Exercise-induced hypoxemia and cardiac arrhythmia in cystic fibrosis

Katharina Ruf *, Helge Hebestreit

University Children's Hospital, Julius-Maximilians-Universität Würzburg, Germany

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Abstract

Background: Physical activity has become part of the therapy of patients with cystic fibrosis (CF) despite possible risks. The objectives of this study were to identify predictors of hypoxemia and to search for cardiac arrhythmia during exercise.

Methods: The data of 75 patients (12 to 41 years old) with CF who underwent a standardized incremental exercise test on a cycle ergometer was analyzed. Oxygen saturation (SpO2) and ECG were monitored. The results were related to spirometric and SpO2 measurements at rest.

Results: During exercise, 17 patients suffered from significant desaturations (SpO2 < 90%). SpO2 at peak exercise was independently related to SpO2 at rest and 1/FEV1 (multiple regression R² = 0.63). Five patients demonstrated ventricular arrhythmias during exercise. No unambiguous prediction of exercise-induced hypoxemia or cardiac arrhythmia was possible.

Conclusion: In order to detect all patients with exercise-induced hypoxemia and cardiac arrhythmia, an incremental exercise test to volitional fatigue must be performed.

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Keywords: Cardiac arrhythmias; Cardiopulmonary exercise testing; Cystic fibrosis; Hypoxemia; Oxygen saturation

1. Background

Physical activity and exercise conditioning have become increasingly important in the treatment of patients suffering from cystic fibrosis (CF). Several studies have shown that these patients benefit from physical activity with respect to exercise capacity, pulmonary function, muscle mass, and quality of life [1–4]. Furthermore, physical fitness is associated with a higher life expectancy [5] and better performance in professional life [6].

Many potential risks, though, have been associated with physical exertion in patients with CF, such as exercise-induced hypoxemia and bronchoconstriction, pneumothorax, hypoglycemia, rupture of oesophageal varices or spleen, and fractures [7,8]. In addition, pulmonary exacerbations have been reported after winter sport [9]. Of all the risks mentioned, exercise-induced hypoxemia has been studied most systematically. Using pulse oximetry, Henke et al. [10] showed that 9 of 91 patients (7 to 35 years of age) became hypoxemic during an exercise test up to volitional fatigue. Although patients with advanced pulmonary disease were most at risk for exercise-induced hypoxemia, no reliable prediction of the decrease in oxygen saturation could be made from the patients’ pulmonary function at rest. In a smaller sample of 21 patients, Lebecque et al. [11] found that six patients with a FEV1 between 17 and 28% predicted and with an impaired single-breath diffusion capacity for carbon monoxide showed a significant exercise-induced hypoxemia (< 90%) while the remaining patients with a FEV1 greater 36% and a diffusion capacity greater 83% did not.

Studies validating pulse oximeters to assess oxygen saturation during exercise have demonstrated that some systems have a large measurement error [12]. Thus, by using a precise and validated pulse oximeter, the relationship between resting pulmonary function and oxygen saturation during exercise might become much clearer.

Studies in patients with chronic obstructive pulmonary disease (COPD) have shown that low oxygen saturation during exercise may predispose to cardiac ventricular dysrhythmia [13], possibly due to cor pulmonale, acid–base disturbances and
myocardial ischemia during exercise [14,15]. Surprisingly, we could not find any report of exercise-induced arrhythmias in patients with CF, although the same risk factors observed in COPD have also been found in CF. There is, however, evidence for cardiac involvement with manifestation during exercise in CF. De Wolf et al. [16] studied left ventricular perfusion during exercise in 18 patients with CF. Among these, six showed defects in perfusion. Interestingly, one of these six patients did not suffer from oxygen desaturation during exercise, whereas five developed exercise-induced hypoxemia. In four of the six patients, cardiac perfusion was also evaluated at rest and was found to be normal. The authors speculated that either an insufficient blood supply to the myocardium and/or regional myocardioopathy were responsible for the perfusion anomalies. Further reports indicate fibrotic changes in the myocardium of patients with CF [17,18]. Anyhow, neither De Wolf et al. [16] nor any of the other studies we could find report on arrhythmias during exercise in CF. Despite this missing evidence, we hypothesized that exercise-induced cardiac dysrhythmia does occur in patients with CF and that exercise-induced hypoxemia would be a risk factor.

Consequently, the objective of this retrospective study was first to identify the prevalence of exercise-induced hypoxemia in a group of patients with CF varying in age and disease severity using validated methodology. We then intended to identify predictors of exercise-induced hypoxemia. Finally, we especially searched for the prevalence of exercise-induced cardiac dysrhythmia and tried to identify potential risk factors.

2. Methods

In order to investigate the prevalence of exercise-induced hypoxemia and dysrhythmia, the exercise tests of 75 CF patients (36 females and 39 males) between 12 and 41 years of age were retrospectively analyzed. These patients were tested between 1999 and 2007 using identical equipment and testing protocols for yearly routine check-ups (n=18) or as part of scientific studies (n=57). The written informed consent of all patients was obtained; the scientific studies had been approved by the local ethics committee. Some of these patients’ data have been included in another publication [4].

On the testing day, the patients first completed a lung function test, assessing forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC), residual volume (RV), and total lung capacity (TLC) (Masterscreen Body, Jaeger, Würzburg, Germany). Data were expressed as % predicted. The subjects’ characteristics are shown in Table 1.

After familiarizing the patients with the exercise testing equipment, incremental exercise tests were carried out according to the Godfrey protocol [19] on a cycle ergometer (Ergomedic 834 E, Monark, Sweden). Work rate was selected depending on the height of the patient: patients with a height between 120 and 150 cm started with 15 W, patients taller than 150 cm started with 20 W. Work rate was increased minute-by-minute by 15 W or 20 W, respectively, up to volitional fatigue. Physical working capacity was determined as the highest work rate performed for 1 min and expressed in % predicted [19].

During exercise, ventilation and gas exchange data were obtained breath-by-breath using a metabolic cart, and averaged every 15 s (CPX/D, MedGraphics, St. Paul, MN, USA). Peak oxygen uptake (VO₂peak) was taken as the highest oxygen uptake over two consecutive 15-s intervals during the test and expressed in % predicted [20]. The respiratory exchange ratio (RER) at peak exercise was determined for the same time interval as VO₂peak.

Before, during and at least 3 min following exercise, a 12-lead ECG was recorded (custocard m, Ottobrunn, Germany). The chest electrodes were placed in standard positions, the limb electrodes were placed suprACLavicular near the shoulders and suprACLavial [21]. Ectopic cardiac beats and cardiac arrhythmias were searched for by visually inspecting the entire ECG. Exercise-induced cardiac arrhythmias were defined as ectopic beats occurring 1) only during exercise and 2) with increasing frequency when exercise intensity rose.

Oxygen saturation (SpO₂) was monitored before and during the exercise test using pulse oximetry with a forehead sensor (Nellcor Reflectance oxygen sensor RS10, Nellcor Puritan Bennet Inc., Pleasanton, CA, USA). By comparison with arterial blood samples, Yamaya et al. [12] could show that this system revealed accurate measurements. Exercise-induced oxygen desaturation was defined as a drop in SpO₂ of more than 4% from resting values to peak exercise [22]. Significant hypoxemia was assumed if SpO₂ fell below 90%, since it has been suggested to limit the intensity of activities in patients who experience SpO₂ values below this threshold [23].

In our CF centre, echocardiography is performed routinely once a year. Hence, many but not all of the patients enrolled in this study had echocardiographic examinations around the time of the exercise test. We were retrospectively able to analyze the echocardiographies of 44 patients, among these all those patients who had shown ectopic beats in the ECG during the exercise-testing.

3. Data analysis

Differences between groups of patients (male vs. female; arrhythmia vs. no arrhythmia) were assessed by Student t-tests. Correlation analyses according to Pearson were used to assess

<table>
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<th>Table 1: Subjects’ characteristics</th>
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<tr>
<td><strong>Female patients (n=36)</strong></td>
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<td><strong>Male patients (n=38)</strong></td>
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<tr>
<td>Age (years)</td>
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<td>19.8±6.9 (12.0–41.6)</td>
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<td>21.8±6.9 (12.3–41.3)***</td>
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<td>Height (m)</td>
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<td>1.61±0.73 (1.41–1.76)</td>
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<td>1.70±0.12 (1.38–1.90)***</td>
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<td>Weight (kg)</td>
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<td>52.1±9.3 (36.5–73.8)</td>
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<td>59.2±14.7 (30.5–95.1)**</td>
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<td>FVC (%pred)</td>
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<td>80.3±21.4 (35.8–116.3)</td>
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<td>79.7±23.7 (33.1–122.2)</td>
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<td>FEV1 (%pred)</td>
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<tr>
<td>67.6±25.4 (22.3–121.7)</td>
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<td>63.0±26.0 (23.2–104.1)</td>
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<td>RV (%pred)</td>
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<td>152.3±61.3 (64.0–306.3)</td>
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<td>168.1±68.3 (63.5–360.0)</td>
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<td>RV/TLC (%)</td>
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<td>39.9±12.6 (18.8–60.5)</td>
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<td>39.7±12.2 (17.4–73.0)</td>
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<td>SpO₂ at rest (%)</td>
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<td>97.3±2.2 (92–100)</td>
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<td>97.2±2.2 (90–100)</td>
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Data is presented as mean±standard deviation (range). Abbreviations: FVC — forced vital capacity; FEV1 — forced expiratory volume in 1 s; RV — residual volume; TLC — total lung capacity; %pred — % of predicted. **Significantly different from female patients (p<0.01); ***Significantly different from female patients (p<0.001).
relationships between the exercise-related drop in SpO₂ (or SpO₂ at peak exercise) and variables obtained at rest. A multiple linear regression analysis entering non-exercise data as possible predictors was used to establish an equation to estimate SpO₂ at peak exercise. The following parameters were tested as correlates of the exercise-induced change in SpO₂, or peak-exercise SpO₂, and were allowed to enter the multiple linear regression model as independent predictors of peak-exercise SpO₂: age, height, weight, SpO₂ at rest, FEV₁, FVC, MEF₂₅, RV, TLC, and RV/TLC. For the multiple linear regression model, gender and 1/FEV₁ were also assessed as possible predictors of peak-exercise SpO₂.

Statistical analyses were performed using BMDP Dynamics (BMDP Statistical Software, Cork, Ireland). Statistical significance was assumed if the probability of the type 1 error was below 5% (p<0.05).

4. Results

Table 2 summarizes peak performance data, and heart rate, SpO₂, and gas exchange data obtained at maximal exercise.

We observed a drop in oxygen saturation of more than 4% in 23 patients (31%). Among these, 17 patients (23% of the total sample) experienced a significant hypoxemia (SpO₂ below 90%) and 4 of those had a SpO₂ at peak exercise below 80%. All patients with a FEV₁ above 70% predicted did not show any significant exercise-induced hypoxemia. Those 4 patients who had a minimum SpO₂ below 80% all had a FEV₁ below 40% predicted. 7 of 65 patients with a FEV₁ of more than 50% FVC experienced a drop in SpO₂ below 90%, while all patients with a FEV₁/FVC below 50% showed significant exercise-induced hypoxemia. One patient with an exercise-induced drop in SpO₂ to 87% had a FEV₁ corresponding to 86% of FVC.

Correlation analyses revealed significant relationships between the exercise-induced drop in SpO₂ and the following variables: SpO₂ at rest (r=0.47, p<0.001), FEV₁ (r=0.59, p<0.001), FVC (r=0.52, p<0.001), MEF₂₅ (r=0.43, p<0.001), RV (r=0.31, p<0.01), TLC (r=0.27, p<0.05), and RV/TLC (r=0.45, p<0.001).

The same variables were also found to be related to SpO₂ at peak exercise: SpO₂ at rest (Fig. 1A, r=0.72, p<0.001), FEV₁ (Fig. 1B, r=0.66, p<0.001), FVC (r=0.59, p<0.001), MEF₂₅ (r=0.47, p<0.001), RV (r=−0.38, p<0.01), TLC (r=0.31, p<0.01), and RV/TLC (r=−0.51, p<0.001). Since the relationship between SpO₂ at peak exercise and FEV₁ appeared to be non-linear (see Fig. 1B), a transformation of FEV₁ (1/FEV₁) was also used and proved a stronger correlate of SpO₂ at peak exercise than FEV₁ itself (Fig. 1C).

Using multiple linear regression analysis, only SpO₂ at rest and 1/FEV₁ were identified as independent predictors of SpO₂ at peak exercise. The equation to estimate SpO₂ at peak exercise resulting from this analysis was:

\[
\text{SpO}_2 \text{ at peak exercise (in %)} = -16.4875 - 309.1474/\text{FEV}_1 \text{ (in % predicted)} + 1.1825*\text{SpO}_2 \text{ at rest (in %); } R^2 = 0.63
\]

This equation had a high sensitivity and specificity (82% and 95%, respectively) to predict an exercise-induced hypoxemia.
desaturation below 90% (see also Fig. 2A). However, 3 of
the 17 patients with a SpO2 at peak exercise below 90% were
not identified from the prediction equation and Bland-
Altman plots revealed that the prediction equation consider-
ably overestimates SpO2 at peak exercise in some patients
(Fig. 2B).

Fig. 2. Performance of a formula to predict peak exercise SpO2 using SpO2 at rest and 1/FEV1 as independent predictors. A) Relationship between predicted SpO2 at peak exercise and observed SpO2 at peak exercise. The continuous line indicates the line of identity, the dashed lines a SpO2 of 90%. The numbers in the four quadrants of the graph represent the number of patients with the respective SpO2 values above or below 90%. B) Bland-Altman plot of residual SpO2 at peak exercise (SpO2measured – SpO2predicted) over SpO2 at peak exercise predicted using SpO2 at rest plus 1/FEV1 as predictors. The continuous line indicates the mean residual, the dashed lines the 95%-confidence interval.

Fig. 3. ECG recordings obtained at rest and at peak exercise from two patients with CF. Both patients developed severe exercise-triggered ventricular arrhythmia. A) Female patient, 18-years-old, FEV1: 22.3% predicted, SpO2 at rest: 92%, SpO2 at peak exercise: 74%. B) Male patient, 40-years-old, FEV1: 23.2% predicted, SpO2 at rest: 95%, SpO2 at peak exercise: 86%.
5 of the 75 patients studied developed ventricular ectopic beats during exercise (see Fig. 3). Interestingly, the two patients with the most severe ventricular arrhythmia shown in Fig. 3 had very poor lung functions. In all five patients, resting ECG was normal without signs of pre-excitation syndrome, long QT-syndrome or right heart stress. The arrhythmias started during the exercise test at a heart rate ranging from 103 to 167 min⁻¹ and at a SpO₂ between 86% and 95%. None of the patients showed a >3 beat run and in all patients, the arrhythmias remained asymptomatic. Heart rhythm normalized within 15 min after the end of exercise. When comparing the characteristics of these five patients with exercise-triggered ventricular arrhythmia to those of the patients without arrhythmia the patients with arrhythmia had a significantly lower SpO₂ at rest and peak exercise and a lower VO₂peak but did not differ significantly in height and weight, lung function (FEV₁ % predicted, FCV % predicted, RV% predicted, RV/TLC) and maximum power (Watt).

Echocardiographic results were available in 44 patients, among these all 5 patients who showed cardiac arrhythmias during exercise. In three of these, echocardiographic studies at rest showed signs of right heart stress, which were, however, also observed in 4 patients without exercise-induced cardiac arrhythmias.

Two additional patients suffered from ectopic ventricular extra systoles at rest which disappeared during exercise. One clinically asymptomatic 37-year-old patient was diagnosed to suffer from a pre-excitation (Wolff–Parkinson–White) syndrome, without showing any irregularities of cardiac rhythm at rest or during exercise-induced stress.

5. Conclusions

This study shows that exercise-induced hypoxemia occurs in a considerable number of patients with CF. Furthermore, 23% of the patients studied experienced a significant drop in SpO₂ below 90% during exercise, a finding which might warrant exercise restrictions in these patients [23]. In line with our findings, Lebecque et al. [11] reported a prevalence of exercise-induced hypoxemia of 26% in 21 patients. However, Henke et al. [10] observed a lower prevalence of exercise-induced hypoxemia more than 20 years ago (10%). These differences among studies might be due to differences in the patients’ characteristics studied, i.e. the patients’ age, and the fact that patients nowadays are likely to be more physically active and can perform exercise at higher work rates, thereby inducing more profound changes. Moreover, we used a forehead sensor to determine SpO₂ while Henke et al. [10] as well as Lebecque et al. [11] employed an ear sensor. In comparison with finger sensors, the forehead sensor has proven to provide more valid readings during exercise [12] which might have enabled us to discover hypoxemia more precisely.

Based on the data of 21 patients, Lebecque et al. [11] stated that dangerous exertional desaturations are most unlikely, if FEV₁ is greater than 35% predicted while FEV₁ in percent of FVC was no useful predictor. Henke et al. [10] concluded that no reliable information with respect to exercise-induced hypoxemia can be drawn from pulmonary function testing. However, in their study, patients with a FEV₁ above 50% of forced vital capacity did not suffer from an exercise-induced drop in SpO₂ below 90% which might lead to the assumption that patients with a better lung function are less at risk for exercise-induced hypoxia. Indeed, Henke et al. [10] suggested that mainly those patients with a FEV₁ less than or equal to 50% of FVC require regular exercise-testing. In the present study, though, about 10% of the patients with a FEV₁ above 50% FVC experienced significant hypoxemia with exercise. Thus, the recommendations of Henke et al. [10] need to be extended to patients with less severe lung disease. Data from our study suggest including at least all patients with a FEV₁ below 70% predicted or with a FEV₁ below 90% of FVC, as we found significant drops of oxygen saturation (below 90%) even in 2 patients with and FEV₁ between 70% and 90% of FVC.

Previous studies have shown that peak exercise SpO₂ is related to FEV₁ and resting SpO₂ in patients with CF [10,11,24]. The present study reproduced these findings and identified other lung function parameters as additional correlates of peak exercise SpO₂. We employed multiple linear regression analysis to improve the identification of patients who will significantly desaturate during exercise. Although a high sensitivity and specificity of the resulting prediction equation was observed, some of the patients who had significant hypoxemia during exercise were not identified. Thus, as with previous attempts to identify patients at risk for exercise-induced hypoxemia using measurements obtained at rest, the approach employed in this study does not provide an acceptable sensitivity at a high level of specificity.

In a group of 21 patients, Lebecque et al. [11] observed that patients with a diffusion capacity for carbon monoxide (DCO) above 80% predicted did not suffer from desaturations during exercise-testing. In the present study, DCO was not measured. However, although we cannot exclude that the additional measurement of DCO might have added to our prediction model, this seems unlikely for the following reasons: 1) Lebeque et al. [11] and others [25] reported a strong correlation between FEV₁ and DCO in CF, so that one of the variables may most likely be substituted by the other in multiple regression analysis. 2) In contrast to the study by Lebecque et al. [11] some of our patients with a FEV₁ greater than 35% and up to 68% showed a drop in oxygen saturation below 90%. Thus, it is quite likely that patients with less severe pulmonary disease than those reported by Lebecque et al. [11] will experience significant exercise-induced hypoxemia.

To our knowledge, this study is the first to report on exercise-triggered ventricular arrhythmias in patients with CF. The prevalence of this potentially hazardous side effect of physical exertion was 5/75 patients or 6–7% of all patients tested. In presumably healthy children and adults, exercise-induced ventricular ectopic activity is extremely rare with a prevalence <1% [26,27].

In patients with severe COPD, Cheong et al. [14] observed a much higher prevalence of ventricular arrhythmia (19.7%) compared with the present study. There are several possible explanations for the different findings in the two studies: The
patients studied by Cheong et al. had FEV1 values of 28.3 ± 1.1% predicted while most of our patients had a far better pulmonary function. Furthermore, the patients with COPD were considerably older than the patients with CF in the present study. It has been shown that the prevalence of exercise-induced ventricular arrhythmias increases with age [28].

In line with our hypothesis that exercise-triggered ventricular arrhythmia would be related to exercise-induced hypoxemia, our five patients with this form of cardiac arrhythmia had significantly lower SpO2 at rest and at peak exercise compared with the patients who showed no cardiac dysrhythmia with exercise. However, only three of the five patients demonstrated a drop in SpO2 below 90% with exercise and only four patients dropped their SpO2 by more than 4%. Using the prediction equation for peak-exercise SpO2 developed in this project, two of the five patients with exercise-triggered cardiac arrhythmia had a SpO2 of >90%predicted. As to their lung function, three of the five patients had a FEV1 <50% predicted, whereas the remaining two had relatively mild lung disease with a FEV1 between 60% and 70% predicted. Standard echocardiography at rest also could not clearly discriminate between patients who are at risk for exercise-induced arrhythmia. Thus, it seems impossible to identify all patients with exercise-induced cardiac arrhythmia using solely measurements taken at rest.

The five subjects in our study did not show any clinically relevant symptoms caused by the exercise-induced arrhythmias. Furthermore, no exercise test had to be terminated according to the actual guidelines (increasing ventricular ectopy including a >3 beat run in children [29] or angina or sustained ventricular tachycardia in adults [30]). As to the clinical consequences of the cardiac arrhythmias for exercise recommendations, no clear evidence-based statement can be made since no reports on exercise-related cardiac deaths could be found in patients with CF. In patients with coronary artery disease causing myocardial ischemia — possibly one reason for the exercise-induced arrhythmias also in CF patients with low oxygen saturation — a bigeminus as observed in one of our patients (Fig. 3A) or couplets of ectopic beats in another (Fig. 3B) are associated with a more frequent occurrence of myocardial infarction [31]. Cheong et al. [14] looked at cardiac arrhythmias in patients with COPD and concluded that potentially serious arrhythmias would be uncommon in these patients if they did not have clinically significant arrhythmias at rest. Nevertheless, in our patients we recommended to limit exercise intensity to levels with no arrhythmias. In one patient with severe exercise-induced hypoxemia and ventricular arrhythmias, supplemental oxygen during exercise also proved effective. None of the five patients with exercise-induced arrhythmias died during or shortly after exercise or of sudden cardiac problems after a 2- to 5-year follow-up.

We have shown in this study that a considerable proportion of patients with CF experience significant hypoxemia and cardiac arrhythmia during exercise. As shown above, those patients at risk are not easily identified from assessment at rest. Based on our data, we recommend performing exercise testing at least in patients with a FEV1 below 70% predicted, if not in all patients old enough to undergo standardized testing, as monitored exercise-testing is the only means to unambiguously discover exercise-induced hypoxia and cardiac arrhythmia. These tests should be carried out according to current guidelines. Although our data are by no means a proof for exercise-limitations if ventricular arrhythmias are detected during testing, we recommend to either limit maximal exercise intensity or to use additional oxygen in order to keep the patients physically active.

Data was presented as poster at the 102nd annual meeting of the German Society of Pediatrics and Adolescent Medicine (Deutscher Gesellschaft für Kinder- und Jugendmedizin) in Mainz/Germany in September 2006.

**Frahradergometrie zur Erfassung von Risiken körperlicher Belastung bei Mukoviszidose**

K. Ruf, H. Hebestreit

Universitäts-Kinderklinik, Josef-Schneider-Str. 2, 97080 Würzburg

**Fragenstellung:** Körperliche Aktivität und Sport werden bei Mukoviszidose zunehmend therapeutisch eingesetzt. Ziel dieser Studie war die Prävalenz von belastungsinduzierter Hypxämie und Herzrhythmusstörungen bei Mukoviszidosepatienten im Rahmen einer standardisierten Belastung zu erfassen.


**Ergebnisse:** Bei 22 Patienten (=32%) fiel die O₂-Sättigung bei Belastung um mehr als 4%, bei 16 Patienten unter 90%. Der Abfall der O₂-Sättigung bzw. die Sättigung bei maximaler Belastung korrelierten mit der FEV1 (r = 0,56 bzw. r = 0,65), es kam jedoch auch bei einem Patienten mit einer FEV1 von 103% zu einem Sättigungsabfall >4%, bei einem weiteren Patienten mit einer FEV1 von 69% fiel die Sättigung unter 90%. 5 Patienten (7%) entwickelten belastungsinduzierte ventrikuläre Extrasystolen (Alter 16,7–35,7 Jahre, FEV1 22,3–69,0%, Start bei Herzfrequenz 103–167 min⁻¹ bzw. Sättigung bei 86–95%). Bei diesen Patienten zeigte sich in der Echokardiographie eine Rechtsherzbelastung. Bei 2 weiteren Patienten konnten Rhythmusstörungen in Ruhe festgestellt werden, die unter Belastung verschwanden (z.B. so genannte benign ventrikuläre Extrasystolen).

**Diskussion:** Patienten mit Mukoviszidose und insbesondere solche mit einer schlechteren Lungenfunktion haben ein erhöhtes Risiko für einen belastungsbedingten Abfall der O₂-Sättigung sowie - besonders bei Rechtsbelastung und belastungssassoziertem Abfall der O₂-Sättigung - belastungsinduzierte ventrikuläre Rhythmusstörungen.
**Fazit:** Ergometrische Untersuchungen sollten bei Patienten mit Mukoviszidose, die eine eingeschränkte Lungenfunktion aufweisen, regelmäßig zur Erfassung von belastungsinduzierten Komplikationen durchgeführt werden. Weiterhin kann die im Rahmen der Ergometrie definierte Herzfrequenz, bei der es zur Hypoxämie bzw. Rhythmusstörung kommt, als Obergrenze für die Intensität sportlicher Aktivität genutzt werden.

Data was presented as oral presentation at the 8th annual meeting of the Society of Pediatric Sports Medicine (Gesellschaft für Pädiatrische Sportmedizin) in Basel/Switzerland in February 2008.

Belastungsinduzierte Hypoxämie und Herzrhythmusstörungen bei Mukoviszidose

K. Ruf, H. Hebestreit

Universitäts-Kinderklinik, Josef-Schneider-Str. 2, 97080 Würzburg

**Fragestellung:** Körpliche Aktivität und Sport spielen in der Therapie der Mukoviszidose zunehmend eine wichtige Rolle. Ziel dieser Studie war es, die Prävalenz von belastungsinduzierter Hypoxämie und Herzrhythmusstörungen zu erfassen sowie mögliche Vorhersagekriterien für deren Auftreten zu identifizieren.

**Methodik:** 75 Patienten mit Mukoviszidose (12–41 Jahre alt) wurden spirotorquematisch unter pulsoxymetrischer und EKG-Kontrolle untersucht. Die Analyse der Vorhersagekriterien erfolgte mittels multipler linearer Regression.

**Ergebnisse:** Bei 17 Patienten (23%) fiel die Sauerstoffsättigung bei maximaler Belastung (SpO2max) unter 90%, davon bei 4 Patienten unter 80%. Sowohl FEV1 (forced expiratory volume in 1 s) als auch die Sauerstoffsättigung in Ruhe (SpO2Ruhe) wiesen signifikante Zusammenhänge mit der Sauerstoffsättigung unter Belastung auf (r=0,66 bzw. r=0,71). In der Regressionsanalyse verhielt sich die Sauerstoffsättigung bei maximaler Belastung unabhängig zum Ruhewert sowie zu den Werten der Lungenfunktion.

5 Patienten entwickelten unter Belastung ventrikuläre Extrasystolen. Diese Patienten hatten eine signifikant niedrigere Sauerstoffsättigung (p<0,05) sowohl in Ruhe als auch unter Belastung.

**Schlussfolgerung:** Trotz der Tendenz, dass Patienten mit niedrigerem FEV1 und niedrigerer SpO2Ruhe eher unter Hypoxämie bzw. Herzrhythmusstörungen litten, war im Individualfall keine klare Vorhersage möglich. Deshalb sind Belastungsuntersuchungen die einzige Möglichkeit, belastungsinduzierte Hypoxämie und Herzrhythmusstörungen sicher zu identifizieren.

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