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Evaluation of sympathetic skin response in Parkinson's disease

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Abstract

There is no clear definition on the role of sympathetic skin response (SSR) in the evaluation of patients with Parkinson's disease (PD). We recorded the SSR of the palms of 64 controls and 46 patients with PD to electrical stimulation of the median nerve at the wrist. We analyzed onset latency and peak-to-peak amplitude. A study of parasympathetic function (R–R interval analysis) was also undertaken. We found that patients with PD had more absent SSRs than controls. The mean amplitude of the SSR was significantly reduced in both lower and upper limbs of PD patients in comparison with control subjects ($p<0.001$). The onset latency was longer in the lower limbs of these patients in respect to the control group ($p<0.003$). There was a significant inverse correlation between SSR amplitudes and age, severity and late onset of the disease. There was no association of these parameters with dysautonomic symptoms or R–R interval variation. In conclusion, there is a significant association between altered SSR and PD and an inverse correlation in this group of patients between SSR values and older age, greater severity and later onset of disease. Therefore, the study of SSR may provide valuable information on cholinergic sympathetic function in patients with PD.
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Keywords: Sympathetic skin response; Sympathetic nervous system; Parkinson's disease; Autonomic nervous system

1. Introduction

Signs and symptoms of autonomic nervous system (ANS) dysfunction are frequently found during the course of Parkinson's disease (PD) [1]. However, there is controversy on the best way to obtain objective proof of this observation. Sympathetic skin response (SSR) is a simple and non-invasive electrophysiological test used to evaluate the reflex activity of sudomotor sympathetic pathways [2–4], and it can help in the diagnosis of dysautonomia irrespectively of its etiology. The alterations of SSR in PD patients has yielded discrepant results regarding the findings on latencies and amplitudes of SSR potentials [5–10]. In most of these studies, nerve conduction tests were not performed, the severity of PD was unknown and antiparkinsonian medications were not discontinued before the sudomotor assessments. Moreover, the association of SSR results with clinical aspects of PD was also not clearly established. This study aims to evaluate the presence of alterations of SSR in patients with PD as compared to healthy controls, as well as to identify associations of these alterations with clinical aspects of the disease and other tests of autonomic function.

2. Methods

We studied patients diagnosed with PD [11] in the movement disorders clinic of a university hospital in southern Brazil from May 2003 to March 2005. All patients with no exclusion criteria who agreed to participate in the study were examined consecutively in a cross-sectional design. Exclusion criteria were as follows: (a) vascular, pharmacological or atypical

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parkinsonism, i.e., multiple system atrophy (MSA), progressive supranuclear palsy; (b) presence of other diseases potentially involving the ANS, i.e., diabetes mellitus, cerebrovascular diseases; (c) use of drugs influencing the ANS, i.e., beta blockers, tricyclic antidepressants; (d) peripheral nerve diseases, i.e., carpal tunnel syndrome, polyneuropathy, etc., detected by nerve conduction studies; (f) absence of signed informed consent. Apart from PD patients, we also studied a group of patients of the same age range (control subjects), selected from those referred for electromyography and nerve conduction studies for a variety of reasons, in whom extensive electrodiagnostic workup did not show signs of peripheral nerve disease or autonomic dysfunction. A conventional electrophysiological testing of nerve conduction was performed according to standard methods [12,13] and patients who had any abnormal electrophysiological sign were excluded. All patients and controls gave their informed consent for the study, which was approved by the Ethical Committee of our Institution.

Patients who met criteria for the study were scheduled for clinical assessment, including the Hoehn and Yahr Scale (HYS) [14], and interviewed using a questionnaire on parkinsonism and dysautonomic symptoms. Blood pressure measurements, electrocardiogram (ECG) and SSR were performed after discontinuation of all antiparkinsonian medications for 14 h. Blood pressure measurements were performed with patients lying down and after 2 and 5 min standing upright. Orthostatic hypotension was defined as a decrease of 20 mmHg or more in systolic blood pressure or 10 mmHg or more in diastolic blood pressure in either one of the upright measurements, according to a consensus statement on the definition of orthostatic hypotension, pure autonomic failure and MSA [15]. ECG and SSR recordings were performed by a trained neurophysiologist, blinded with respect to the clinical evaluation of the patients, using a *Medelec Synergy*® electroneuromyography equipment. ECGs were recorded over a 5-s screen with simultaneous recording of respiratory movement by the use of an elastic belt, with filter settings of 0.1 and 30 Hz. The average interval between R waves was recorded, and R–R interval variation was defined as the difference between the average of the six longest intervals during expiration and the average of the six shorter intervals during inspiration. Interval variations of less than 10 beats per minute were considered as abnormal [16].

SSR evaluation was performed under suitable environmental conditions [3,4]. Bilateral upper limb and left lower limb studies were performed in PD patients, while left upper limb and left lower limb studies were performed in controls, as there are no significant differences between SSR values for both sides of the body in normal individuals [5,17]. Upper limb recordings were performed with surface electrodes placed on the palmar region (second interdigital space, 3 cm proximally to the metacarpophalangean articulation), with reference electrodes placed on the dorsum of the hand. Lower limb recordings were performed on the plantar region (first interdigital space, 3 cm proximally to the metatarsophalangean joint), with reference electrodes placed in the distal phalanx of the second digit. A 10-s screen was used for lower limbs and a 5-s screen was used for upper limbs, with 200–1000 μV sensitivity and filter settings of 0.1 and 2 kHz. Electric stimuli were administered on the contralateral limb, at an intensity of 30 mA and duration of 0.2 ms. Five recordings were performed for each limb, with a minimum interval of 30 s between stimuli. The widest amplitude response, measured by peak-to-peak, and its corresponding latency were used for each limb. For patients in whom both upper limbs were studied, the limb presenting the largest amplitude was used. Amplitudes and latencies were defined as abnormal when they were lower than 1st percentile and higher than the 99th percentile from the normal group, respectively. Absent responses were considered when no consistent change in the baseline larger than 50 μV s was observed in the 2 s following the stimulus in any one of the recordings.

The data were entered in Microsoft Excel 2003 and later transferred to SPSS 10.0 for Windows. A paired *t*-test was performed to compare data of amplitude and latency obtained in both hands from the same individuals. Normality of distribution of the data was assessed using Kolmogorov–Smirnoff. Posteriorly, comparisons between PD patients and control subjects regarding SSR latencies were performed using Student's *t*-test, while SSR amplitudes were compared using Mann–Whitney's *U*-test. Absent responses were not included in the statistical analysis. Correlations between continuous variables were performed using Spearman's correlation coefficient for amplitudes and Pearson's correlation coefficient for latencies. Categorical variables were analyzed using a χ^2 test. A value of $p<0.05$ was considered to indicate statistical significance.

3. Results

Forty-six PD patients (27 men, 19 women, 39–82 years, mean age 63.7 years) and 64 controls (30 men, 34 women, 44–85 years, mean age 65 years) were studied. The mean age of disease onset was 56.7 ± 11.5 years old, with an average time of disease of 6.9 ± 4.1 years. The mean score on the HYS was 2.7 ± 1.0, while the mean score on the Daily Living Activities Scale was $74.1\pm20.9\%$. There was no difference between PD patients and control subjects (64 subjects) with regard to age ($p = 0.4$), sex distribution ($p = 0.1$), weight ($p = 0.2$) and height ($p = 0.2$).

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