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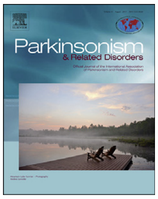

Trunk muscle activation pattern in parkinsonian camptocormia as revealed with surface electromyography

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ABSTRACT

Introduction: Camptocormia is frequently seen in Parkinson's disease (PD) and multiple system atrophy. It is characterized by a pathological forward bending of the trunk during standing, often combined with a lateral trunk deviation. The etiology of camptocormia in PD is still unknown. Muscle MRI studies show abnormalities mainly of the erector spinae confirmed by muscle biopsies. Quantitative electromyographic examination of trunk muscle activity is missing.
Methods: Ventral (rectus and obliquus externus abdominis) and dorsal (iliocostalis lumborum, longissimus, multifidus) trunk muscles and the rectus femoris were recorded bilaterally with surface electromyography in standing PD patients with camptocormia ($n = 10$) and matched healthy controls ($n = 10$) who mimicked the patients' posture. EMG amplitudes were compared quantitatively. In controls, the relation between varying degrees of trunk flexion and EMG was established systematically.
Results: Increasing forward trunk flexion was associated with increasing back muscle activity in controls, while abdominal muscle activity was negligible. During anterolateral trunk flexion, back muscle activity increased particularly on the contralateral side. The patients showed a similar pattern. However, normalized EMG activity of their trunk extensors was significantly higher than in controls, often reaching half-maximal amplitudes. Their rectus femoris and oblique abdominal muscles were overactive, but to a lesser extent.
Conclusions: PD patients with camptocormia must use the functional reserve of their lumbar trunk muscles to counteract gravity. We interpret this as a weakness of the paravertebral muscles. Compared to the other examined muscles the paravertebral muscles are most affected. The increased EMG activity of the rectus femoris warrants further research.

1. Introduction

Camptocormia is a postural abnormality characterized by a pathological forward bending of the trunk seen in myopathic disorders, but also in Parkinson's disease (PD) and multiple system atrophy. Camptocormia is present when the patients stand or walk, but shows an almost complete resolution in the supine position [1,2]. The causes of camptocormia are not uniform. In myopathic camptocormia, the myopathically induced weakness is obviously responsible for the forward bending [3]. In dystonic camptocormia, the dystonic movements lead to an abnormal bending of the trunk [4,5]. In Parkinson's disease (PD), the cause is unknown and different approaches have been used to understand parkinsonian camptocormia. Muscle biopsies of paravertebral muscles revealed myositis in rare cases [6,7], but the majority of PD patients with camptocormia have shown a myopathic pattern reproducible and characteristic for camptocormia [8]. An MRI study of paravertebral muscles displays a dynamic process of muscle abnormalities initially with edema and swelling followed by a progressive fatty degeneration [9]. The MRI findings imply an impairment of lumbar trunk muscles beyond an isolated involvement of the paravertebral muscles. The present study was designed to understand ventral and dorsal trunk muscle activation during forward bending in healthy controls and to compare quantitatively their electromyographic (EMG) pattern with patients with parkinsonian camptocormia.

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2. Methods

2.1. Patients and controls

Ten patients with idiopathic PD, according to British Parkinson's Disease Society Brain Bank criteria [10], and camptocormia were recruited: age 76.1 ± 6.8 years (mean ± SD), body height 167 ± 10 cm, weight 58.8 ± 9.8 kg, body mass index 21.1 ± 3.6 kg/m^2 (see supplementary data for details). Camptocormia is defined as an anterior flexion of the thoracolumbar spine of at least 30°, appearing while standing or walking but disappearing in a recumbent position. It is mostly combined with an additional laterodeviation of the trunk. The angles of anterior and lateral bending are determined from pictures taken from the front and side as previously published [11] of patients standing free without any effort to straighten the trunk. The patients were on their regular parkinsonian medication. Ten healthy control subjects (age 75.0 ± 5.0 years, body height 172 ± 9 cm, weight 69.3 ± 11.7 kg, body mass index 23.4 ± 2.1 kg/m^2) were matched to each patient for age and sex. The ethics committee of the Medical Faculty of Kiel University approved the study and all participants gave informed consent.

Surface electromyography was performed while the PD patients with camptocormia were standing in their own typically bent posture for at least 30 s. Each control subject imitated the posture of the matched patient based on photographs and aided by an investigator. EMG signals of six muscles pairs were recorded in that posture simultaneously from both sides of the body: rectus femoris (RF), obliquus externus abdominis (OEA), rectus abdominis (RA), iliocostalis lumborum (ICL) muscle, and paravertebral erector spinae at the thoracic level (EST; longissimus m.) and lumbar level (ESL; multifidus m.). Following skin preparation, pairs of Ag–AgCl electrodes (Arbo® H124SG, Germany) with a circular uptake area of 1.5 cm diameter and an inter-electrode distance of 3 cm were attached to the skin above each muscle. Electrode positions followed the literature [12] and established guidelines [13]; see supplementary fig. A. For EST and ESL, electrode pairs were placed 3 cm lateral of the spinous process of vertebra T12 (EST) and vertebra L3 (ESL). Raw EMG data were recorded with a telemetry EMG system (12 channel Noraxon® Desktop DTS, Scottsdale, AZ, USA) with differential amplifiers at a bandwidth of 10–1500 Hz (common mode rejection ratio >100 dB, baseline noise < 1 μV rms, input impedance >100 MOhm). EMG signals were digitally low-pass filtered (500 Hz cut-off) and stored in a computer together with synchronized video images for offline processing using Noraxon® software (MR3 MyoResearch).

The EMG data were full-wave rectified and smoothed with a root mean square window of 50 ms. The absolute EMG signal amplitudes (μV) were then normalized to the individually highest activity level reached during maximal voluntary contractions (MVC, mean amplitude over 0.5 s) and were then expressed as a percentage of this maximal signal (%MVC). Such normalization is an established method to minimize the influence of varying anatomical and physiological conditions (e.g. skin thickness and conductance) between different subjects and muscles. Normalization is essential if comparisons between subjects or groups are sought [14]. Exercises to generate maximum activity for each muscle were performed by control subjects [15], and also by the patients who were encouraged to expend a maximum effort.

The average EMG activity of each muscle was calculated across 30 s of quiet standing in each participant. The absolute and the normalized EMG amplitudes were compared between groups with Mann-Whitney U-tests; patient data were not normally distributed (Kolmogorov-Smirnov test). Wilcoxon-tests were used to contrast EMG activity on the leaning side (ipsilateral muscles) with activity of the contralateral muscles. The same tests were also used to compare the activation increases found in different muscles amongst each other. Here the EMG activity measured in the control subjects served as baseline level.

In addition, we collected EMG data from all control subjects during predefined angles of anterior and anterolateral trunk flexion. Flexion angles were controlled with an optoelectronic motion analysis system (MacReflex Qualisys®, Partille, Sweden). Two spherical reflective markers were attached to the body above the spinous processes of cervical vertebra C7 and lumbar vertebra L4. These markers appeared as bright dots on a monitor located in front of the standing participant. To reach a particular angle of trunk flexion, the standing participant bent the trunk until the two markers aligned with the lines on the monitor that marked a defined degree of flexion. We collected data during upright standing (baseline), forward trunk flexions (of 30°, 50°, 80°), and anterolateral trunk flexions. Here the subjects bent forward (30°, 50°, 70°) and simultaneously toward one side (left/right) in oblique directions that were marked by arrows on the floor (pointing 45° leftward or rightward). Each posture (i.e. trunk flexion angle) was maintained for at least 30 s. Mean normalized EMG amplitudes (% MVC) were calculated for each flexion angle and also for the baseline condition (upright standing) over 30 s each. Increasing muscle activity during trunk flexion was calculated as a deviation from the baseline signal measured during upright standing. To identify significant changes, we performed separate analyses of variance for repeated measures (ANOVA) for the different muscles during forward and anterolateral trunk flexions. Within-subject factors were the postural angle, the side of the body (left/right muscles) and the direction of the flexion (leftward/rightward). Paired t-tests were then applied to compare EMG activity during a specific posture (e.g. 30° forward flexion) with the baseline activity during upright standing. Thus we established the physiological relationship between the degree of anterior/anterolateral trunk flexion and the associated EMG activity in the control group.

3. Results

3.1. Control subjects

Anterior flexion of the trunk was associated with significant (Anova and post-hoc t-tests, $p < 0.05$) increases in the EMG activity of the three examined back muscles (EST, ICL, ESL) above baseline level (Fig. 1A, and see supplementary Table 1 for statistics). These increases were bilaterally symmetrical so that data on left and right muscles were pooled. The steep initial rise in the EMG activity attenuated between 30° and 50° of flexion and then arrived at a plateau. In contrast, activity of the abdominal (OEA, RA) and thigh (RF) muscles did not change significantly during forward trunk flexion in controls. Anterolateral flexion of the trunk resulted in a similar EMG pattern (Fig. 1B) but was modified by a somewhat higher activity of back muscles (ICL, ESL) contralateral to the leaning side compared to the ipsilateral side (see supplementary fig. B) and by stronger activation of the obliquus abdominis externus muscle of the contralateral side. RA and RF activity again did not deviate significantly from the baseline values of standing upright.

3.2. Patients

On clinical examination, all patients showed an almost complete resolution of the bending in the supine position. The paravertebral muscles of all patients had a wooden consistency during standing and sitting. Severe knee bending or relevant contractures were not observed in any of the patients. We did not encounter clinically

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