# Antipanic drugs and pulmonary function in panic disorder patients

## Medicação antipânico e função pulmonar em pacientes com transtorno de pânico

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## Abstract

**Background:** Studies suggest an association between panic disorder (PD) and impairment of lung function. **Objectives:** To evaluate lung function in 11 asymptomatic PD patients and to investigate antipanic drug effects on respiratory function. **Method:** Lung function was evaluated on two different occasions (with antipanic drugs and after drug washout). It was comprised of a spirometric evaluation and a bronchodilation test (salbutamol inhalation). Subjective Units of Disturbance Scale (SUDS) was applied before and after each spirometric assessment. **Results:** One patient showed mild obstructive airway impairment. Before bronchodilation test forced expiratory volume in 1 sec ($FEV_1$) and forced expiratory flow between 25% and 75% of the forced vital capacity ($FEF_{25-75}$) were significantly higher in patients on antipanic drugs than in those in the washout period. After salbutamol inhalation, only $FEV_1$ was significantly higher in patients with antipanic drugs in comparison to the other group, whereas a significant increase in $FEV_1$ and $FEF_{25-75}$ after salbutamol inhalation was detected in patients without antipanic drugs. The subjective anxiety level was not different among PD patients in both test days. **Discussion:** These results suggest a possible beneficial effect of the antipanic drug on lung function in PD patients.

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**Keywords:** Lung function, spirometry, respiration, salbutamol, anxiety disorder.

## Resumo

**Contexto:** Estudos sugerem uma associação entre transtorno de pânico (TP) e prejuízos na função pulmonar. **Objetivos:** Avaliar a função pulmonar em 11 pacientes com TP assintomáticos e investigar efeitos da medicação antipânico na função respiratória. **Método:** A função pulmonar foi avaliada em duas ocasiões diferentes (com medicação antipânico e após "*washout*"). Consistiu de uma avaliação espirométrica e do teste de broncodilatação (inalação de salbutamol). Subjective Units of Disturbance Scale (SUDS) foi aplicada antes e após cada teste espirométrico. **Resultados:** Um paciente apresentou obstrução leve de vias aéreas. Antes do teste de broncodilatação, o volume expiratório forçado no primeiro segundo ($VEF_1$) e o fluxo expiratório forçado entre 25% e 75% da capacidade vital forçada ($FEF_{25-75}$) foram significativamente maiores em pacientes com medicação antipânico do que no período de "*washout*". Após a inalação de salbutamol, apenas o $VEF_1$ foi significativamente maior em pacientes com medicação antipânico em comparação ao outro grupo, embora tenha sido detectado aumento significativo em $VEF_1$ e $FEF_{25-75}$ em pacientes sem medicação antipânico depois da inalação de salbutamol. O nível de ansiedade subjetiva não foi diferente entre os pacientes em ambos os dias de testes. **Conclusão:** Os resultados sugerem uma possível ação benéfica da medicação antipânico na função pulmonar em pacientes com TP.

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**Palavras-chave:** Função pulmonar, espirometria, respiração, salbutamol, transtorno de ansiedade.

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## Introduction

The suffocation false alarm[1] and dyspnea-fear theories of panic disorder (PD)[2] emphasize the central role of dyspnea/suffocation symptom in panic attacks. Recently several studies have tested these hypotheses relating PD with respiratory system[3-6]. The cingulate gyrus and limbic cortex are suprabulbar structures involved in the respiratory regulation[7]. The anterior cingulate gyrus produce an inhibitory effect on respiration, and serotonin may act as a synaptic transmitter in respiratory neurons, may change their sensitivity to external regulatory influences, and affect the respiratory response to limbic cortex stimulation[7].

A growing number of clinical[8,9] and epidemiological[10] studies suggests an association of anxiety disorder and asthma, although the specificity of this relationship remains unclear.

In order to explain the higher lifetime prevalence of PD in patients with respiratory disease, some studies investigated possible differences in lung function or in cognition between asthmatic patients with or without PD[11-13]. Van Peski-Oosterbaan *et al.*[14] observed that baseline levels of forced expiratory volume in 1 sec ($FEV_1$) and bronchial responsiveness to histamine were not significantly different in asthmatic patients with or without PD. They suggested that the presence of an airway disease or the degree of pulmonary function impairment is not related to panic symptoms. Perna *et al.*[15] assessed lung function in 17 PD patients with or without agoraphobia and 20 healthy controls. They showed that PD patients had significantly lower values for some dynamic lung parameters, namely, peak expiratory flow rate (PEFR or $FEF_{max}$), forced expiratory flow at 75% of vital capacity ($FEF_{75}$) and maximal mid-expiratory flow rate (MMEF or $FEF_{25-75}$), suggesting subclinical impairment of lung airways. These findings, however, could not be replicated[16]. Carr *et al.*[17] assessed airway impedance in response to a psychological stressor among 113 subjects divided in 4 groups: 61 with asthma alone, 10 with asthma and PD, 24 with PD alone, and 18 healthy controls. They demonstrated that PD patients (with or without asthma) present lower airway impedance (more dilated airways) than those without PD, suggesting a respiratory system more prepared to react to stress.

Since clinical data suggest that the susceptibility to spontaneous as well as $CO_2$-induced anxiety and hyperventilation is attenuated by selective serotonin reuptake inhibitor (SSRI) antidepressants, Olsson *et al.*[18] explored the possible effect of paroxetine (SSRI) on baseline respiration and $CO_2$-induced hyperventilation in rats. They suggested that serotonin may influence brainstem regulation of baseline respiratory rate and ventilatory response to hypercapnia. Notably in their study tidal volume was not influenced by paroxetine treatment. Another animal study[19] suggested that serotonin can induce the contraction of intrapulmonary arteriole smooth muscle cells via 5-HT2 receptors. Yeragani *et al.*[20] studied the influence of paroxetine on respiratory rate and tidal volume among humans with PD. They found that paroxetine decreases some linear measures of variability of lung volume after treatment[20].

The present work was designed to evaluate lung function in asymptomatic PD patients with or without agoraphobia, who had developed no panic attacks or agoraphobic symptoms in the last three months prior to the study. The aims were: 1) to investigate possible differences in lung function and in the subjective anxiety level on two occasions, with and without antipanic drugs; 2) to evaluate the influence of antipanic drugs on bronchial responsiveness to salbutamol inhalation in PD patients. We hypothesized that PD patients do not present any impairment in their lung function.

## Methods

We consecutively enrolled 12 PD patients with or without agoraphobia who were under treatment at the Laboratory of Panic and Respiration of the Federal University of Rio de Janeiro, ranging from 18 and 65 years old, and agreed to participate in this protocol. The diagnosis was confirmed by the Structured Clinical Interview[21] for DSM-IV[22]. Patients were clinically in good physical condition and were asymptomatic, i.e., did not report any panic attacks and agoraphobic symptoms in the last three consecutive months before the first lung function test. We considered three months a safe period to consider the patient remitted from his symptoms and consequently in a minimal level of risk for recurrency of the panic symptoms during discontinuation and washout periods of antipanic medication. Exclusion criteria were the existence of any other current mental disorder other than PD, a history of psychosis or bipolar disorder, epilepsy, substance abuse within the last 6 months, past or current diagnoses of respiratory disorders, significant concurrent medical problems, personal history of smoking, and pregnant or nursing women. The study design was explained to the patients and they signed a voluntary written informed consent for their participation in this study. They were informed that the objective was to assess the possible relationship between panic and lung function. The protocol, which complied with the principles laid down in the Declaration of Helsinki, was approved by our Hospital Ethics Committee.

Lung function was assessed on two different days and comprised of spirometric evaluation and bronchodilation test (salbutamol inhalation). On the first test day all the patients were taking their regular antipanic drugs (antidepressant and/or benzodiazepine). It is worthy to note

that this clinical sample followed the treatment protocol of the Laboratory of Panic and Respiration of the Federal University of Rio de Janeiro on that time, what indicated imipramine as the first choice antidepressant and clonazepam as the second medicine of choice to treat panic disorder patients. These drugs were reduced gradually to avoid discontinuation symptoms and recurrence of panic attacks. The patients were asked to washout the drugs for at least seven days before the second test day. The protocol was carried out in a single-blind fashion, so the experimenters performing the lung function test did not know whether the patients were taking antipanic drugs or not.

### Procedure and Data Analysis

Lung function was assessed by spirometry (flow-volume and volume-time curves), according to recommendations of the American Thoracic Society[23], at the Laboratory of Lung Function, Clementino Fraga Filho University Hospital. All patients were tested in the morning, in the sitting position, by a pulmonary physician and a nurse trained to perform the test (Pulmonary Function System – Modular GS and DSIIA, Warren E. Collins Inc., Braintree, MA, USA). Patients were asked to perform three forced vital capacity maneuvers, and the highest values were used in the analysis. A maximum of eight attempts was allowed to obtain three acceptable curves, according to acceptability and reproducibility criteria[23]. The following parameters were measured: forced vital capacity (FVC); forced expiratory volume in 1 sec ($FEV_1$); the ratio between $FEV_1$ and FVC ($FEV_1$/FVC); forced expiratory flow between the 25% and 75% of forced vital capacity ($FEF_{25-75}$); the highest instantaneous flow during the maneuver ($FEF_{max}$); and forced expiratory flow at 25% ($FEF_{25}$), 50% ($FEF_{50}$) and 75% ($FEF_{75}$) of forced vital capacity. All variables (except $FEV_1$/FVC) were expressed as absolute values as well as percentages of the predicted value according to Knudson *et al.*[24] After the first session of tests, patients inhaled 400 µg of a beta 2-agonist bronchodilator (salbutamol) divided in four doses every five minutes. After fifteen minutes, the second spirometric evaluation was performed. Patients were informed that they might experience some discomfort like headache, dizziness, trembling, palpitation or symptoms of anxiety with bronchodilator administration.

Subjective Units of Disturbance Scale (SUDS)[25], which reflects the degree of global subjective anxiety (ranging from 0 = none to 10 = extreme anxiety) was applied immediately before the first spirometric evaluation (SUDS 1), immediately after it (SUDS 2), immediately before the second spirometric evaluation (after inhaled bronchodilator) (SUDS 3) and at the end of the experiment (SUDS 4).

To compare the results, firstly, the normality of the data (Kolmogorov-Smirnov test with Lilliefors' correction) and the homogeneity of variances (Levene median test) were tested. If both conditions were satisfied, one-way repeated measures ANOVA was used; in the negative case, Friedman repeated measures ANOVA on ranks was selected instead. If multiple comparisons were then required, Student-Newman-Keuls' (parametric) or Dunn's (nonparametric) test was applied. The significance level was always set at 5%.

## Results

One patient did not participate in the second test day ("without antipanic drugs" day) and was excluded. The remaining patients were 6 men (54.5%) and 5 women (45.5%). Their ages ranged from 26 to 47, with a mean (± SD) age of 35.6 (± 7.2) years. Ten patients (90.9%) also suffered from agoraphobia.

Tables 1 and 2 show the clinical variables in 11 PD patients before the first and the second test days, respectively.

Two patients reported limited symptoms of panic during washout (cases 6 and 8).

Only one patient (case 2) reported symptoms just after salbutamol inhalation on the second test day (without antipanic drugs). These symptoms were: dizziness, sweating, trembling and palpitation, but the subject did not report anxiety or panic symptoms.

One patient (case 4) presented a mild obstructive airway disturbance on both test days that was reversible with bronchodilator administration on second test day.

The SUDS level was not significantly different between the two test days, before and after inhaled bronchodilator (Figure 1).

Table 3 shows the mean values (± SD) of all lung function parameters.

Considering % predicted value, before bronchodilation test $FEV_1$ and $FEF_{25-75}$ were significantly higher in patients on antipanic drugs than in those in the washout period (Table 3). After salbutamol inhalation, only $FEV_1$ was significantly higher in patients on antipanic drugs than in those in the washout period (Table 3). We also observed a significant increase in $FEV_1$ and $FEF_{25-75}$ after salbutamol inhalation in patients without antipanic drugs (Table 3).

Considering absolute values, before bronchodilation test $FEV_1$ and $FEF_{25-75}$ were significantly higher in patients on antipanic drugs than in those in the washout period (Table 3). We observed a significant increase in $FEV_1$ and $FEF_{25-75}$ after salbutamol inhalation in patients without antipanic drugs (Table 3).

**Table 1.** Clinical variables in 11 panic disorder patients before the first test day

| Cases | Latency[1] (months) | Antipanic drugs | SUDS 1 | SUDS 2 | SUDS 3 | SUDS 4 |
|---|---|---|---|---|---|---|
| 1 | 16 | 75 mg/day of imipr plus 2 mg/day of clonaz | 5 | 7 | 7 | 7 |
| 2 | 10 | 75 mg/day of imipr plus 1 mg/day of clonaz | 2 | 2 | 3 | 0 |
| 3 | 11 | 20 mg/day of parox plus 0.25 mg/day of clonaz | 6 | 1 | 2 | 1 |
| 4 | 5 | 1 mg/day of clonaz | 1 | 0 | 0 | 0 |
| 5 | 14 | 50 mg/day of imipr plus 1 mg/day of clonaz | 10 | 6 | 5 | 3 |
| 6 | 84 | 75 mg/day of imipr plus 10 mg/day of diaz | 10 | 5 | 2 | 0 |
| 7 | 24 | 25 mg/day of imipr | 2 | 2 | 3 | 0 |
| 8 | 6 | 75 mg/day of imipr plus 2 mg/day of clonaz | 4 | 0 | 2 | 0 |
| 9 | 35 | 0.5 mg/day of clonaz | 2 | 2 | 4 | 3 |
| 10 | 7 | 25 mg/day of imipr plus 1 mg/day of clonaz | 0 | 1 | 0 | 0 |
| 11 | 21 | 75 mg/day of imipr plus 0.8 mg/day of clonaz | 3 | 2 | 2 | 3 |

*[2] Latency: the interval between the first (on medication) and the second (without antipanic drugs) test days (mean 4.3 ± 3.5 months).*
*[3] Washout period (median: 8.0 days; 25th percentile = 7.0, 75th percentile = 9.5).*
*SUDS: Subjective Units of Disturbance Scale. 1: immediately before first spirometric evaluation; 2: immediately after spirometry; 3: immediately before the second spirometric evaluation (after salbutamol inhalation); 4: at the end.*

**Table 2.** Clinical variables in 11 panic disorder patients before the second test day

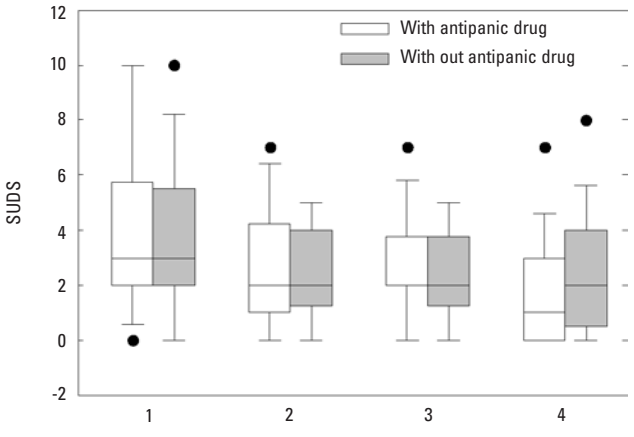
| Cases | Latency[2] (months) | Washout[3] (days) | Discontinuation Symptoms | SUDS 1 | SUDS 2 | SUDS 3 | SUDS 4 |
|---|---|---|---|---|---|---|---|
| 1 | 5.5 | 8 | dizziness, insomnia | 4 | 4 | 4 | 4 |
| 2 | 12 | 7 | dizziness, shortness of breath | 7 | 2 | 2 | 2 |
| 3 | 4.5 | 7 | dizziness, nausea, "heavy-head" | 2 | 1 | 1 | 0 |
| 4 | 2 | 15 | none | 3 | 3 | 3 | 3 |
| 5 | 3.5 | 8 | dizziness, trembling, hot wave | 2 | 2 | 2 | 8 |
| 6 | 2 | 7 | chest pain, shortness of breath, arterial hypertension, crying | 10 | 5 | 5 | 4 |
| 7 | 1 | 15 | none | 0 | 0 | 0 | 0 |
| 8 | 4.5 | 7 | none | 6 | 4 | 2 | 2 |
| 9 | 2 | 10 | none | 2 | 2 | 2 | 2 |
| 10 | 1.5 | 8 | trembling, sweating | 0 | 0 | 0 | 0 |
| 11 | 9.5 | 60 | none | 4 | 5 | 5 | 4 |

*[2] Latency: the interval between the first (on medication) and the second (without antipanic drugs) test days (mean 4.3 ± 3.5 months).*
*[3] Washout period (median: 8.0 days; 25th percentile = 7.0, 75th percentile = 9.5).*
*SUDS: Subjective Units of Disturbance Scale. 1: immediately before first spirometric evaluation; 2: immediately after spirometry; 3: immediately before the second spirometric evaluation (after salbutamol inhalation); 4: at the end.*

**Table 3.** Lung function parameters

| | Panic disorder patients on antipanic drug | | | |
|---|---|---|---|---|
| | Before salbutamol | | After salbutamol | |
| | Observed | % Predicted | Observed | % Predicted |
| FVC (L) | 4.31 ± 0.95 | 104.63 ± 12.66 | 4.31 ± 0.94 | 104.72 ± 12.24 |
| $FEV_1$(L) | 3.58 ± 0.71 | 107.09 ± 13.74 | 3.66 ± 0.75 | 109.27 ± 13.54 |
| $FEV_1$/FVC (%) | 83.81 ± 7.69 | 102.81 ± 8.94 | 85.36 ± 6.10 | 104.81 ± 7.32 |
| $FEF_{25-75}$ (L/sec) | 4.00 ± 1.06 | 110.18 ± 27,84 | 4.33 ± 1.11 | 118.72 ± 29.19 |
| $FEF_{max}$ (L/sec) | 9.11 ± 2.57 | 117.54 ± 22.65 | 9.23 ± 2.15 | 119.90 ± 19.89 |
| $FEF_{25}$ (L/sec) | 7.48 ± 1.81 | 106.81 ± 24.75 | 7.36 ± 1.92 | 105.36 ± 25.97 |
| $FEF_{50}$ (L/sec) | 4.80 ± 1.30 | 106.18 ± 30.69 | 4.96 ± 1.64 | 108.81 ± 35.11 |
| $FEF_{75}$ (L/sec) | 1.80 ± 0.57 | 88.36 ± 33.29 | 1.99 ± 0.74 | 96.63 ± 42.26 |
| | **Panic disorder patients without antipanic drug** | | | |
| | Before salbutamol | | After salbutamol | |
| | Observed | % Predicted | Observed | % Predicted |
| FVC (L) | 4.15 ± 0.85 | 102.90 ± 11.64 | 4.16 ± 0.92 | 102.90 ± 12.42 |
| $FEV_1$ (L) | 3.42 ± 0.67* | 101.81 ± 14.40* | 3.54 ± 0.70# | 105.18±13.25*# |
| $FEV_1$/FVC (%) | 82.81 ± 4.66 | 98.72 ± 5.48 | 85.72 ± 4.24 | 102.27 ± 4.56 |
| $FEF_{25-75}$ (L/sec) | 3.67 ± 0.84* | 100.45 ± 25.70* | 4.11 ± 0.83# | 112.09 ± 24.44# |
| $FEF_{max}$ (L/sec) | 8.30 ± 1.85 | 108.18 ± 15.97 | 8.84 ± 2.48 | 114.09 ± 20.39 |
| $FEF_{25}$ (L/sec) | 6.98 ± 1.75 | 98.18 ± 21.98 | 7.33 ± 1.77 | 102.81 ± 21.55 |
| $FEF_{50}$ (L/sec) | 4.52 ± 1.16 | 104.36 ± 29.51 | 4.89 ± 1.16 | 112.81 ± 31.73 |
| $FEF_{75}$ (L/sec) | 1.70 ± 0.36 | 91.36 ± 19.85 | 1.90 ± 0.43 | 101.63 ± 22.10 |

*Values are means ± SD of 11 panic disorder patients with and without antipanic drug consumption, before and after salbutamol inhalation. FVC, forced vital capacity; $FEV_1$, forced expiratory volume in 1 sec; $FEV_1$/FVC, ratio between $FEV_1$ and FVC; $FEF_{25-75}$, forced expiratory flow between the 25% and 75% of forced vital capacity; $FEF_{max}$, the highest instantaneous flow during the maneuver; forced expiratory flow at 25% ($FEF_{25}$), 50% ($FEF_{50}$) and 75% ($FEF_{75}$) of forced vital capacity. # Significantly different from value before salbutamol (p < 0.05). * Significantly different from value with antipanic drug (p < 0.05).*




SUDS: Subjective Units of Disturbance Scale. 1: immediately before first spirometric evaluation; 2: immediately after spirometry; 3: immediately before the second spirometric evaluation (after salbutamol inhalation); 4: at the end. The boundary of the box closest to zero indicates the 25th percentile, a line within the box marks the median, and the boundary of the box farthest from zero indicates the 75th percentile. Whiskers above and below the box indicate the 90th and 10th percentiles. Points represent outliers.

**Figure 1.** Subjective anxiety level in 11 panic disorder patients in four different moments during the lung function assessment with and without antipanic drug consumption.

## Discussion

Our results suggest that lung function in PD patients is normal, as mentioned by Verburg *et al.*[16] Only one patient presented a mild reversible airway obstruction in response to inhalation of 400 µg of salbutamol. This finding could result from either sampling error or asymptomatic asthma[26].

We observed higher values of $FEV_1$ and $FEF_{25-75}$ in patients on antipanic drugs than when they were in the washout period. This finding persisted in $FEV_1$ even after the inhalation of bronchodilator. Salbutamol acted to a larger extent in PD patients without antipanic drugs. They presented a significant increase in $FEV_1$ and $FEF_{25-75}$.

Olsson *et al.*[18] found, in rats, a significant increase in baseline respiratory rate after 5 and 15 weeks of treatment with paroxetine. After 15 weeks of treatment the rats presented a reduction in the respiratory rate in response to $CO_2$ exposure and suggested that the regulation of respiration may be an important factor for the paroxetine antipanic effect. Olsson *et al.*[18] did not report

the change in response of $CO_2$-induced hyperventilation to an anxiety-reducing effect of paroxetine because they considered that the concentrations of $CO_2$ could not provoke anxiety in rats.

Some preliminary studies found sertraline (SSRI) useful in the management of dyspnea in chronic obstructive pulmonary disease and comorbid anxiety and mood disorders[27,28]. It has been reported that the levels of free serotonin in plasma are increased in symptomatic patients with asthma[29,30]. Lechin *et al.*[29] randomly assigned 69 children with asthma to receive tianeptine, an antidepressant drug, and/or placebo in a double-blind crossover trial that lasted 52 weeks. Tianeptine caused a sudden decrease in both clinical rating and free serotonin plasma levels and an improvement in pulmonary function. Nardi and Perna also discuss the serotonergic role of clonazepam[31].

Perna *et al.*[15] speculated that small airway subclinical obstruction in their study might be related to abnormalities in the mechanisms controlling bronchial tone, principally the tone of the smooth muscles in the small airways, finely regulated by autonomic nervous system (there are cholinergic, adrenergic and noncholinergic nonadrenergic influences). On the other hand, Carr *et al.*[17] found that PD patients had wider airways in both stressful and non-stressful conditions.

Although vital capacity maneuvers might increase the anxiety level in PD patients owing to hyperventilation and catastrophic misinterpretation of respiratory symptoms[32], mainly after inhalation of salbutamol, we observed that the median of SUDS ranged from 1 to 3 (Figure 1) without a significant difference. PD patients showed similar subjective anxiety levels with and without antipanic drugs. Perhaps the control of panic and agoraphobic symptoms is responsible for the low anxiety index in this sample.

To our knowledge the present study differs from other studies because it includes pulmonary asymptomatic PD patients, whose function tests were determined with and without antipanic drugs. Some studies found an association between panic attacks and anxiety sensitivity[11,13] that reflects an individual's concern about the consequences of experiencing anxiety-related symptoms. Carr *et al.*[13] demonstrated that PD patients, asthmatic and nonasthmatic, displayed greater fear of bodily sensations on the Body Sensations Questionnaire[33] and more negative beliefs about the consequences of anxiety on the Anxiety Sensitivity Index[34] relative to those without PD. In addition, the presence of asthma alone had no effect on these measures of panic-related cognitions, since only asthmatic with PD had elevated scores. Finally, scores among asthmatics with PD tended to be lower than those among nonasthmatics with PD. Carr *et al.*[13] found that anxiety sensitivity was unrelated to any of the pulmonary parameters measured (FCV, $FEV_1$, $FEV_1$/FCV or $FEF_{50}$), thus indicating that the relationship between anxiety sensitivity and PD was not mediated by extra impairment in lung function. Romano *et al.*[35] observed a significant reduction in anxiety sensitivity after 6 weeks of treatment with citalopram (SSRI), suggesting that fear of the consequences of body sensation could be normalized by pharmacological treatment and that a serotonergic mechanism might be involved in changing a cognitive distortion present in PD. These authors suggest that the decrease in anxiety sensitivity after drug treatment alone challenges the idea that the decrease of anxiety sensitivity is selectively mediated by cognitive/behavioral treatment[36].

The limitations of the study were: 1) the small sample. Our results should be interpreted cautiously, because the power of the performed test is bellow the desired one. The small sample also determined the non-stratification of the patients in groups according to their prescription scheme, for example one group with persons medicated only with SSRI and another one only on tricyclic antidepressants; 2) the PD patients were not submitted to a complete medical evaluation; 3) it was not possible to discriminate which drugs act in lung function (antidepressant, benzodiazepine or both) because of our small sample.

Further studies are required to evaluate if the improvement in lung function in asymptomatic PD patients is due to the antipanic effects in controlling bronchial tone or by decreasing anxiety sensitivity and panic-related cognitions. Follow-up studies with samples of asthmatic PD patients may also show the impact of PD treatment in the respiratory symptoms of asthma. Further prospective double-blind studies to evaluate pulmonary function on PD patients are required, since the acute phase until a remission stage, and stratified according to the medication under use (SSRI alone, tricyclic antidepressant alone, or these associated with benzodiazepines).

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