was to superimpose tidal and maximal flow-volume (FV) loops, by using inspiratory capacity (IC) maneuvers to determine the end-expiratory lung volume (EELV), as proposed by Hyatt in 1961 (11); FL was thought to be present when the individual expired along his or her maximal expiratory flow-volume (MEFV) curve. The validity of this technique, however, has been challenged on several grounds, leaving the assessment of FL in previous studies of athletes open to question. On one hand, thoracic gas compression artifacts (12) and differences in time-volume histories of spontaneous breathing cycles and forced vital capacity (FVC) render tidal and maximal FV curves noncomparable (6). The appreciable changes in respiratory mechanics that take place during exercise (14, 28) also make the superimposition of these curves inappropriate.

A simple alternative technique for detecting FL was recently described by Valta et al. (26). Negative expiratory pressure (NEP) is applied at the mouth, and the FV curve during the ensuing expiration is compared with that of the preceding breath. The NEP technique for detection of FL during mechanical ventilation was validated by concomitant determination of isovolume flow-pressure relationships. The NEP technique was later applied to assess FL at rest and during exercise in both normal subjects and patients with chronic obstructive pulmonary disease (17, 18). In the present study, we have used the NEP technique to assess whether highly trained cyclists exhibit FL during exercise progressing to exhaustion.

MATERIALS AND METHODS

Ten male competition cyclists were enrolled. Two were members of the National Spanish Mountain Bike Olympic...
Flow Limitation During Exercise in Cyclists

Exercise test. A cycle ergometer with electromagnetic brake (Jaeger, Würzburg, Germany) was used for an incremental work test, which began with a warm-up period of 3–5 min at 100 or 200 W, followed by progressive load increases of 30 W every 3 min. The test ended at the point of peak exercise, which is defined as inability to maintain the load reached. Electrocardiographic monitoring was continuous. Ventilatory variables were recorded, and gas analysis was performed breath by breath using a turbine pneumotachograph (linear response up to a flow of 20 l/s, with a precision of ±2% of scale) and a gas analyzer (Champion, Jaeger). The pneumotachograph and mouthpiece had a combined dead space of 35 ml. During the last minute of each exercise level, a NEP test was performed, followed immediately by an IC maneuver so that changes in EELV and end-inspiratory lung volume (EILV) could be assessed.

Lung function testing. Before the incremental exercise test, the MVV15 was determined by using a Fleisch no. 3 pneumotachograph (Datospir-100, Sibelmed, Barcelona, Spain). The best of three maneuvers was chosen for subsequent analysis. With the subject seated on the cycle ergometer, forced spirometry was performed before the exercise test by following recommended procedures. In eight subjects, spirometry was repeated within 10 min after the test was completed. Maximum inspiratory pressure was measured at residual volume with a manometer (model 163, Sibelmed). Maximum expiratory pressure was measured at total lung capacity (TLC) with the same device.

NEP technique. The system for applying NEP was similar to that described by Eltayara et al. (7), with the use of a Venturi device (Aeromech Devices, Almonte, Ontario) capable of generating a range of negative pressures when compressed gas is delivered through it. Gas was supplied from a tank through a flowmeter and an electrically operated valve that opened when a control box received a signal from the pneumotachograph, after a previously programmed delay after onset of expiration. The time that the valve remained open was also set by the control box. After the start of expiration, the system took ∼100 ms to reach the desired negative pressure (∼10 cmH2O) (27). The Venturi device was placed at the distal end of the Fleisch no. 3 pneumotachograph that recorded the tidal FV loops. The signal recorded by the pneumotachograph was filtered and digitized at 100 Hz (Datospir-500, Sibelmed), and volume was obtained by numeric integration of the flow signal. The FV curves were monitored in real time by using a program that allowed successive FV curves to be superimposed and viewed (assuming equal lung volume at the start of expiration). In all instances, we recorded the FV loop of the breath during which NEP was applied and that of the preceding control breath. The data were stored on a floppy disk for subsequent analysis. In each case, NEP was applied after the Fleisch pneumotachograph and Venturi device were connected in series with the turbine transducer that measured V˙e during the exercise test (see Fig. 1). After connection, we waited the required number of cycles for the respiratory pattern to become regular, as observed by visual inspection of the tidal FV curves on the computer screen.

Data analysis. Expiratory FL was assessed both at rest and during exercise. NEP was applied throughout the tidal expiration, except for the initial 100-ms delay (see NEP technique). As previously described (26), we considered FL to be present when the tidal expiratory FV curve with NEP was superimposed on that of the preceding expiration. We then determined what percentage of the tidal volume (Vt) was encompassed by FL (18, 26). Immediately after the NEP test, the subjects were asked to perform an IC maneuver to determine EELV in relation to TLC. When possible, the procedure was repeated at the same exercise level. When the observer saw that the subject had not taken a deep breath for the IC maneuver, the data were discarded.

Statistical analysis. Results are expressed as means with SD or range. To compare variables recorded at different times of testing, either a Wilcoxon t-test for comparison of two means or a Friedman test for multiple comparisons was used. The level of significance was P < 0.05.

RESULTS

The subjects’ mean age was 21 yr (range: 16–33 yr). Their lung function data were within the normal range. Individual anthropometric and peak exercise data are shown in Table 1. Lung function data are given in Table 2. In the eight subjects in whom spirometry was repeated after exercise, forced expiratory volume in 1 s increased by 8% (range: 2–18%), and forced maximal midexpiratory flow increased by 12% (range: −2–31%). Both increments were statistically significant. A nonsignificant tendency toward increase was observed for FVC.

Table 1. Individual anthropometric and peak exercise data

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>HRmax, beats/min</th>
<th>V˙O2max, ml·kg−1·min−1</th>
<th>V˙O2predicted %predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>28</td>
<td>170</td>
<td>66</td>
<td>174</td>
<td>74</td>
<td>166</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>178</td>
<td>71</td>
<td>176</td>
<td>82</td>
<td>196</td>
</tr>
<tr>
<td>3</td>
<td>16</td>
<td>185</td>
<td>69</td>
<td>187</td>
<td>73</td>
<td>143</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>168</td>
<td>56</td>
<td>173</td>
<td>80</td>
<td>158</td>
</tr>
<tr>
<td>5</td>
<td>20</td>
<td>179</td>
<td>63</td>
<td>182</td>
<td>72</td>
<td>147</td>
</tr>
<tr>
<td>6</td>
<td>20</td>
<td>171</td>
<td>60</td>
<td>189</td>
<td>67</td>
<td>137</td>
</tr>
<tr>
<td>7</td>
<td>18</td>
<td>183</td>
<td>69</td>
<td>180</td>
<td>81</td>
<td>162</td>
</tr>
<tr>
<td>8</td>
<td>22</td>
<td>170</td>
<td>72</td>
<td>194</td>
<td>70</td>
<td>146</td>
</tr>
<tr>
<td>9</td>
<td>18</td>
<td>170</td>
<td>66</td>
<td>187</td>
<td>69</td>
<td>137</td>
</tr>
<tr>
<td>10</td>
<td>21</td>
<td>183</td>
<td>74</td>
<td>184</td>
<td>71</td>
<td>146</td>
</tr>
</tbody>
</table>

Mean ± SD: 21.7 ± 176 ± 7 67 ± 7 183 ± 7 72 ± 6 15 ± 8

%Predicted values are from Jones and Campbell (15). HRmax, maximal exercise heart rate; V˙O2max, maximal exercise O2 uptake.
FLOW LIMITATION DURING EXERCISE IN CYCLISTS

Table 2. Pre- and postexercise lung function data from 10 subjects

<table>
<thead>
<tr>
<th>Preexercise</th>
<th>% Increase Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, liters</td>
<td>6.1 ± 0.8 (118 ± 9)</td>
</tr>
<tr>
<td>FEV₁, liters</td>
<td>5.2 ± 0.7 (120 ± 12)</td>
</tr>
<tr>
<td>FEF₂₅₋₇₅%, l/s</td>
<td>5.5 ± 1.1 (115 ± 23)</td>
</tr>
<tr>
<td>MVV₁₅, l/min</td>
<td>168 ± 21 (90 ± 15)</td>
</tr>
<tr>
<td>Pimax cmH₂O</td>
<td>127 ± 20 (117 ± 27)</td>
</tr>
<tr>
<td>PEFmax cmH₂O</td>
<td>178 ± 34 (141 ± 36)</td>
</tr>
</tbody>
</table>

Values are means ± SD with corresponding values expressed as %predicted value in parentheses; n = 8 subjects for % increase Postexercise. FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; FEF₂₅₋₇₅%, forced maximal midexpiratory flow; MVV₁₅, maximal voluntary ventilation in 15 s; Pimax and PEFmax, maximal inspiratory and expiratory pressures, respectively. For FVC, FEV₁, and FEF₂₅₋₇₅%, predicted values from Roca et al. (24); for MVV, predicted values from Kory et al. (16); for Pimax and PEFmax, predicted values from Morales et al. (20). *Statistically significant difference from preexercise value (P < 0.05).

Exercise test. The group exercised on average for 23 ± 6 min and reached a maximal power of 380 W (range: 320–440 W). All athletes terminated the exercise test voluntarily because of inability to maintain the level reached. Maximum O₂ consumption averaged 72 ml·kg⁻¹·min⁻¹, and the maximum heart rate averaged 182 beats/min (151 and 92% of predicted maximum, respectively). Table 3 shows the ventilatory variables at maximal exercise. Peak V˙E averaged 147 l/min (a mean of 88% of MVV₁₅). Breathing frequency increased gradually to a greater extent during the final phases of exercise, when V˙E had surpassed −40% of MVV₁₅. The increase in Vt was faster in the initial stages and reached a plateau when V˙E surpassed −40% of MVV₁₅. In some subjects, however, Vt decreased at the end of the test. Mean inspiratory flow (ratio of Vt to inspiratory time) increased gradually from the resting level until maximum effort. The ratio of inspiratory time and total breath time also increased gradually, rising from 0.36 at rest to 0.50 at the final stages of exercise.

Airflow limitation and EELV-EILV changes. In nine athletes, FL was absent at all levels of exercise, whereas in one (subject 2) it was present only at peak exercise, encompassing 26% of Vt. Figure 2 shows the individual tidal FV curves at peak exercise for the 10 subjects studied. One subject’s data (subject 8) was discarded for analysis of lung volume changes because of his inability to perform IC maneuvers correctly during heavy exercise. Figure 3 shows the mean changes in EELV and EILV data obtained for the remaining nine subjects, expressed as percentage of FVC at rest and at different exercise levels. During exercise, EELV decreased initially by 0.76 liter (range: 0.29–1.77 liter), representing 13% (range: 5–33%) of preexercise FVC. Later in the test it rose again, such that at maximal exercise it was not significantly different from the resting level. In all but one cyclist, the maximum decrease in EELV took place before 75% of maximum load had been reached. Eight subjects had the same pattern. The ninth subject for whom IC data were available (subject 10) did not exhibit the terminal increase of EELV. The EILV increased gradually in all athletes, to reach an average of 97% of FVC. EILV reached TLC in four subjects, one of whom was the subject with FL.

DISCUSSION

The importance of V˙E as a possible factor limiting the exercise capacity in highly fit individuals has not been clearly established. Three factors that potentially limit exercise capacity have been described: 1) diffusing capacity, 2) respiratory muscle aerobic capacity, and 3) expiratory FL (2, 4, 28). The question of whether well-trained normal young subjects attain FL during exercise has been addressed in previous studies as follows: using the technique of superimposing tidal FV with MEFV curves (method A) and based on comparison of the expiratory transpulmonary pressures achieved during exercise with the Pcrit at V˙max (method B) (8, 14). Based on method B, Grimby et al. (8) found that, during exercise on a cycle ergometer, one subject attained Pcrit, whereas two did not. In all three subjects, the corresponding tidal FV curves impinged on the MEFV curve, suggesting the presence of FL. This discrepancy was probably caused in part by errors due to thoracic gas compression because both tidal and maximal FV curves were based on changes in volume obtained by integration of flow measured at the mouth (12). Because at rest and during exercise there is little thoracic gas compression, such errors can be corrected by comparing the tidal FV curves with MEFV curves obtained in a body plethysmograph (modified method A). Alternatively, thoracic gas compression can be taken into account by making a graded series of FVC maneuvers at varying expiratory efforts and drawing an outer envelope to get a composite MEFV curve, which will show little effect of gas compression (1, 4). Johnson et al. (14) used modified method A in eight endurance runners together with method B. They found that, during exercise on a treadmill, four athletes had attained Pcrit during maximal exercise, whereas another four did not. However, the results obtained with modified method A were discrepant in the sense that the tidal FV curves impinged on the preexercise MEFV curves at exercise levels at which the transpulmonary pressure during expiration did not attain Pcrit, as shown in Fig. 1 of Ref. 14. If the analysis had been made by using the postexercise MEFV curves, all eight runners should have reached FL even before maximal exercise, whereas, according to method B, FL was present in only four of them. These discrepancies are probably due to volume and time-dependent changes in airway resistance and lung recoil during the maximal inspiration before the FVC maneuver (6, 18). This
implies that the maximal flows that can be reached during expiration depend on the volume and time history of the preceding inspiration. Because, by definition, the previous volume and time history vary between tidal and maximal inspiration, it follows that assessment of FL based on comparison of tidal FV with MEFV curves may lead to erroneous conclusions, even if errors due to thoracic gas compression are allowed for (5, 17, 18). No such problem pertains to the NEP method because the control and NEP test breath have similar volume and time histories. The NEP method also takes into account changes in respiratory mechanics during exercise (mainly resulting in increased maximal flows, as shown by the comparison of pre- and postexercise MEFV curves in our study and previous studies (13, 14)). Furthermore, the NEP method does not require a body plethysmograph because the intrathoracic pressure is the same during the control and NEP test breath (17). No study is yet available comparing results obtained with the modified method A with those obtained with the NEP method.

Although the results obtained with modified method A may be unreliable, those with method B probably provide a valid assessment of FL. Using this method, Johnson et al. (14) found evidence of FL in four out of eight endurance athletes, whereas we found that only one of our 10 competition cyclists (who reached O2 consumption levels similar to those achieved by the runners of Johnson et al.) became flow-limited at maximal exercise. This discrepancy may reflect differences due to 1) the different methodology used in assessment of FL, 2) the different sport practiced by the athletes, and 3) the different type of exercise performed while FL was assessed (treadmill and a discontinuous protocol for the runners of Johnson et al. vs. cycle ergometer and a continuous progressive protocol for our athletes). Different exercises involve differences in body posture, with different postural activity of respiratory muscles. Arm support brings about changes in the

Fig. 2. Individual tidal flow-volume curves from 10 athletes (subjects 1–10) at maximal exercise before (dashed lines) and with negative expiratory pressure (solid lines).

Fig. 3. Subdivisions of lung volume, expressed as %forced vital capacity (FVC) at rest and at different levels of maximal exercise (Wmax) in 9 cyclists. EILV and EELV, end-inspiratory and -expiratory lung volume, respectively; Vt, tidal volume; ▲, Average value; bars, SD. *Significant difference from rest (P < 0.05).
shape of the thoracic cage and provides anchorage to the shoulder girdle muscles, thus increasing the maximal sustainable vein (3), and could also increase the $V_{max}$ values. Furthermore, near the end of the test some of our athletes stood on the pedals and extended their necks, strategies that may affect expiratory flows (19, 23). Individual characteristics such as gender and age should also be taken into account. The elastic recoil of the lung decreases with age, promoting the development of FL during exercise in elderly, healthy trained individuals and limiting their exercise capacity (13). This is consistent with our finding of FL only in the oldest subject of the group. Expiratory FL involves increased transpulmonary pressure without further increase in expiratory flow. Although we have not measured the transpulmonary pressures in our cyclists, detection of expiratory FL by the NEP method meets both criteria, because NEP increases transpulmonary pressure (17, 25).

Another noteworthy observation from our study pertains to the behavior of EELV and EILV. The group strategy consisted of decreasing EELV at first and then increasing it to reach, and sometimes surpass, the resting level, while EILV gradually approached, and in four cases reached, TLC. Various studies have shown, like ours, that light-to-moderate exercise in healthy individuals generally induces a variable decrease in EELV (8, 9, 14, 17, 29), a condition that enhances inspiratory muscle function and promotes elastic work during expiration. During exercise, there is a balance between factors that promote increased EELV (gradually increased EILV and shorter expiratory time) and decreased EELV (progressive decrease in inspiratory muscle braking and increase in expiratory muscle activity). Our findings suggest that, at the lower exercise levels, this balance favors a lower EELV, whereas with intense and prolonged exercise a higher EELV is attained. It has been suggested that, in the presence of FL, increasing ventilatory demand causes dynamic hyperinflation because of premature reflex ending of expiration due to dynamic airway compression (22). Changes in EELV similar to those seen in our subjects were previously found by Johnson et al. (14), who suggested that the terminal increase in EELV was related to the appearance of FL, as detected by the FV curve superimposition method. However, nine of our 10 subjects did not exhibit FL, and the remaining one showed FL only at peak exercise. Although FL per se does not explain the terminal increase in EELV observed in our study, it is possible that there are reflex mechanisms, which are triggered when an individual approaches FL, to avoid dynamic compression. Younes and Kivinen (29) studied healthy young individuals who were not competitive athletes but who performed a truly maximal exercise test and found changes in EELV and EILV similar to those in our study. Although FL was not assessed, it is unlikely that it occurred in their subjects because $V_e$ only reached 81 l/min (range: 59–101 l/min). Younes and Kivinen (29) considered that expiratory muscle fatigue was an unlikely explanation for the terminal increase in EELV because the expiratory pressures attained were rather low. It is unlikely that falsely low ICs were recorded at high levels of exercise in our athletes, leading to a false terminal increase in EELV. Measurement of IC has been previously shown to be a reliable method for assessing EELV even in individuals with pulmonary disease (2, 9, 11, 13, 14, 17, 29). The fact that eight of nine of our subjects showed similar changes also argues against the possibility of misleadingly low IC data having been collected.

The present results indicate that, in young, male competition cyclists, there is a small volitional ventilatory reserve during maximal exercise because maximal $V_e$ amounted, on average, to 88% of MVV. In endurance runners, Johnson et al. (14) have shown that increasing the stimulus to breathe during maximal exercise by inducing either hypercapnia or hypoxemia failed to increase $V_e$, inspiratory pressure, or expiratory pressure. Whether this is also the case in competition cyclists remains as yet to be determined.

We conclude that FL is not commonly attained in top-performing, young male cyclists even during maximal exercise. Nevertheless, EELV, which decreases at the start of exercise, increases gradually thereafter, eventually returning to resting levels. This terminal increase of EELV is caused by factors other than the presence of expiratory FL.

This investigation was partially funded by a grant from the Societat Catalana de Pneumologia 1997.

Address for reprint requests: J. Sanchis, Dept. de Pneumologia, Hospital de la Santa Creu i de Sant Pau, Av. Sant Antoni M. Claret, 167, 08025 Barcelona, Spain.

Received 5 January 1998; accepted in final form 4 November 1998.

REFERENCES


