

Research Article

Cognitive Theory of Mind Deficit Associated with Executive Dysfunction in Cervical Dystonia

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Introduction. Cervical dystonia (CD) is viewed as a circumscribed movement disorder. However, beyond motor symptoms, it can imply subtle cognitive change, among others with respect to theory of mind (ToM) capacities. Here, affective and cognitive ToM performances and potential relations to other mental functions were investigated to refine the concept of social cognitive dysfunction in CD. *Methods*. 20 persons with CD were clinically assessed, together with 20 healthy controls engaged in overview cognitive testing, executive function (EF) tasks, and the Faux Pas Recognition Test (FPRT) as well as the Reading the Mind in the Eyes Tests (RMET) addressing cognitive and affective ToM functions, respectively. *Results*. Persons with CD showed lower cognitive, but not affective ToM performance than controls. Further, they had abnormally low word production in verbal fluency (VF) tasks, imposing high EF demands. Generally, ToM performance correlated with VF and, further, with the decreased quality of life score in persons with CD. *Conclusion*. Cognitive ToM deficits seem to occur in the context of executive dysfunction in CD. They belong to an underrecognized spectrum of nonmotor symptoms of likely clinical relevance.

1. Introduction

Cervical dystonia (CD) is a condition with relatively circumscribed motor symptoms. Typical features are abnormal postures of the head, neck, and shoulders due to involuntary spasmodic and sometimes tremulous muscle activity [1]. Mainly originating from subcortical motor network dysfunction, CD is categorized as a neurological movement disorder [2]. Accordingly, medical treatment aims at reducing the positional abnormalities, mostly by injecting botulinum toxin into affected muscles. Only in cases refractory to this therapy, deep brain stimulation is used to counteract pathological basal ganglia signaling.

Against the background of these leading symptoms, cognitive abnormalities are generally less noticed in CD

[3], but dedicated studies consistently identified reduced processing speed as well as attentional, executive, visuospatial, and language deficits in patients with primary dystonia, including CD [4, 5]. Only scarce and heterogeneous findings are available with respect to social cognitive, in particular theory of mind (ToM) capacities in CD patients [6–8]. ToM as a prerequisite of interactional behaviors is of paramount importance in daily living. It reflects different processes for building assumptions about the mental condition of others, for example, from the observation of mimics or gestures [9], and is commonly divided into two domains. "Affective" ToM denotes rather automatic processes enabling one to capture the emotional state of perceived persons, altogether giving rise to empathy-related behaviors. "Cognitive" ToM is considered as the active reasoning about

others' beliefs and thoughts, in order to interpret social situations and interactions [10, 11]. Cognitive ToM is considered to imply overlap with executive functions, e.g., for updating the roles of characters during the evolution of their interactions, switching between the perspectives they take on each other, or retrieving memory information matching with the ongoing event [12]. Whereas a widespread cortical network with prefrontal and bilateral temporoparietal components appears to be active in either ToM dimension, specific functions for cognitive ToM were identified with respect to cuneus, precuneus, and superior temporal regions, as opposed to ventromedial frontal, anteromedial temporal, and posterior cingulate involvement in affective ToM [10, 13, 14]. Remarkably, however, ToM impairments were also found in conditions with subcortical motor pathology, most consistently in Parkinson's disease, another primary movement disorder with predominant basal ganglia motor dysfunction [15]. These findings, particularly deficits of affective ToM, were often discussed in the framework of embodied cognition (EC) theories. A central claim in these concepts is that the motor system supports the simulation of observed movements, so that body language signs like postures or facial expressions can be internally reenacted, giving rise to an experienced understanding of the perceived information [16-18]. If this "modal concept" of affective ToM were true, it would have important clinical consequences, since then conditions with motor pathologies should generally impact on corresponding abilities.

Results from studies in persons with CD remain ambiguous in this regard, suggesting both largely unaffected and impaired ToM capacities [6-8, 19]. We therefore analyzed ToM functions in persons with CD under two perspectives. Firstly, we sought to assess their performances in comparison to those of healthy controls in dedicated tests of affective and cognitive ToM for pragmatic reasons. Clinical awareness and knowledge about the nonmotor, including ToM, profile of persons with CD are scarce, although ToM deficits were found associated with lowered quality of life [19, 20]. Further, the altogether heterogeneous reports about social cognitive abilities in CD could point to distinct subgroups with spared versus declined ToM function, implying distinct disease burden. Of note, in multiple sclerosis as a neurological condition with prevalent ToM impairment, specific ToM assessment and training was proposed to strengthen social interaction and to keep the support from peers high [21]. Secondly, the assessment of either ToM dimension in relation to other cognitive test results is of interest with respect to theoretical positions and their potential practical implications. Concretely, if ToM deficits prevailed in CD as the consequence of disturbed motor function in line with EC concepts, it should become overt on the level of affective ToM and, noteworthy, be susceptible to treatments of the movement disorder. Alternatively, if only cognitive ToM functions were disturbed, whereas affective ToM remained unaffected, a relation to disturbed motor processing would appear unlikely. Instead, this constellation could be understood as the result of CD-related executive dysfunction impacting on cognitive ToM, if performances in corresponding tasks also turned out as compromised.

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2. Materials and Methods

2.1. Participants. Twenty persons with CD and twenty controls without CD took part in the present study. All participants in the CD group suffered from adult-onset idiopathic disease, characterized by dystonic muscle activity in the neck and shoulder region resulting in abnormal posture. Symptomatic causes, i.e., structural and metabolic abnormalities, as well as neurodegenerative conditions were ruled out. The final diagnosis was confirmed by the senior neurologist of the outpatient clinic for movement disorders of the Charité Campus Benjamin Franklin, where they were regularly treated with botulinum neurotoxin at intervals of three months. Exclusion criteria were comorbidities with psychiatric disease, substance abuse, overt or reported cognitive problems, and screening test results indicative of a relevant cognitive dysfunction (assessed as described below), as well as the intake of centrally acting drugs. Since German test versions were used for clinical and cognitive assessments, participants had to be native German speakers. All participants performed the tests in the same order in sessions in the outpatient clinic. They could take breaks as desired. Self-report paper-and-pencil tests (PDQ-39, FSS, and ESS; see below) were handed out and explained to the participants. After completion, they were sent back to the investigators. The participants of the CD and control groups were matched with respect to sex, age, and years of education. All participants were recruited from the pool of patients with CD and accompanying persons of the outpatient clinic for movement disorders of the Charité Campus Benjamin Franklin.

2.2. Clinical Assessments. To determine the severity of dystonic symptoms, the Tsui scale for CD was used [22, 23]. Factors included the rotational amplitude, the tilt and sagittal movement of the head, the duration of these symptoms, shoulder elevation, and tremor. The maximum score indicating highest CD severity amounted to 25 points. Health-related quality of life was addressed by the Parkinson's Disease Questionnaire (PDQ-39) [3, 24], modified for the current purpose, in that the term "cervical dystonia" replaced "Parkinson's disease" where it appeared in thirtynine questions. Response possibilities ranged from 0 (absence of the symptom) to 4 (symptom always present). Domains assessed by the questionnaire were activities of daily life, emotional well-being, social support, communication, bodily discomfort, perceived stigma, and subjective cognitive and communicative impairment. The score was transformed to values between 0 and 100, the latter indicating the highest possible reduction of health-related quality of life. Fatigue as a common symptom in chronic disease with possible impact on task performance was measured by the Fatigue Severity Scale (FSS, 9-item Likert type), comprising nine questions with gradings from 1 to 7, so that a maximum score of 63 points reflected most severe fatigue [25]. With respect to mood, the 21-item version of the Hamilton depression rating scale (HDRS) was used with a theoretical maximum score of 66 points, indicating the highest severity of depression (absence of depression presumed below nine points) [26]. To assess the participants' daytime sleepiness,

the Epworth sleepiness scale (ESS) was used [27]. Here, subjects rated their tendency of falling asleep in eight different scenarios on a four-point scale (0-3). Scores above ten points indicated abnormal daytime sleepiness.

2.3. Cognitive Assessments. Concerning social cognition, the Reading the Mind in the Eyes Task (RMET) and the Faux Pas Recognition Test (FPRT) were used as two standard instruments to assess affective and cognitive ToM [28, 29]. In the RMET, participants saw thirty-six photos of the eyes' region of different persons. Per picture, they had to choose the adjective (e.g., angry, sad, or happy), correctly describing the person's mental state, out of four predefined options. Scores from 0 and 36 were possible, since each correct choice accounted for one point (incorrect choices equate to zero) [28]. The RMET was used to test affective ToM performance, while the text-based FPRT focused on cognitive ToM aspects [30]. In this study, a shortened version of the FPRT was used, which comprised 10 out of the 20 original stories, of which five either did or did not describe situations entailing a social faux pas in the sense of a tactless remark [29]. After listening to the stories, participants had to judge whether a person said anything awkward, which addressed the general ability to recognize a faux pas (detect). Further questions tested the understanding of the inappropriateness of remarks (inappropriateness), intentions of involved persons (intentions), unintentionality of the faux pas (belief), and feelings of affected persons (empathy). Per category (detect/inappropriateness/ intentions/belief/empathy), single scores expressed the percentage of correct answers of the achievable maximum across the different stories. From this, the faux pas total was built as the average of single scores (faux pas total).

To test executive functions (EF) and their potential interaction with cognitive ToM performance, the participants engaged in further tasks with high demands for lexical access, retrieval and release, conceptual switching, and working memory operations. Verbal fluency (VF) was tested by the German standard task (Regensburger Wortflüssigkeitstest) [31], demanding 2-minute production spurts for words beginning with a letter, namely, "S," (phonemic task) or belonging to a semantic category, namely, "vegetables" (semantic task). In a second run, participants had to alternate between words beginning with "G" and "R" (alternating phonemic task) and the two semantic categories "animals" and "pieces of furniture" (alternating semantic task). The participants were instructed not to name numbers and proper names and not to repeat words or words with the same word stem. Task performance was expressed as correct words per condition and the sum score over all conditions, based on the transcription of digital recordings. Further, the participants performed the digit span forward and backward tests. In these tasks, sequences from two to nine numbers were verbally presented and had to be recalled in the order of their presentation (forward) and in the reverse order (backward), respectively [32]. Each correct sequence provided one point; sum scores were built separately for backward and forward sequences as the sum of correct sequences. Since the study was part of a series of investigations implying persons with Parkinson's disease, the Parkin-

son Neuropsychometric Dementia Assessment (PANDAcognition) was used for cognitive screening, a test construct similar to the Montreal Cognitive Assessment [33, 34] and to the Mini Mental State Examination [35]. It consisted of subtasks for word paired-associate learning (for immediate (max. 5 points) and delayed recall (max. 7 points)), alternating semantic VF (naming animals and pieces of furniture alternatingly (max. 7 points)), visuoconstruction (mental mirroring around diagonal axis (max. 5 points)), and working memory (repetition of numbers reordered as sequences of rising values (max. 7 points)). Total scores ranged from 0 (worst) to 30 points (best), values below 15 points being suggestive for a major cognitive dysfunction [36]. Since the PANDA-cognition contained an alternating semantic VF subtask (requiring word generation over one minute), it was performed and calculated without this particular demand as NET-PANDA [37, 38], since otherwise only this VF condition would have been tested twice. Cognitive and clinical data were raised by the first and, to a lesser extent, the second author of the study.

2.4. Statistical Analyses. The statistical comparison between the groups was based on the results of the tests as described above expressed as raw sum scores. In the case of the PANDA, scores were age-adjusted. FPRT scores expressed the percentage of correct answers of the achievable maximum as described above. Group comparisons were run in the nonparametric Wilcoxon rank-sum tests, as most data were not normally distributed (tested with the Shapiro-Wilk test). For the analysis of the faux pas subdimensions, we corrected the *p* values for multiple comparisons by the FDR method. Effect sizes for the Wilcoxon testing (r) were reported and evaluated according to the criteria suggested by Ferguson [39]. As subdimensions of the VF test were normally distributed, we computed an analysis of variance (ANOVA) with the between-subject factor group (CD group, control group) and the within-subject factor task (semantic, phonemic) and condition (alternating, nonalternating). Effect sizes η_p^2 and the Greenhouse-Geisser corrections were reported. If interactions involving the factor group reached significance (p < 0.05), post hoc pairwise comparisons were computed. To identify clustered dysfunctions and their potential interconnections, we calculated the Spearman correlations between cognitive task performances, which were significantly different between groups. Further, to identify a possible impact on quality of life, these performances were also correlated with the PDQ-39 values. Finally, to test associations of the affected performances with CD severity, they were correlated with the Tsui scores in the CD group and, in order to identify possible confounding factors, with clinical scales significantly different between the groups.

The analyses were computed in RStudio, Version 1.2.5033, R Core Team, 2019 (https://posit.co/, RRID:SCR_000432), by the third and fourth author of the study.

3. Results

No group differences were identified with respect to age, sex distribution, and years of education (see Table 1).

TABLE 1: Demographic features of the CD and control group.

	CD group $(N = 20)$	Control group ($N = 20$)	Statistics
Sex distribution	9 females, 11 males	9 females, 11 males	
Age (years)	57.40 (13.20)	59.00 (13.50)	t(38) = 0.36, p = 0.724
High school years	11.00 (1.49)	11.30 (1.99)	<i>W</i> = 243.50, <i>p</i> = 0.224, <i>z</i> = -0.76, <i>r</i> = -0.01

Note. Standard deviations (SD) in brackets. W = test-statistics of the Wilcoxon test; z = z-value of the Wilcoxon test; r = effect size.

Our patients had mild (Tsui score below 6 points) to moderate dystonic symptoms (Tsui score of 6 to 10 points) with a median Tsui score of 4 (min = 1, max = 10; Q1 = 3, Q3 = 6.50; IQR = 3.50) [40, 41].

With respect to social cognition, the results showed that persons with CD were significantly less proficient in the FPRT than controls, indicated by lower scores in any of the particular test dimensions (i.e., detect, inappropriateness, intentions, belief, and empathy). The effect sizes of these differences were small [39]. In contrast, no group difference was identified with respect to the RMET results (see Table 2).

Persons with CD and controls did not significantly differ in the NET-PANDA score, indicating similar global cognition in both groups. Regarding the EF tests, the analysis of variance (ANOVA) for the VF data revealed a small but significant main effect of group (F(1, 27) = 5.78, p = 0.024, $\eta_p^2 = 0.18$), based on an overall reduced word production in the CD compared to the control group (16.8 (SD = 5.88))versus 20.9 (SD = 5.80)) (see Figure 1). Further, main effects of task $(F(1, 27) = 13.91, p \le 0.001, \eta_p^2 = 0.34)$ and condition $(F(1, 27) = 5.48, p = 0.027, \eta_p^2 = 0.17)$ were identified, as well as an interaction between task and condition $(F(1, 27) = 51.98, p \le 0.001, \eta_p^2 = 0.66)$. This was due to the fact that both groups produced more words in the semantic alternating than in the semantic nonalternating condition ($p \le 0.001$) as well as more words in the phonemic nonalternating than in the semantic nonalternating condition ($p \le 0.001$).

In contrast to the VF results, performance in the digit span tests did not differ between the groups (see Table 2). Post hoc power analyses (based on $G^*Power 3.1$) for the statistical models showing significant group differences in cognitive performances (i.e., in VF and FPRT) showed values of 81% and 88%, respectively [42, 43].

The CD and control groups showed similar results in the HDRS and ESS scores for depression and sleepiness, respectively (see Table 2). Significant group differences in the FSS and modified PDQ-39 suggested more fatigue and reduced quality of life in persons with CD compared to controls.

3.1. Association between ToM Performance and VF in Relation to Clinical Score Differences. Results of the correlational analyses including both groups regarding the variables which differed between groups showed that the faux pas total score and all subdimensions correlated positively with the VF sum score (r = 0.37 to 0.49, all p < 0.019). Further, the faux pas total score and all subdimensions were correlated negatively with the quality of life score (r = -0.32 to

-0.50, all p < 0.05; higher PDQ-39 scores indicating worse quality of life). In other words, higher ToM performance was associated with higher VF performance and better quality of life. However, the FSS (fatigue) score did not correlate with any other variable. Further, we did not identify a correlation between the VF or FPRT performances with the Tsui scores in the CD group.

4. Discussion

Persons with CD showed lower performances in FPRT and VF performances than controls. These group differences, altogether of small effect size, were loosely associated with reduced quality of life scores. Given the operations demanded for accomplishing social cognitive and word production tasks, overlapping underpinnings of the identified abnormalities are conceivable. Cognitive ToM depends on operations such as changing the perspective or suppressing contextually irrelevant information. In so doing, it involves key executive functions (EF) like shifting, updating, and inhibition [44-49], also required for word production in VF tasks [50]. In line with this, decline of cognitive ToM and further EF-related capacities was identified as a coupled lifespan effect of physiological aging [51], and across the participants of the current study, ToM and VF performances also correlated with each other. In contrast to FPRT performance, the RMET results did not significantly differ between the groups.

With respect to CD, executive dysfunctions count among the most regularly identified nonmotor findings. In a meta-analytic approach, their effect size was determined larger than reported here, which could, e.g., reflect the inclusion of persons with global cognitive deficits, psychiatric comorbidities, or centrally acting drugs in previous studies, so that the avoidance of these factors might have facilitated comparably favorable results in the current CD group. The same may hold true for the altogether mild to moderate degree of CD symptoms, although in the present study an association between abnormal cognitive test results and the Tsui scores was missing.

A superordinate executive dysfunction could also explain some seemingly contradictory findings about social cognitive functions in persons with CD. Similarly to the current results, the latter were found to perform normally in affective ToM tasks in two recent investigations [52, 53]. In a further study, deficits in cognitive ToM were related to working memory and semantic VF deficits [6, 8]. Besides, in persons with blepharospasm, a facial form of primary focal dystonia, deficient performance in the Wisconsin Card Sorting Test was interpreted as an indication of impaired

TABLE 2: Cognitive functions and clinical assessments of persons with CD and controls.

	CD group $(N = 20)$		Control group $(N = 20)$		Statistics			
	Mean (SD)	Median (IQR)	Mean (SD)	Median (IQR)	W	z	p	r
Social cognition								
RMET	23.20 (5.26)	23 (6)	24.80 (4.11)	24.50 (7)	240	-0.57	0.283	-0.09
Faux pas total score	0.894 (0.087)	0.88 (0.13)	0.96 (0.069)	0.99 (0.03)	291	-2.25	0.012	-0.36
Detect	0.91 (0.081)	0.90 (0.10)	0.97 (0.059)	1 (0.01)	291	-2.25	0.012^{1}	-0.36
Inappropriateness	0.90 (0.086)	0.86 (0.13)	0.98 (0.055)	0.98 (0)	303.5	-2.34	0.009^{1}	-0.37
Intentions	0.89 (0.093)	0.90 (0.20)	0.96 (0.095)	1 (0.10)	293	-2.25	0.012^{1}	-0.36
Belief	0.89 (0.104)	0.90 (0.20)	0.95 (0.099)	1 (0.10)	278	-1.98	0.024^{1}	-0.31
Empathy	0.89 (0.097)	0.90 (0.20)	0.96 (0.058)	1 (0.10)	289	-2.25	0.012^{1}	-0.36
Cognitive functions								
Digit span forward	9.53 (2.44)	10 (2.50)	9.45 (2.68)	10 (4.00)	185	1.27	0.898	0.2
Digit span backward	7.37 (2.45)	8 (3.00)	8.45 (2.11)	8 (2.25)	244	-1.14	0.127	-0.18
NET-PANDA	20.10 (2.86)	21 (4.00)	21.30 (2.60)	23 (3.00)	255	-1.55	0.061	-0.24
VF sum score*	16.90 (4.22)	17.10 (5.75)	21.00 (4.35)	21 (4.00)				
Clinical assessment and quality of life								
HDRS	4.32 (5.44)	2 (7.00)	2.5 (2.76)	2 (2.25)	179	0.72	0.763	0.72
Sleepiness (ESS)	6.89 (4.46)	5 (5.50)	7.15 (4.53)	6 (6.25)	200	0.8	0.788	0.13
Fatigue (FSS)	31.7 (12.4)	29 (15.00)	21.9 (5.63)	22.50 (5.50)	91.5	-2.53	0.006	-0.39
PDQ-39	28.8 (19.3)	23 (32.50)	8.4 (9.02)	7 (13.25)	61	-3.44	< 0.001	-0.55
Tsui score	4.95 (2.74)	4 (4.00)	—	_				

Note. Standard deviations (SD) and interquartile range (IQR) are presented in parentheses. RMET = Reading the Mind in the Eyes Task; HDRS = Hamilton depression rating scale; PDQ-39 = Parkinson's Disease Questionnaire (quality of life); W = test statistics of the Wilcoxon test; z = z-value of the Wilcoxon test; r = effect size. ¹p values are FDR-corrected. *See ANOVA results in Results.

rule inference as a specific aspect of executive dysfunction [54]. However, other studies did describe CD-related deficits of affective ToM. For example, low performances were observed in emotion attribution tasks, requiring judgements on the affective color of perceived words or the type of emotion felt by characters in short stories [8, 53, 55]. Further, persons with CD showed impairments in verbally labelling facial or prosodic expressions or matching mimics with the emotional tone of perceived speech [8, 53, 55]. Interestingly, these tasks imply higher lexical or syntactic demands compared to the RMET, which could be a relevant difference [4]. For example, as in the current investigation, impaired performance in the text-based empathy dimension of the FPRT together with unaffected RMET performance was recently identified in persons with chronic inflammatory demyelinating polyneuropathy, altogether showing a subtly dysexecutive cognitive profile [56]. Thus, the above mentioned findings could reflect executive deficits exacerbating under complex biolinguistic demands, shown to prevail in CD [4], rather than impairments of processes genuinely related to empathy functions. Having said that, it is, of course, also possible that heterogeneous results with respect to cognitive and affective ToM in CD represent different cognitive profiles within the spectrum of primary dystonia, as, for example, suggested by the report of higher cognitive ToM impairment in tremulous than nontremulous persons with CD [6].

Concerning pathophysiological aspects of low cognitive ToM and VF performances, result patterns similar to the

current findings were identified in other movement disorders with prominent basal ganglia pathophysiology. For instance, in persons with Parkinson's disease, associations between ToM and EF deficits were reported [57]. In this regard, aberrant striatal input to prefrontal [58, 59] and frontal-opercular cortical areas could impact on social cognitive and word production capacities [60], respectively. Similar underpinnings of VF and cognitive ToM deficits are conceivable in CD. Further, it is of interest that the basal ganglia are generally seen as a pacemaking system for the procedural steps, which different, among others, languagerelated behaviors are composed of [61-64]. Remarkably, the meta-analytical largest effect for cognitive alterations in CD was identified with respect to the reduction of processing speed [4], which could be particularly relevant for the decelerated word production in VF tasks.

Finally, the identified result pattern does not support assumptions derived from EC theories. According to this view, in which the motor system supports affective ToM functions by simulatory processing of observed gestural, mimical, or postural movement patterns, CD as a primary neurological movement disorder should have affected the RMET performance in persons with CD. This, however, could not be demonstrated.

Of course, this study has several limitations. For example, the reductions of cognitive ToM and VF in CD were small, and it can reasonably be asked whether larger cohorts and the inclusion of persons with a higher severity of CD symptoms would have delineated possibly subtle effects also

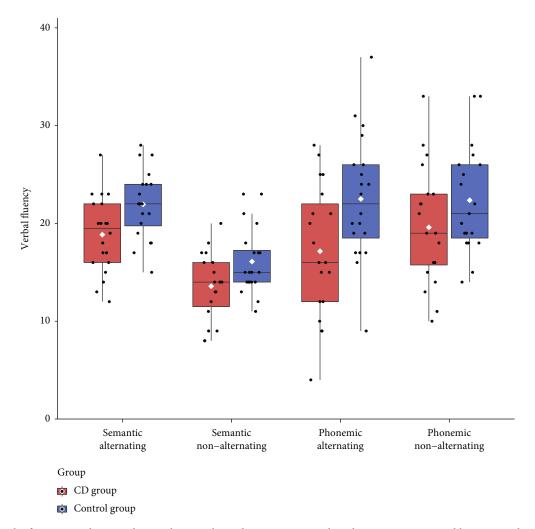


FIGURE 1: VF task of persons with CD and controls per task condition. *Note*. Box plots show mean, upper and lower quartiles, and the range of performances per VF task and condition. Dots represent word production per participant.

in the domain of affective ToM. Further, since we did not assess task engagement, this could theoretically have differed between the groups and, thus, possibly have influenced the results. Accordingly, further investigations should control for this potential confounding factor, among others, because of group differences in fatigue scores (which, however, did not correlate with the cognitive abnormalities identified). Finally, despite an association of the FPRT and VF results with quality of life scores in persons with CD, the small effect sizes and the used tests raise the question which meaning the current findings have for social interactions in natural scenarios. In this regard, future longitudinal studies could address the ecological validity of social cognitive test results, e.g., by assessing features of the relationships between persons with CD and their attachment figures, or their potential changes in the course of disease and its treatment.

5. Conclusion

In conclusion, persons with CD performed worse than controls in cognitive, but not affective ToM tasks. Accompanying reductions of VF suggest this to be embedded in a broader context of executive dysfunction [49]. An association with the used quality of life measure calls for clinical awareness and future determination of the real-life relevance of lowered cognitive ToM in CD.

Data Availability

The data that support the findings of this study are available from the corresponding author upon request.

Ethical Approval

All participants gave written informed consent to the study protocol approved by the ethics committee of the Charité (EA4/165/17).

Disclosure

The research was performed as part of the employment of the authors at Charité – Universitätsmedizin Berlin (Department of Neurology, Motor and Cognition Group).

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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References

- Epidemiological Study of Dystonia in Europe Collaborative, G, "A prevalence study of primary dystonia in eight European countries," *Journal of Neurology*, vol. 247, no. 10, pp. 787– 792, 2000.
- [2] D. T. Corp, J. Joutsa, R. R. Darby et al., "Network localization of cervical dystonia based on causal brain lesions," *Brain*, vol. 142, no. 6, pp. 1660–1674, 2019.
- [3] D. J. Kuyper, V. Parra, S. Aerts, M. S. Okun, and B. M. Kluger, "Nonmotor manifestations of dystonia: a systematic review," *Movement Disorders*, vol. 26, no. 7, pp. 1206–1217, 2011.
- [4] S. L. Aita, V. A. Del Bene, D. A. Marotta et al., "Neuropsychological functioning in primary dystonia: updated and expanded multidomain meta-analysis," *Movement Disorders*, vol. 37, no. 7, pp. 1483–1494, 2022.
- [5] L. Niccolai, S. L. Aita, H. C. Walker et al., "An examination of the neurocognitive profile and base rate of performance impairment in primary dystonia," *Journal of Clinical Neuroscience*, vol. 74, pp. 1–5, 2020.
- [6] K. Czekóová, P. Zemánková, D. J. Shaw, and M. Bareš, "Social cognition and idiopathic isolated cervical dystonia," *Journal of Neural Transmission*, vol. 124, no. 9, pp. 1097–1104, 2017.
- [7] B. Ellement, Y. Jasaui, K. Kathol et al., "Social cognition in cervical dystonia: phenotype and relationship to anxiety and depression," *European Journal of Neurology*, vol. 28, no. 1, pp. 98–107, 2021.
- [8] G. Lagravinese, G. Santangelo, G. Bonassi et al., "Affective and cognitive theory of mind in patients with cervical dystonia with and without tremor," *Journal of Neural Transmission*, vol. 128, no. 2, pp. 199–206, 2021.
- [9] J. Call and M. Tomasello, "Does the chimpanzee have a theory of mind? 30 years later," *Trends in Cognitive Sciences*, vol. 12, no. 5, pp. 187–192, 2008.
- [10] S. G. Shamay-Tsoory and J. Aharon-Peretz, "Dissociable prefrontal networks for cognitive and affective theory of mind: a lesion study," *Neuropsychologia*, vol. 45, no. 13, pp. 3054– 3067, 2007.
- [11] S. G. Shamay-Tsoory, S. Shur, L. Barcai-Goodman, S. Medlovich, H. Harari, and Y. Levkovitz, "Dissociation of cognitive from affective components of theory of mind in schizophrenia," *Psychiatry Research*, vol. 149, no. 1-3, pp. 11–23, 2007.
- [12] P. Thoma, N. Winter, G. Juckel, and P. Roser, "Mental state decoding and mental state reasoning in recently detoxified alcohol-dependent individuals," *Psychiatry Research*, vol. 205, no. 3, pp. 232–240, 2013.
- [13] F. Campanella, T. West, C. Corradi-Dell'Acqua, and M. Skrap, "Cognitive and affective theory of mind double dissociation after parietal and temporal lobe tumours," *Brain*, vol. 145, no. 5, pp. 1818–1829, 2022.

- [14] L. Schlaffke, S. Lissek, M. Lenz et al., "Shared and nonshared neural networks of cognitive and affective theory-of-mind: a neuroimaging study using cartoon picture stories," *Human Brain Mapping*, vol. 36, no. 1, pp. 29–39, 2015.
- [15] F. Van Overwalle, S. De Coninck, E. Heleven et al., "The role of the cerebellum in reconstructing social action sequences: a pilot study," *Social Cognitive and Affective Neuroscience*, vol. 14, no. 5, pp. 549–558, 2019.
- [16] S. Gallagher, "Understanding interpersonal problems in autism: interaction theory as an alternative to theory of mind," *Philosophy, Psychiatry, & Psychology*, vol. 11, no. 3, pp. 199– 217, 2004.
- [17] S. Stuart, "Alvin I. Goldman, simulating minds: the philosophy, psychology and neuroscience of mindreading," *Minds and Machines*, vol. 19, no. 2, pp. 279–282, 2009.
- [18] A. Weber, "Embodied cognition," in *Die körperliche Konstitution von Kognition*, pp. 57–119, J.B. Metzler, Wiesbaden, 2017.
- [19] S. Rafee, R. Monaghan, D. McCormack et al., "Social cognition deficits are associated with lower quality of life in cervical dystonia: a single centre study," *Clinical Parkinsonism & Related Disorders*, vol. 9, article 100214, 2023.
- [20] I. Ndukwe, S. O'Riordan, C. B. Walsh, and M. Hutchinson, "Trust the patient not the doctor: the determinants of quality of life in cervical dystonia," *Frontiers in Neurology*, vol. 11, p. 991, 2020.
- [21] A. d'Arma, A. Valle, D. Massaro et al., "A cultural training for the improvement of cognitive and affective theory of mind in people with multiple sclerosis: a pilot randomized controlled study," *Frontiers in Psychology*, vol. 14, 2023.
- [22] W. H. Jost, H. Hefter, A. Stenner, and G. Reichel, "Rating scales for cervical dystonia: a critical evaluation of tools for outcome assessment of botulinum toxin therapy," *Journal of Neural Transmission*, vol. 120, no. 3, pp. 487–496, 2013.
- [23] J. K. Tsui, A. Eisen, A. J. Stoessl, S. Calne, and D. B. Calne, "Double-blind study of botulinum toxin in spasmodic torticollis," *The Lancet*, vol. 328, no. 8501, pp. 245–247, 1986.
- [24] C. Jenkinson, V. Peto, R. Fitzpatrick, R. Greenhall, and N. Hyman, "Self-reported functioning and well-being in patients with Parkinson's disease: comparison of the shortform health survey (SF-36) and the Parkinson's disease questionnaire (PDQ-39)," Age and Ageing, vol. 24, no. 6, pp. 505– 509, 1995.
- [25] L. B. Krupp, N. G. LaRocca, J. Muir-Nash, and A. D. Steinberg, "The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus," *Archives of Neurology*, vol. 46, no. 10, pp. 1121–1123, 1989.
- [26] M. Hamilton, "A rating scale for depression," *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 23, no. 1, pp. 56–62, 1960.
- [27] M. W. Johns, "A new method for measuring daytime sleepiness: the Epworth sleepiness scale," *Sleep*, vol. 14, no. 6, pp. 540–545, 1991.
- [28] S. Baron-Cohen, S. Wheelwright, J. Hill, Y. Raste, and I. Plumb, "The "Reading the mind in the eyes" test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism," *Journal of Child Psychology and Psychiatry and Allied Disciplines*, vol. 42, no. 2, pp. 241–251, 2001.
- [29] V. E. Stone, S. Baron-Cohen, and R. T. Knight, "Frontal lobe contributions to theory of mind," *Journal of Cognitive Neuroscience*, vol. 10, no. 5, pp. 640–656, 1998.

- [30] J. D. Henry, D. G. Cowan, T. Lee, and P. S. Sachdev, "Recent trends in testing social cognition," *Current Opinion in Psychiatry*, vol. 28, no. 2, pp. 133–140, 2015.
- [31] S. Aschenbrenner, O. Tucha, and K. W. Lange, *Regensburger Wortflüssigkeits-Test: RWT*, Hogrefe, Verlag für Psychologie, 2000.
- [32] A. Owen, A. Lee, and E. Williams, "Dissociating aspects of verbal working memory within the human frontal lobe: further evidence for a 'process-specific' model of lateral frontal organization," *Psychobiology*, vol. 28, no. 2, pp. 146–155, 2000.
- [33] Z. S. Nasreddine, N. A. Phillips, V. Bedirian et al., "The Montreal cognitive assessment, MoCA: a brief screening tool for mild cognitive impairment," *Journal of the American Geriatrics Society*, vol. 53, no. 4, pp. 695–699, 2005.
- [34] J. F. Scheffels, L. Frohlich, E. Kalbe, and J. Kessler, "Concordance of mini-mental state examination, Montreal cognitive assessment and Parkinson neuropsychometric dementia assessment in the classification of cognitive performance in Parkinson's disease," *Journal of the Neurological Sciences*, vol. 412, 2020.
- [35] M. F. Folstein, S. E. Folstein, and P. R. McHugh, ""Mini-mental state": a practical method for grading the cognitive state of patients for the clinician," *Journal of Psychiatric Research*, vol. 12, no. 3, pp. 189–198, 1975.
- [36] E. Kalbe, P. Calabrese, N. Kohn et al., "Screening for cognitive deficits in Parkinson's disease with the Parkinson neuropsychometric dementia assessment (PANDA) instrument," *Parkinsonism & Related Disorders*, vol. 14, no. 2, pp. 93–101, 2008.
- [37] F. Ehlen, I. Vonberg, H. O. Tiedt et al., "Thalamic deep brain stimulation decelerates automatic lexical activation," *Brain* and Cognition, vol. 111, pp. 34–43, 2017.
- [38] F. Klostermann, M. Wyrobnik, M. Boll, F. Ehlen, and H. O. Tiedt, "Tracing embodied word production in persons with Parkinson's disease in distinct motor conditions," *Scientific Reports*, vol. 12, no. 1, p. 16669, 2022.
- [39] C. J. Ferguson, "An effect size primer: a guide for clinicians and researchers," *Professional Psychology-Research and Practice*, vol. 40, no. 5, pp. 532–538, 2009.
- [40] H. Hefter, S. Samadzazeh, and D. Rosenthal, "The impact of the initial severity on later outcome: retrospective analysis of a large cohort of botulinum toxin naïve patients with idiopathic cervical dystonia," *Journal of Neurology*, vol. 268, no. 1, pp. 206–213, 2021.
- [41] H. Hefter, I. Schomaecker, M. Schomaecker et al., "Lessons about botulinum toxin a therapy from cervical dystonia patients drawing the course of disease: a pilot study," *Toxins*, vol. 15, no. 7, p. 431, 2023.
- [42] J. Cohen, "A power primer," *Psychological Bulletin*, vol. 112, no. 1, pp. 155–159, 1992.
- [43] F. Faul, E. Erdfelder, A. G. Lang, and A. Buchner, "G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences," *Behavior Research Methods*, vol. 39, no. 2, pp. 175–191, 2007.
- [44] T. Aboulafia-Brakha, B. Christe, M. D. Martory, and J. M. Annoni, "Theory of mind tasks and executive functions: a systematic review of group studies in neurology," *Journal of Neuropsychology*, vol. 5, no. 1, pp. 39–55, 2011.
- [45] M. Bertoux, C. O'Callaghan, B. Dubois, and M. Hornberger, "In two minds: executive functioning versus theory of mind

in behavioural variant frontotemporal dementia," *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 87, no. 3, pp. 231–234, 2016.

- [46] N. Moreau and M. Champagne-Lavau, "Théorie de l'esprit et fonctions exécutives dans la pathologie," *Revue de Neuropsychologie*, vol. 6, no. 4, pp. 276–281, 2015.
- [47] A. W. Qureshi, R. L. Monk, D. Samson, and I. A. Apperly, "Does interference between self and other perspectives in theory of mind tasks reflect a common underlying process? Evidence from individual differences in theory of mind and inhibitory control," *Psychonomic Bulletin & Review*, vol. 27, no. 1, pp. 178–190, 2020.
- [48] F. Schmid, F. Moreau, F. Benzerouk et al., "Faux pas recognition and executive processes in patients with alcohol use disorder: toward an investigation of interindividual heterogeneity," *Archives of Clinical Neuropsychology*, vol. 37, no. 3, pp. 608– 620, 2022.
- [49] M. Wade, H. Prime, J. M. Jenkins, K. O. Yeates, T. Williams, and K. Lee, "On the relation between theory of mind and executive functioning: a developmental cognitive neuroscience perspective," *Psychonomic Bulletin & Review*, vol. 25, no. 6, pp. 2119–2140, 2018.
- [50] J. Amunts, J. A. Camilleri, S. B. Eickhoff, S. Heim, and S. Weis, "Executive functions predict verbal fluency scores in healthy participants," *Scientific Reports*, vol. 10, no. 1, article 11141, 2020.
- [51] S. Bottiroli, E. Cavallini, I. Ceccato, T. Vecchi, and S. Lecce, "Theory of mind in aging: comparing cognitive and affective components in the faux pas test," *Archives of Gerontology* and Geriatrics, vol. 62, pp. 152–162, 2016.
- [52] T. Burke, R. Monaghan, D. McCormack et al., "Social cognition in cervical dystonia: a case-control study," *Clinical Parkinsonism & Related Disorders*, vol. 3, article 100072, 2020.
- [53] R. Monaghan, C. Cogley, T. Burke et al., "Non-motor features of cervical dystonia: cognition, social cognition, psychological distress and quality of life," *Clinical Parkinsonism & Related Disorders*, vol. 4, p. 100084, 2021.
- [54] F. Lange, C. Seer, C. Salchow, R. Dengler, D. Dressler, and B. Kopp, "Meta-analytical and electrophysiological evidence for executive dysfunction in primary dystonia," *Cortex*, vol. 82, pp. 133–146, 2016.
- [55] Z. T. Nikolova, A. Fellbrich, J. Born, R. Dengler, and C. Schroder, "Deficient recognition of emotional prosody in primary focal dystonia," *European Journal of Neurology*, vol. 18, no. 2, pp. 329–336, 2011.
- [56] H. Lochmann, M. Wyrobnik, C. Kupper, C. Rewitzer, and F. Klostermann, "Theory of mind and executive dysfunction in chronic inflammatory demyelinating polyneuropathy," *European Journal of Neurology*, vol. 14, article 1208638, 2023.
- [57] G. Maggi, D. Di Meglio, C. Vitale, M. Amboni, I. Obeso, and G. Santangelo, "The impact of executive dysfunctions on theory of mind abilities in Parkinson's disease," *Neuropsychologia*, vol. 176, article 108389, 2022.
- [58] C. Corradi-Dell'Acqua, R. Ronchi, M. Thomasson, T. Bernati, A. Saj, and P. Vuilleumier, "Deficits in cognitive and affective theory of mind relate to dissociated lesion patterns in prefrontal and insular cortex," *Cortex*, vol. 128, pp. 218–233, 2020.
- [59] C. Trompeta, B. Fernandez Rodriguez, and C. Gasca-Salas, "What do we know about theory of mind impairment in Parkinson's disease?," *Behavioral Sciences*, vol. 11, no. 10, 2021.

- [60] J. E. Castner, H. J. Chenery, P. A. Silburn et al., "Effects of subthalamic deep brain stimulation on noun/verb generation and selection from competing alternatives in Parkinson's disease," *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 79, no. 6, pp. 700–705, 2007.
- [61] D. Aarsland and M. W. Kurz, "The epidemiology of dementia associated with Parkinson's disease," *Brain Pathology*, vol. 20, no. 3, pp. 633–639, 2010.
- [62] D. Plenz and S. T. Kital, "A basal ganglia pacemaker formed by the subthalamic nucleus and external globus pallidus," *Nature*, vol. 400, no. 6745, pp. 677–682, 1999.
- [63] H. O. Tiedt, F. Ehlen, and F. Klostermann, "Increased conceptual switching by dopaminergic treatment in patients with Parkinson's disease," *Brain and Cognition*, vol. 144, 2020.
- [64] I. Vonberg, F. Ehlen, O. Fromm, A. A. Kuhn, and F. Klostermann, "Deep brain stimulation of the subthalamic nucleus improves lexical switching in Parkinsons disease patients," *PLoS One*, vol. 11, no. 8, article e0161404, 2016.