Effect of Cold Air on Exercise Capacity in COPD: Increase or Decrease?

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*Chest* 1998;113;1560-1565
DOI 10.1378/chest.113.6.1560

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Effect of Cold Air on Exercise Capacity in COPD*

Increase or Decrease?

Heikki Koskela, MD; Jussi Pihlajamäki, MD; Heikki Pekkarinen, MD; and Hannu Tukiainen, MD

Study objective: To clarify the effect of cold air on exercise capacity in COPD.

Design: Cycle ergometer tests under different environmental conditions.

Setting: Pulmonary function laboratory and an environmental chamber at a university hospital.

Participants: Eighteen patients with stable COPD; 14 completed the study.

Interventions: A preliminary cycle ergometer test followed by two incremental, symptom-limited cycle ergometer tests, one at 24°C and the other at −20°C.

Measurements: On the first study day: arterial blood gas analysis, 12 to 15 s maximal voluntary ventilation, maximal expiratory flow-volume curves before and 1 h after inhalation of 80 µg of ipratropium bromide, and diffusion capacity of the lung. During the exercise challenges: spirometric indices, minute ventilation (Ve), oxygen consumption (Vo2), carbon dioxide production (VCO2), facial skin temperature, and heart rate. The feeling of dyspnea was assessed with a visual analogue scale.

Results: The maximal work load was 87.5±7.3 W at −20°C compared with 96.4±6.9 W at 24°C (p<0.05). Accordingly, the exercise duration was shorter in the cold. Exercise dyspnea was more severe in the cold at equal work loads. The shortening of exercise duration induced by cold air correlated with the enhancement of exercise dyspnea. Furthermore, cold air cooled the facial skin and induced immediate bronchoconstriction. Ve, Vo2, VCO2, and heart rate did not differ between the warm and cold challenges.

Conclusions: Cold air decreases exercise capacity in COPD, probably by increasing exercise dyspnea.

(CHEST 1998; 113:1560-65)

Key words: bronchoconstriction; cold climate; COPD; dyspnea; exercise; lung diseases, obstructive; reflex/physiology; symptom limitation

Abbreviations: DC02=diffusion capacity of the lung; VAS=visual analogue scale; VCO2=carbon dioxide output; Ve=minute ventilation; Vo2=oxygen consumption; WL=work load

When exposed to very low environmental temperatures, patients with COPD commonly experience dyspnea while performing tasks that they are well capable of doing at more moderate temperatures. For this reason, some patients are completely unable to take part in outdoor activities at temperatures such as those encountered in the Scandinavian winter. To our knowledge, there has been only one previous study about the effect of cold air on exercise capacity in patients with COPD. In that study, breathing subfreezing air instead of warm air during exercise was found to increase exercise capacity and decrease exercise dyspnea by inducing relative hypoventilation.1 It is noteworthy that in the study by Spence et al,1 only the respiratory tract was exposed to the cold. However, when an individual exercises in cold weather, the facial skin is also exposed. We have previously shown that cooling of the facial skin induces an increase in ventilation and reflex bronchoconstriction in patients with COPD.2 Thus, it seems that cooling of the respiratory tract increases exercise capacity, but cooling of the facial skin might have opposite effects. The present study was carried out to clarify the total effect of cold air on exercise capacity in patients with COPD: Does it cause an increase, a decrease, or does it have no effect at all?
Subjects

Eighteen stable patients who met the American Thoracic Society criteria for chronic bronchitis and/or emphysema participated in the study. Entry criteria included a smoking history of at least 10 pack-years, FEV₁ less than 75% of the predicted value, and the absence of other disorders likely to affect exercise. They were not selected on any anamnestic grounds; ie, both patients with and without a history of cold weather-associated dyspnea were included. Table 1 shows the basic characteristics of the 14 subjects who completed the study. Of the 14 patients, 11 used inhaled corticosteroids, 10 used inhaled anticholinergic drugs, nine used inhaled β₂ sympathomimetic drugs, and three used theophylline preparations. All gave their informed consent for participation in the study, which was approved by the ethics committee of the University of Kuopio.

The patients were not allowed to take any inhaled drugs for 12 h nor any oral theophylline preparations for 24 h prior to each visit, and were instructed not to smoke cigarettes for 12 h. None of the patients had experienced any exacerbation of their disease during the 1-month period before the study.

Experimental Design and Protocol

On the first day, the patients were interviewed and underwent the following lung function tests: arterial blood gas analysis in a supine position after 15 min of rest (Model 1302; Instrumentation Laboratory; Milan, Italy), 12 to 15 s maximal voluntary ventilation measured with a pneumotachograph spirometer (Medikro 909; Medikro; Kuopio, Finland), three maximal expiratory flow-volume curves before and 1 h after inhalation of 80 μg of inhaled ipratropium bromide (Atrovent inhalation powder; Boehringer Ingelheim; Ingelheim, Germany), and diffusion capacity of the lung (Dco) by the single breath method (2200 Pulmonary Function Laboratory; SensorMedics; Aachen, Germany). To exclude patients with underlying heart disease and to accustom the patients to the use of the cycle ergometer and the visual analogue scale (VAS) described below, a preliminary exercise test was performed with a mechanically braked cycle ergometer (Monark 818E; Monark-Crescent; Vansbro, Sweden). The exercise protocol was similar to the later exercise tests with the following exceptions: During the preliminary test, the arterial saturation of oxygen was measured continuously with a pulse oximeter (Ohmeda Biox 3700e; Ohmeda; Tokyo, Japan) but, because of technical difficulties due to very cold ambient temperatures, this variable could not be measured during the later exercise tests. In addition, the inspiratory air volume and gas concentrations were not measured during the preliminary exercise test.

After the first evaluation day, two incremental cycle ergometer tests were performed in random order on separate days, always at the same time of day, separated by an average of 8 days. The only difference between the two tests was the temperature in the environmental chamber: 24.5±0.9°C (mean±SD) and 35.7±4.9% relative humidity on the warm day vs −19.9±0.1°C on the cold day (the absolute water content of subfreezing air is near zero irrespective of the relative humidity). On the warm day, the patients exercised wearing a T-shirt and light trousers. On the cold day, they wore arctic clothing appropriate for a cold, wintry day. They wore woollen hats and gloves, but their faces were always uncovered; the face was the only exposed area of the skin.

The Incremental Cycle Ergometer Test

Prior to the test, the facial skin temperature was measured outside the environmental chamber with a thermocouple attached to the patient’s right cheek with tape (GTH 1200 Digitathermometer; Greisinger Electronic; Regenstauf, Germany). At least three maximal expiratory flow-volume curves were determined with the spirometer. The intensity of dyspnea was assessed with the VAS described below. ECG leads (Schiller Cardiovit 3; Schiller AG; Baar, Switzerland) were attached. The patient dressed and then entered the environmental chamber. Initially, patients sat in the chamber for 3 min while breathing tidally through the nose. At this stage, two maximal flow-volume curves were determined using the transmural breathing system of the chamber. The cycle ergometer was always placed in the chamber just prior to the exercise challenge in order to minimize freezing of the cycle parts.

### Table 1—Patient Characteristics and Pulmonary Function Test Results*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, yr/sex</th>
<th>Pred HR (per min)</th>
<th>FEV₁, L</th>
<th>Reversib, L</th>
<th>MVV, L/min</th>
<th>Dco, % pred</th>
<th>PaO₂, mm Hg</th>
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<tbody>
<tr>
<td>1</td>
<td>70/M</td>
<td>170</td>
<td>1.55</td>
<td>0.00</td>
<td>59</td>
<td>65</td>
<td>78</td>
</tr>
<tr>
<td>2</td>
<td>65/M</td>
<td>172</td>
<td>2.45</td>
<td>-0.14</td>
<td>116</td>
<td>120</td>
<td>79</td>
</tr>
<tr>
<td>3</td>
<td>55/M</td>
<td>178</td>
<td>1.56</td>
<td>0.40</td>
<td>60</td>
<td>53</td>
<td>82</td>
</tr>
<tr>
<td>4</td>
<td>72/M</td>
<td>169</td>
<td>0.83</td>
<td>0.14</td>
<td>32</td>
<td>45</td>
<td>77</td>
</tr>
<tr>
<td>5</td>
<td>35/M</td>
<td>178</td>
<td>1.86</td>
<td>0.24</td>
<td>83</td>
<td>45</td>
<td>77</td>
</tr>
<tr>
<td>6</td>
<td>65/M</td>
<td>172</td>
<td>1.40</td>
<td>0.04</td>
<td>45</td>
<td>54</td>
<td>74</td>
</tr>
<tr>
<td>7</td>
<td>44/M</td>
<td>183</td>
<td>2.77</td>
<td>0.29</td>
<td>119</td>
<td>61</td>
<td>71</td>
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<tr>
<td>8</td>
<td>68/M</td>
<td>171</td>
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<td>9</td>
<td>58/M</td>
<td>176</td>
<td>1.61</td>
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<td>85</td>
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<tr>
<td>10</td>
<td>61/M</td>
<td>174</td>
<td>1.65</td>
<td>-0.08</td>
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<td>80</td>
<td>70</td>
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<tr>
<td>11</td>
<td>58/F</td>
<td>176</td>
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<td>0.11</td>
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<tr>
<td>12</td>
<td>67/M</td>
<td>176</td>
<td>1.17</td>
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<td>118</td>
<td>63</td>
</tr>
<tr>
<td>13</td>
<td>61/M</td>
<td>174</td>
<td>0.84</td>
<td>0.15</td>
<td>43</td>
<td>52</td>
<td>68</td>
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<tr>
<td>14</td>
<td>69/M</td>
<td>171</td>
<td>0.76</td>
<td>0.41</td>
<td>34</td>
<td>56</td>
<td>72</td>
</tr>
</tbody>
</table>

Mean±SD 61±7 174±4 1.53±0.57 0.16±0.17 62±28 72±24 75±7

*Pred HR = maximal predicted heart rate, calculated as 205−(0.5×age); reversib = improvement of FEV₁ after 80 μg of inhaled ipratropium bromide; MVV = maximal voluntary ventilation; Dco = diffusion capacity of the lung, as a percentage of predicted.5

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The patient sat on the cycle and started to breathe the ambient air orally through the mouthpiece wearing nose clips. For the first minute of recording, the patient sat without pedalling. The initial work load (WL) was 25 W, subsequently increased in increments of 25 W every 3 min until exhaustion. The maximal WL was defined as the highest WL maintained by the patient for at least 1 min. Pedalling rates were maintained at 60 revolutions/min by listening to a metronome. The time from the start of the pedalling to exhaustion was also measured. The heart rate (determined from the ECG), minute ventilation (Ve), oxygen consumption (Vo2), carbon dioxide output (VCO2) and facial skin temperature were measured throughout the exercise challenge. Heart rates at the end of each 3-min WL period and the mean Vo2, VCO2, and Ve from the last 30 s of each WL period were used for analysis. The intensity of dyspnea was assessed with the VAS at rest and during the last minute of each WL period.

After exercise, analysis of the expired air was discontinued. During the recovery period, the patient sat in the environmental chamber while tidally breathing the ambient air. At 3, 6, 10, and 15 min after the exercise challenge, two expiratory flow-volume curves were obtained using the transmural breathing facility of the chamber.

**Visual Analogue Scale**

A 210-mm line was placed horizontally on a page. Above the line the heading “Dyspnea” was printed; the left end of the line represented “not at all” and the right end “extremely severe” dyspnea. The patient was asked to make a vertical marking on the line with a pencil. Its distance from the left end of the line was measured to a resolution of 1 mm. A new page was used each time so that the patient did not know the locations of the previous markings.

**Measurement of Vo2, VCO2 and Ve**

The valve of the mouthpiece (Y-valve no. 720 068; Erich-Jaeger GmbH, Wurzburg, Germany) allowed no mixing of inspired and expired gas. The expired gas was directed to a large-bore tube 3 m long. The tube passed through the wall of the environmental chamber to the ergospirometer (Medikro 919 Ergospirometer), which was located outside the chamber at room temperature. The gas analyzers were calibrated before each test with appropriate mixtures of oxygen and carbon dioxide.

Because it was suspected that cold air within the environmental chamber might cool the expired air and thus lower the temperature inside the mixing chamber of the ergospirometer, a thermocouple was installed in the mixing chamber. These temperatures were continuously monitored during the exercise challenges. It was noted that the mean temperature in the mixing chamber during the cold exercise challenge was not more than 1.0°C lower than that during the warm challenge at equal WLs.

To determine whether differences in the temperature inside the environmental chamber might affect the air volume measurements (by altering the temperature in the mixing chamber of the ergospirometer as described above), the ergospirometer was volume calibrated by passing warm, moist air through the tube with a 3-L pump while keeping the tube at −20°C or at +20°C. It was noted that the volume recordings did not differ significantly for the two conditions.

Measurements of gas volumes relate to conditions in the lung, where the gas is at body temperature and pressure and is saturated with water vapor. The measurements of Vo2 and VCO2 relate to conditions where the temperature is 0°C, pressure is 1 atm, and the air is dry.

**Statistical Analysis**

All data are expressed as mean±SEM unless otherwise stated. The Wilcoxon signed-rank test and Spearman rank correlation coefficient were used to establish statistical significance. A p value of <0.05 was accepted as the level of significance.

**RESULTS**

Four patients did not complete the study: one because of a deep venous thrombosis in the calf, one because of lymphoma, one for social reasons, and one because the preliminary exercise challenge provoked an attack of atrial fibrillation. Only the 14 patients who completed the study were included in the analysis. During the preliminary exercise test, 11 of the 14 patients showed a significant reduction (>3%) in arterial saturation of oxygen. Table 1 shows the results of the lung function tests performed on the first study day. The interviews conducted before the challenges revealed that 11 of the 14 patients had a history of an enhanced exercise-associated dyspnea in cold weather. These patients were also asked how cold the weather must be to exacerbate their dyspnea. The mean response was −15.0°C (range, −10 to −25°C).

The comparison of the two exercise challenges revealed that the maximal WL was lower in the cold: 87.5±7.3 W vs 96.4±6.9 W in the warm (p<0.05). Accordingly, the duration of exercise was shorter in the cold environment, 596±54 s vs 642±49 s in the warm environment (p<0.05). The dyspnea intensity was more severe during exercise in the cold than during exercise in the warm (Table 2 and Fig 1). The difference between the VAS values for the highest equal WL completed in both challenges correlated significantly with the difference in the duration of exercise (r=0.54; p<0.05).

In addition, the cold environment cooled the skin of the face (Fig 2, top left) and induced an immediate bronchoconstriction (Fig 2, top right). Fig 2 shows the individual responses in the warm (bottom left) and cold (bottom right). Inside the chamber, before exercise, FEV1 was 0.16±0.03 L less in the cold than in the warm (10.0±2.0%). Bronchodilation was observed shortly after both exercise challenges. Comparison of the postexercise FEV1 values with those recorded immediately before exercise within the environmental chamber revealed no exercise-induced bronchoconstriction, except in patient number 3 (Fig 2, bottom right).

The FEV1 value recorded immediately prior to exercise was correlated with the duration of the exercise challenge (r=0.69; p<0.01). However, the cold air-induced decrease in FEV1 did not correlate with either the cold air-induced decrease in exercise duration or the reduction in maximal WL.
No significant differences in $V_e$, $V_O_2$, $V_CO_2$, or heart rate were found between the two exercise challenges (Table 2).

**DISCUSSION**

This study demonstrates that cold air decreases exercise capacity in patients with stable COPD. In our exercise challenges, the maximal WL achieved was lower and the duration of exercise shorter at $-20^\circ$C than at $24^\circ$C. In addition, the patients experienced more dyspnea during exercise in the cold. The results of this experimental study corroborate our clinical impressions and were in agreement with the patients' own experience: 11 of the 14 patients who completed the study had a history of an enhanced exercise-associated dyspnea in cold weather. Since the mean threshold temperature for this enhancement in exercise dyspnea was as low as $-15^\circ$C, this problem mainly concerns patients living in a very cold climate, such as that found in Scandinavia and Canada.

Although the results corroborate our clinical impressions they are diametrically opposed to the findings of Spence et al,1 who have, to our knowledge, conducted the only previous study on this issue. However, the opposite results can be explained by methodologic differences.

One important methodologic feature of the study by Spence et al1 is that they exposed only the respiratory tracts to the cold, while the participants' faces remained warm. This is not a physiologic situation. We tried to mimic the situation of everyday life by conducting the exercise challenges in an environmental chamber, with patients dressed lightly under warm conditions and clad warmly for the cold conditions. Thus, both the face and the respiratory tract were exposed. Our study, in turn, may be criticized in that there were two independent variables: ambient temperature and clothing. However, as $V_O_2$ and heart rates were similar in the cold and the warm at equal WLs, the additional clothing in the cold probably did not influence exercise capacity. Previous investigators have come to similar conclusions about the effect of arctic clothing on exercise capacity.7

From the viewpoint of methodologic differences between our study and that of Spence et al,1 it is possible to explain why cold air decreased exercise ventilation in the previous study but did not affect it in our study. Activation of cold-sensitive receptors in the upper airways is capable of inhibiting ventilation,8-11 and this probably occurred in the previous study. In contrast, activation of cold-sensitive receptors in the skin is capable of stimulating ventilation,12-15 and this probably took place in our study.

**Figure 1.** The VAS values for the highest equal WL completed in both challenges in each individual. Warm=exercise challenge at warm ambient temperature; Cold=exercise challenge at cold ambient temperature. The horizontal lines indicate the mean values. $p<0.01$.  

**Table 2—Physiological Variables Measured During the Exercise Challenges**

<table>
<thead>
<tr>
<th>WL</th>
<th>No. of Patients Completing</th>
<th>HR, per min</th>
<th>$V_e$, L/min</th>
<th>$V_O_2$, L/min</th>
<th>VAS, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Warm</td>
<td>Cold</td>
<td>Warm</td>
<td>Cold</td>
</tr>
<tr>
<td>Rest</td>
<td>14</td>
<td>88±4</td>
<td>87±4</td>
<td>12±1</td>
<td>12±1</td>
</tr>
<tr>
<td>25 W</td>
<td>14</td>
<td>96±3</td>
<td>98±3</td>
<td>22±1</td>
<td>22±1</td>
</tr>
<tr>
<td>50 W</td>
<td>12</td>
<td>106±4</td>
<td>104±4</td>
<td>29±2</td>
<td>30±2</td>
</tr>
<tr>
<td>75 W</td>
<td>9</td>
<td>114±2</td>
<td>116±4</td>
<td>36±2</td>
<td>35±2</td>
</tr>
<tr>
<td>100 W</td>
<td>3</td>
<td>125±4</td>
<td>124±7</td>
<td>40±3</td>
<td>40±7</td>
</tr>
<tr>
<td>Hi iso W</td>
<td>14</td>
<td>117±4</td>
<td>118±4</td>
<td>37±2</td>
<td>37±3</td>
</tr>
<tr>
<td>Max</td>
<td>14</td>
<td>128±4</td>
<td>126±4</td>
<td>45±3</td>
<td>45±3</td>
</tr>
</tbody>
</table>

*Data are expressed as mean±SEM. HR=heart rate; Hi iso W=highest WL completed in both challenges; Max=maximal value.  
1$p<0.05$ vs exercise at warm ambient temperature.  
2$p<0.01$ vs exercise at warm ambient temperature.
in addition to activation of the receptors in the upper airways. Our results suggest that under physiologic conditions, the inhibitory effects nullify the stimulative effects and the sum result is that ventilation does not change.

Methodologic differences also explain why we observed a definite cold air-induced bronchoconstriction but Spence et al1 did not. In our study, the cold air-induced bronchoconstriction was fully developed before the patients started to exercise, during resting nasal breathing. This bronchoconstriction was probably a reflex response to the cooling of the facial skin.6 Exercise, however, did not induce any further bronchoconstriction, even though the inhaled air was very cold and dry: an identical finding was noted in the previous study. These findings support the view that cooling of the face is the most important stimulus for bronchoconstriction related to cold weather in patients with COPD, and that direct airway effects of cold air are of lesser importance.2 In the future, this issue could be studied more precisely by conducting exercise challenges in a cold environment both with and without protective clothing on the face.

The consequences of the methodologic differences between our study and that of Spence et al1 explain why cold air did not increase exercise capacity in our study as it did in the previous study: relative hypoventilation, the probable reason for the cold-induced increase in exercise capacity in the previous study, did not take place in our study. Another methodologic difference, bronchoconstriction induced by facial cooling in our study, provides an indication why exercise capacity decreased in the cold during our study. However, the degree of bronchoconstriction did not correlate with the decrease in the exercise capacity. Thus, we cannot simply claim that this bronchoconstriction is the reason for the decrease in exercise capacity.

What was the mechanism underlying the decrease in exercise capacity in the cold? To understand this, it must be pointed out that most patients in the present study stopped exercise at submaximal heart rates and submaximal ventilation. This can be shown by comparing the maximal predicted heart rates and maximal voluntary ventilation values in Table 1 with the values achieved during the exercise challenges in Table 2. Thus, symptoms, and not physiologic factors, usually limited the exercise capacity in both challenges. The same phenomenon has been ob-

Figure 2. Top left: the temperature of the facial skin. Top right: the mean percentage changes in FEV1 from baseline. Bottom left: the individual responses in the warm. Bottom right: the individual responses in the cold at various stages of the challenges. Closed circles indicate exercise challenge at warm ambient temperature; open circles, exercise challenge at cold ambient temperature. Out=outside the chamber, at rest; In=inside the chamber, at rest; E1= during the first min of exercise; E2= during the highest WL; 3, 6, 10, 15: time (min) after the end of exercise, still inside the chamber. The bold line on the stage axis indicates the period of exercise. *p<0.05, between-challenge comparison. **p<0.01, between-challenge comparison.
served in many previous studies in patients with COPD.16,17 If this is the case, an increase in dyspnea should decrease exercise capacity. Indeed, this was confirmed in the present study: the cold-induced increase in dyspnea at the largest equal WL was correlated with the cold-induced reduction in exercise duration.

A cold air-induced increase in exercise dyspnea thus seems to be an essential factor in cold-induced decrease in exercise capacity, and it deserves further discussion. It has been thought that exercise dyspnea in COPD is linked with inspiratory muscle loading due to dynamic hyperinflation. This latter phenomenon, in turn, is probably a consequence of the expiratory airflow limitation in COPD.10 Based on this, we speculate that the facial cooling-induced airway narrowing may have enhanced dynamic hyperinflation and increased the inspiratory muscle load, thereby increasing exercise dyspnea. However, confirmation of this theory would require a new study in which indices of dynamic hyperinflation and inspiratory muscle function were recorded before and during exercise at various ambient temperatures.

Some of the limitations of our study warrant discussion. The small population size reflects our difficulties in recruiting suitable subjects for the following reasons. First, because management of any severe cardiac attacks would have been very difficult in a freezing environmental chamber because the patients were heavily dressed, we had to exclude subjects with cardiac diseases. However, cardiac disorders are very common in patients with COPD. Second, the fear of severe dyspnea in cold environment was so strong among many possible candidates that they declined to participate. Thus, there was a selection bias in our study, possibly favoring patients with mild forms of COPD. One might also suspect that many of our patients had an asthmatic component in their disease because most used inhaled corticosteroids. However, we do not believe that to be the case, as only one patient showed a clear exercise-induced bronchoconstriction (patient no. 3, the youngest of the group, who also showed a significant improvement in FEV1 after ipratropium bromide; see Fig 2, bottom right). More probably, the usage of inhaled corticosteroids merely reflects the COPD treatment policy in our hospital. Finally, one limitation of our study is the lack of a healthy control group. However, we believe that this does not affect the main conclusions.

In conclusion, this study shows that cold air decreases exercise capacity in COPD, probably by increasing exercise dyspnea. Further studies are required to identify the mechanisms of cold air-induced increase in exercise dyspnea.

REFERENCES


3 American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. Am Rev Respir Dis 1987; 136:225-44


6 Koskela H, Tukiainen H. Facial cooling, but not nasal breathing of cold air, induces bronchoconstriction: a study in asthmatic and healthy subjects. Eur Respir J 1995; 8:2088-93


14 Mekjavic IB, Bligh J. The increased oxygen uptake upon immersion: the raised external pressure could be a causative factor. Eur J Appl Physiol 1989; 58:556-62


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