Fast voluntary neck movements in patients with cervical dystonia: A kinematic study before and after therapy with botulinum toxin type A

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Abstract

Objective: To study fast voluntary neck movements in patients with cervical dystonia (CD) before and after therapy with botulinum toxin type-A (BTX-A).

Methods: A selected sample of 15 patients with CD (with prevalent torticollis) and 13 age-matched control subjects performed both right and left rotational, and flexion and extension neck movements as fast as possible. Movements were recorded with a motion analysis system (SMART, BTS). Movement time, angular amplitude, and peak angular velocity were analyzed. In patients, rotational neck movements were pooled as “pro-dystonic” (toward the dystonic side) and “anti-dystonic” (toward the non-dystonic side). Results obtained in patients before BTX-A treatment were compared with those of control subjects. The effect of BTX-A treatment was evaluated by comparing movement performance before and after treatment.

Results: Before receiving BTX-A, patients performed pro- and anti-dystonic movements with lower peak angular velocity than control subjects. Pro-dystonic movements had a reduced angular amplitude. Anti-dystonic movements showed an abnormally long movement time. Flexion and extension movements required longer movement times, but the other kinematic variables were normal. After BTX-A injections, pro-dystonic movement amplitude and anti-dystonic movement peak angular velocity increased, whereas flexion and extension movements remained unchanged.

Conclusions: Before BTX-A injection patients with CD perform fast voluntary neck movements abnormally and BTX-A injections improved their peak velocity and amplitude.

Significance: Kinematic studies can detect specific neck movement disturbance in patients with CD, and can quantify both the severity of clinical picture and the effect of BTX-A injections in these patients.

Keywords: Cervical dystonia; Neck movements; Kinematics; Botulinum toxin

1. Introduction

Cervical dystonia (CD), one of the most common forms of focal dystonia, is characterized by prolonged muscle contractions, causing abnormal postures and involuntary movements of the head and neck. In patients with CD neurophysiological investigations show abnormalities at various central nervous system levels. These changes suggest altered inhibitory mechanisms due to basal ganglia abnormalities and to dysfunction of the cortico-striato-thalamo-cortical circuits (Berardelli et al., 1998). Kinematic studies show that patients with focal arm or generalized dystonia are bradykinetic when performing fast voluntary arm movements (Van der Kamp et al., 1989; Agostino et al., 1992; Inzelberg et al., 1995; Berardelli et al., 1998; Currà et al., 2000, 2004; Prodoehl et al., 2006a). Patients with dystonia are also slow in switching from one movement to the next during fast sequential
arm movements (Agostino et al., 1992; Currà et al., 2000) and fast internally triggered movements are more affected than fast externally triggered movements (Currà et al., 2000). Whether CD patients performing fast neck movements are bradykinetic is unknown.

Only two studies have examined voluntary neck movements in patients with CD. In a small study sample, Carpaneto et al. (2004) reported that head maximal excursion is altered in CD. In a study performed with an electromiometer, Salvia et al. (2006) reported a decrease in the velocity of voluntary head movements and, to a lesser degree, in the range of head motion. Both studies had the limitation that movement instructions gave no indications on how fast subjects should move. The lack of explicit instruction on the velocity of movement to be performed makes it difficult to establish if CD patients were bradykinetic in performing neck movements.

Although botulinum toxin type-A (BTX-A) injected into the cervical muscles improves the abnormal head posture in patients with CD (Jankovic, 2004) no kinematic study has addressed the effects of BTX-A injections on fast voluntary neck movements in patients with CD.

In this study, we analyzed movement time, angular amplitude and peak angular velocity of fast voluntary rotational (i.e. lateral rotation in the yaw plane) and flexion and extension neck movements in patients with CD who had prevalent rotational head movements. First, we were interested to see whether CD patients had bradykinesia during fast voluntary head movements. Movements toward the side of the dystonic head deviation (“pro-dystonic movement”) were compared with movements toward the side opposite to the dystonic head deviation (“anti-dystonic movement”) and both were compared with neck movements of a healthy control group. The reason for separating pro-dystonic and anti-dystonic movements is because during pro-dystonic movements agonist muscles are those involved by the dystonic activity whereas during anti-dystonic movements the dystonic activity is present in the antagonist muscles.

We then investigated the effects of BTX-A injected into neck muscles on voluntary neck movements. We predict that the effect of BTX-A treatment on kinematic features of fast head movements should be different for pro- and anti-dystonic head movements. The effect of BTX-A treatment on flexion and extension head movements should be less evident than that seen during rotational head movements, since flexion and extension head movements use as agonist both treated and untreated muscles.

2. Materials and methods

2.1. Subjects

We studied 13 right handed control subjects (6 men and 7 women; mean age: 58 ± 18.1 years; range: 27–75 years) and 15 right handed patients who had primary CD with prevalent torticollis (7 men and 8 women; mean age 59.5 ± 14.9; range: 26–85 years). The diagnosis of CD was made following previously published standard criteria (Dauer et al., 1998). Exclusion criteria were primary segmental or generalized dystonia, dystonia plus, heredo-degenerative disease, and secondary or iatrogenic dystonia. All patients with CD we studied had been treated with BTX-A (Botox) for a mean 5.8 ± 3.7 years (Table 1). All patients were clinically evaluated and selected by clinicians expert in movement disorders. Patients were also videotaped and the recordings were separately observed by two expert clinicians. A selected sample of CD patients with clear rotational head movements (i.e. patients with unequivocal involuntary head rotation in the yaw plane) and with matching dystonic side between the two observers were included in the study. Patients with concomitant shoulder elevation or with significant painful symptoms which could interfere with neck movements were excluded.

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<th>Patients</th>
<th>Age</th>
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<th>BTX-A therapy duration</th>
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Age, disease and botulinum toxin type-A (BTX-A) therapy duration are expressed in years. Abreviations: SPL, splenius capitis muscle; SCM, sternocleidomastoideus muscle; TRP, trapezius muscle; SCAL, scalenus anterior muscle; r, right; l, left.
All the participants gave their informed consent to the study.

2.2. Analysis of movements and experimental setting

The head movements were recorded with the motion analyzer system “SMART” (Bioengineering Technology and System – BTS, Milan, Italy). The system used three infrared ray TV cameras at 120 Hz sampling frequency put in front of the subject. The distance between the cameras was 70 cm and the subject seated in front of the central camera at a distance of one meter and half with the other two cameras on the left and on the right at a distance of approximately two meters. These distances were maintained constant in all sessions of the study. The cameras separately recorded the movements of an array of reflecting passive markers placed on six body parts, three over the head (two over the frontal orbital processes and one over the nose) and three over the trunk (two over the acromions and one at the level of the sternum). A dedicated software digitized the data recorded by the three infrared cameras and reconstructed the coordinates of the head markers movements in a three-dimensional X–Y–Z reference system. The used camera configuration allows to have the same accuracy in each direction in the calibrated 3D space. Head kinematics data were calculated by performing a 3D kinematic computational analysis of head angular movements in terms of displacement of the head coordinate system (derived from the three head markers) within a fixed trunk coordinate system (derived from the three trunk markers). In the head coordinate system the X axis is parallel to the segment connecting the two frontal markers, the Z axis is perpendicular to the X axis and the Y axis is perpendicular to the X–Z plane. In the trunk coordinate system the X axis is parallel to the segment connecting the shoulder markers, the Z axis is perpendicular to the X axis and the Y axis is perpendicular to the X–Z plane. In the Smart Analyzer software the angular velocity of each movement is computed as the time derivative of the angular displacement of the head coordinate system within the fixed trunk reference system. This procedure allowed us to avoid measurement pitfalls from different head morphology and trunk position and orientation.

The subjects were seated comfortably on a chair placed in front of the three infrared cameras. Before each movement an operator kept the subject’s head in the primary position and then released it just before the go signal (see below). The subjects were asked to perform right and left rotational, flexion and extension neck movements, and to move “as fast and widely as possible”. For each type of neck movement, three blocks of five consecutive movements were performed in randomized order, so that for each subject a total of 60 neck movements were recorded (15 movements each for right and left rotational and 15 movements each for flexion and extension). In order to avoid fatigue between each block there was a time interval of about five minutes. Each movement was performed after a verbal go-signal although subjects were not specifically instructed to start as soon as possible after the mandatory signal to go and were left free to initiate the movements at their will. In each block, neck movements were performed at intervals long enough (5–10 s) to allow the subject to return to the primary position. In order to study movements with the same starting position, after each movement the head of the subjects was positioned in the primary position before the go-signal for the next movement was given.

2.3. Kinematic measurements

Neck movements were analyzed off-line by means of sub-routines included in the dedicated software which automatically calculated movement time, angular amplitude and peak angular velocity. The beginning and end of each movement were arbitrarily defined on the basis of angular velocity so that the movement began and finished when 10% of the peak angular velocity was reached. Since a preliminary analysis of the data showed that peak angular velocity was approximately halved in patients as compared to controls, a fixed velocity value in both groups would have added a bias in the measurement of the beginning and end of each movement, leading to an under-estimation of movement time in patients and to an over-estimation of movement time in controls. Hence, the 10% value allowed to have consistent inter-group measurements. The angular amplitude and velocity were measured on the three axes of the reference system and the vectorial modules of these kinematic variables were calculated and used for subsequent analysis.

Because a preliminary analysis showed that control subjects performed right and left rotational neck movements with similar kinematic variables, for subsequent analysis, the data from right and left movements were pooled. In patients, regardless of whether rotational neck movements were performed toward the right or the left side, “pro-dystonic” movements (toward the side of the dystonic head movements) were pooled and compared with “anti-dystonic” movements (toward the side opposite to the direction of the dystonic head movements). As the patients were prevalently affected by torticollis, neck flexion and extension movements (which are not involved by the dystonic movements) were analyzed separately.

2.4. BTX-A treatment

Patients were studied in two separate sessions: first at baseline (i.e. at least three months after the last BTX-A treatment and just before the new BTX-A injections) and three weeks after the new BTX-A treatment (Table 1). In each session patients were also evaluated by the same clinician face-to-face separately using the Tsui clinical scale for spasmodic torticollis (Tsui et al., 1986) (Table 1). Patients were also evaluated by recording a videotape of head dystonic movements before and after BTX-A treatment. Total Tsui scale scores and sub-scores for amplitude and
duration of dystonic movements, and for tremor were considered in statistical analysis.

2.5. Statistical analysis

In each subject and for each type of neck movements, the grand-averages of 15 head movements were calculated and then used for statistical analysis. Analysis was performed with the “STATISTICA” software package for windows release 6.1. Rotational and flexion–extension neck movements were analyzed separately because CD patients presented mainly rotational dystonic movements in the yaw plane.

In order to analyze multiple variables in two groups of subjects, a multivariate analysis of variance (MANOVA) was used to compare the kinematic variables of the different groups of subjects with the between group factor MOVEMENT (control, pro-dystonic and anti-dystonic for analysis of rotational neck movements, and control-extension, control-flexion, CD-flexion, CD-extension for analysis of flexion and extension neck movements). A Within-Subjects (Repeated Measures) ANOVA was used to compare data obtained in patients before and after BTX-A injections with repeated measure factor TIME (pre and post BTX-A injections). For both ANOVA tests, Tukey honestly significant difference (HSD) test was used for post hoc analysis of data.

A multiple regression analysis was used to study correlations between kinematic variables and clinical data. In CD patients, all kinematic variables of each type of neck movements were correlated with age, disease duration, treatment duration and BTX-A dose injected, and Tsui scale score. Correlations between kinematic variables of rotational neck movements were also studied in controls and CD patients. A correlation analysis was also performed between percentage variation of each head kinematic variable and Tsui scale score before and after BTX-A treatment. P-values < 0.05 were considered significant.

3. Results

3.1. Pro-dystonic and anti-dystonic rotational neck movements before BTX-A therapy

In patients with CD, pro-dystonic and anti-dystonic neck movements were both altered (Fig. 1). MANOVA showed a significant effect of between-group factor MOVEMENT ($F_{6,58} = 4.61, p < 0.001$).

Post hoc analysis showed that movement times for anti-dystonic rotational neck movements were longer in patients than in control subjects ($p < 0.001$). Their peak angular velocity only tended to increase after BTX-A treatment. BTX-A treatment left pro-dystonic and anti-dystonic rotational neck movement times unchanged (Table 2).

3.2. Flexion and extension neck movements before BTX-A therapy

MANOVA showed a significant effect of the between-group factor MOVEMENT ($F_{9,122} = 3.11, p < 0.001$).

Post hoc analysis showed that patients with CD performed both flexion and extension neck movements with longer movement times than control subjects ($p < 0.001$). The angular amplitude and peak angular velocity were not significantly different from control subjects, although these measures tended to have lower values in patients than in control subjects (Table 3).

3.3. Pro-dystonic and anti-dystonic rotational neck movements after BTX-A therapy

In patients with CD, after treatment with BTX-A pro-dystonic and anti-dystonic rotational neck movements both improved. ANOVA showed a significant effect of the between-group factor MOVEMENT ($F_{3,15} = 12.21, p < 0.01$) and of repeated-measure factor TIME ($F_{3,15} = 12.05, p < 0.01$), while the interaction between the two factors was not significant ($F_{3,15} = 1.33, p = 0.38$).

Post hoc analysis showed that, after treatment with BTX-A, peak angular velocity of anti-dystonic rotational neck movements significantly increased ($p < 0.01$). The pro-dystonic movements significantly increased in angular amplitude ($p < 0.05$). Their peak angular velocity only tended to increase after BTX-A treatment. BTX-A treatment left pro-dystonic and anti-dystonic rotational neck movement times unchanged (Table 2).

3.4. Flexion and extension neck movements after BTX-A therapy

In patients, the treatment with BTX-A left flexion and extension neck movements unchanged. ANOVA showed no significant effect of the between-group factor MOVEMENT ($F_{3,20} = 1.5, p = 0.22$) and of the repeated-measure factor TIME was not significant ($F_{3,20} = 1.02, p = 0.34$).

The two-way interaction MOVEMENT × TIME was not significant ($F_{5,20} = 0.78, p = 0.77$) (Table 3).

3.5. Clinical correlations

In patients with CD, no significant correlations were found between the clinical factors studied, age, disease duration, duration of treatment and BTX-A doses, and kinematic variables for the four neck movements studied.
Fig. 1. Curves of angular amplitude and velocity of rotational head movements in one control subject and in one CD patient. The figure shows five consecutive angular amplitude (one peak trace – gray line) and velocity (two peak trace – black line) of rotational head movements of a single trial. The rising phase of the amplitude trace and the first peak of the velocity trace correspond to a fast voluntary head movement. The decline phase of the amplitude trace and the second peak of velocity trace represent the slow voluntary movement performed by the subjects in order to reach the starting position. Angular amplitude is expressed in degrees. Angular velocity is expressed in degree/s.
Before BTX-A treatment, no significant correlations were found between the total Tsui scale scores and kinematic variables for the neck movements studied.

Multiple regression analysis identified no significant correlations between the percentage variations in the total Tsui scale scores and kinematic variables after BTX-A treatment. Conversely, the analysis of Tsui scale sub-scores disclosed a significant inverse correlation between changes in scores for dystonic movement amplitude and peak angular velocities of pro-dystonic movements ($p < 0.05, R^2 = 0.60$). A significant inverse correlation was also found between variations in Tsui scale sub-scores for dystonic movement duration and peak angular velocities of anti-dystonic movements ($p < 0.05, R^2 = 0.62$).

In controls and CD patients correlation analysis between kinematic variables of rotational neck movements showed a significant inverse correlation between movement time and peak angular velocity (controls: $p < 0.05 R^2 = -0.80$). Pro-dystonic neck movement time was inversely correlated with peak angular velocities before ($p < 0.05 R^2 = -0.72$) and after BTX-A treatment ($p < 0.05 R^2 = -0.69$). Anti-dystonic neck movement time also showed an inverse correlation with peak angular velocities before ($p < 0.05 R^2 = -0.71$) and after BTX-A treatment ($p < 0.05 R^2 = -0.59$). On the other hand, angular amplitudes showed no correlations with the other kinematic variables in controls and in CD patients.

### 4. Discussion

Our kinematic results provide evidence for the first time that patients with CD manifested predominantly by torticollis are bradykinetic in performing fast pro-dystonic and anti-dystonic rotational voluntary neck movements. Their pro-dystonic head rotations are also significantly reduced in amplitude. The amplitude reduction might explain why, differently from anti-dystonic movement, the movement time was not prolonged in pro-dystonic movements. Patients are also bradykinetic in performing flexion and extension neck movements.

Carpaneto et al. (2004) and Salvia et al. (2006) reported an abnormal movement amplitude, but since the authors did not give any instructions about movement speed, no clear conclusion about the presence or not of bradykinesia in CD patients can be drawn from their studies. In our study, we analyzed neck movement performed under the instruction to move as fast as possible and this allowed us to conclude that CD patients are bradykinetic. Indeed, since during fast movements peak velocity covariates with movement amplitude (Marsden et al., 1983; Berardelli et al., 1996), it could happen that the primary alteration in patients would be a reduced amplitude. The prolonged movement time and/or the reduced peak angular velocity found in this study lead us to conclude that CD patients were truly bradykinetic. After BTX-A injections pro-dystonic rotational neck movements become significantly larger in amplitude and anti-dystonic movements become faster. Treatment with BTX-A leaves head flexions and extensions unchanged.

Our kinematics data in patients with CD agree with previous studies showing slow performance of fast voluntary arm movements in patients with arm dystonia (Van der Kamp et al., 1989; Agostino et al., 1992; Inzelberg et al., 1995; Currà et al., 2000). Movement slowness has also been reported in patients with hand dystonia performing finger
movements (Currà et al., 2004). Electromyographic studies in patients with arm dystonia showed that the execution of voluntary arm movements is characterized by co-contraction of antagonist muscles and by activation of remote muscles not directly involved in the voluntary movement (Van der Kamp et al., 1989).

The reason why fast rotational neck movements are slow in patients with CD is unclear. Although we did not measure neck muscle strength, in agreement with recent data in patients with focal arm dystonia (Prodoehl et al., 2006b), it is possible that slowness of movements is due to a reduced muscle force of neck muscles. Another mechanism, similar to that proposed for rapid arm movements in patients with arm dystonia (Van der Kamp et al., 1989), is that the slowness of fast voluntary neck movements we observed could arise from co-contraction of agonist and antagonist neck muscles. Indeed, co-contraction during rotational neck movements has already been reported in patients with CD (Kaji et al., 1995). Because all our patients had CD manifesting mainly as torticollis and the dystonic muscles causing rotational neck dystonia are also active during head flexions and extensions, the slowness during these head movements may depend also on inappropriate force and EMG activation of neck muscles.

The slowness of fast voluntary head movements may reflect basal ganglia dysfunction in CD that in turn results in abnormal activation of cortical motor areas as it has been suggested by functional imaging studies in patients with CD (Stoessl et al., 1986; Galardi et al., 1996; Magyar-Lehmann et al., 1997). Changes in cortical excitability in patients with CD have been reported in studies using neurophysiologic investigations. Studies recording the contingent negative variation showed abnormal preparation for phasic neck movement (Kaji et al., 1995) and transcranial magnetic stimulation investigations suggested altered excitability of the primary motor cortex in patients with CD (Hanajima et al., 1998; Kanovsky et al., 2003).

In the present study, BTX-A treatment improved both pro-dystonic and anti-dystonic neck movements. With respect to pro-dystonic movements, it is worthy to note that despite the expected weakness due to BTX-A injections in the agonist muscles, this treatment did not impair movement duration and velocity. Our patients’ improved performance of fast voluntary neck movements after BTX-A therapy fits in well with current knowledge on the clinical effects of BTX-A in dystonia. Clinical studies show that BTX-A improves dystonic posture and reduces involuntary movements in patients with CD (Jankovic, 2004). Neurophysiologic studies suggest that the clinical effects of BTX-A are primarily due to peripheral chemodenervation of the muscles injected (Hamjian and Walker, 1994; Abbruzzese and Berardelli, 2006). In a previous study, performed in six CD patients with torticollis, BTX-A injections induced a significant improvement in the range of head motion (Albani et al., 2001), but no other kinematic measures were assessed, nor were instructions given on the speed of movement to be executed. In the patients with CD we studied, BTX-A did not change movement times and peak velocity of pro-dystonic neck movements, probably because the toxin weakened the neck muscles injected with BTX-A. The reason why BTX-A improved movement amplitude of pro-dystonic movements is not easy to explain. One possibility is that the toxin reducing the dystonic activity in the agonist muscles facilitates the phasic voluntary activation (Corneil et al., 2001).

We also found that after BTX-A treatment the peak angular velocity of anti-dystonic movements improved. By reducing involuntary co-contraction in the antagonist muscles, BTX-A probably facilitates the agonist muscles, thus producing a faster neck movement. A further finding was that BTX-A left patients’ motor performance of voluntary flexion and extension neck movements unchanged, possibly because it weakened the muscles acting as agonists. Nevertheless, we cannot exclude the contribution of other neck muscles that were not treated with BTX-A, but participate in head flexions and extensions (Gabriel et al., 2004; Siegmund et al., 2007).

In the patients with CD we studied, before BTX-A therapy we found no significant correlations between kinematic variables and clinical scores on the Tsui scale. This observation agrees with a previous study in patients with CD reporting no correlations between severity scales, disability scores and kinematics and goniometric analysis of slow rotational, flexion, extension and lateral bending neck movements (Salvia et al., 2006). In our CD patients, after BTX-A treatment a correlation was present between the improvement in some of the clinical scores and changes in kinematic variables of fast voluntary head movements. These data suggest that in CD patients treated with BTX-A, the improvement of neck dystonia is associated with a better performance in executing fast voluntary head movements. Interestingly, the beneficial effect of BTX-A treatment on kinematics of pro-dystonic fast movements is related to the improvement of the Tsui scale sub-scores for dystonic movement amplitude, while the kinematics improvement of anti-dystonic movements is related to the improvement of the Tsui scale sub-scores for dystonic movement duration. It is likely that in CD patients, BTX-A reduced the amplitude and duration of dystonic movements by reducing abnormal dystonic muscle contractions.

In conclusion, CD patients with prevalent rotational head movements are bradykinetic in performing fast voluntary pro-dystonic and anti-dystonic neck movements and perform pro-dystonic movements with reduced amplitude. These patients also have difficulty in flexing and extending the head. These abnormalities can be explained by reduced muscle force, co-contraction and dystonic activity in the neck muscles, probably arising from basal ganglia-thalamo-cortical circuit dysfunction, ultimately producing an abnormal output from cortical motor areas. In CD patients with prevalent torticollis, BTX-A reduces the involuntary dystonic activity thereby improving pro-dystonic and
anti-dystonic head rotation, but it has no significant effects on head flexion and extension which are less involved in abnormal dystonic movements. The analysis of kinematics of neck movements in CD patients can be a reliable tool in quantifying both the severity of clinical picture and the effects of BTX-A treatment.

References


