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Modeling effects of cerebellar and basal ganglia lesions on adaptation and anticipation during sensorimotor synchronization

M.C. (Marieke) van der Steen,[1] Michael Schwartze,[1,2] Sonja A. Kotz,[1,2] and Peter E. Keller[1,3]

[1]Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany. [2]School of Psychological Sciences, University of Manchester, Manchester, England, United Kingdom. [3]The MARCS Institute, University of Western Sydney, Sydney, New South Wales, Australia

Address for correspondence: M.C. (Marieke) van der Steen, Max Planck Institute for Human Cognitive and Brain Sciences, Stephanstraße 1A, Leipzig, Saxony 04103, Germany. steen@cbs.mpg.de

This study addressed the role of subcortical brain structures in temporal adaptation and anticipation during sensorimotor synchronization. The performance of patients with cerebellar or basal ganglia lesions was compared with that of healthy control participants on tasks requiring the synchronization of drum strokes with adaptive and tempo-changing auditory pacing sequences. The precision of sensorimotor synchronization was generally lower in patients relative to controls (i.e., variability of asynchronies was higher in patients), although synchronization accuracy (mean asynchrony) was commensurate. A computational model of adaptation and anticipation (ADAM) was used to examine potential sources of individual differences in precision by estimating participants' use of error correction, temporal prediction, and the amount of variability associated with central timekeeping and peripheral motor processes. Parameter estimates based on ADAM indicate that impaired precision was attributable to increased variability of timekeeper and motor processes as well as to reduced temporal prediction in both patient groups. Adaptive processes related to continuously applied error correction were, by contrast, intact in patients. These findings highlight the importance of investigating how subcortical structures, including the cerebellum and basal ganglia, interact with a broader network of cortical regions to support temporal adaptation and anticipation during sensorimotor synchronization.

Keywords: sensorimotor synchronization; timing; patients; computational modeling; prediction; error correction

Introduction

Daily life necessitates the coordination of actions with events in the environment. In the context of rhythmic behaviors (e.g., dancing or playing in a musical ensemble), this involves the temporal coordination of movements with events in a predictable external rhythm.[1] Although such sensorimotor synchronization is a fundamental human skill, it is subject to large individual differences. Sensorimotor synchronization can be highly precise and flexible in trained individuals (e.g., musicians)[2,3] and severely impaired in patients with disorders affecting rhythm perception and production (e.g., stroke and Parkinson's disease).[4,5] Such impairments can compromise everyday functioning and negatively impact the quality of life.

Recent research has sought to identify the sources of individual differences in sensorimotor synchronization skill by combining behavioral experimentation with computational modeling.[6–8] Behavioral experiments on sensorimotor synchronization typically employ laboratory tasks that require repetitive movements (e.g., finger taps or drum strokes) to be produced in time with sequences of auditory or visual events generated by a computer.[1,9] The results of such experiments suggest that sensorimotor synchronization relies on adaptive processes that enable the individual to react to deviations

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in event timing[10,11] and anticipatory processes that allow an individual to predict upcoming event timing.[12] Computational modeling has shed light on the mechanisms that underlie these adaptive and anticipatory processes.[13–18]

Temporal adaptation

Temporal adaptation involves adjusting the timing of movements in order to maintain synchrony in the face of irregularities that arise because of variations in the timing of the external pacing sequence and biological "noise" in the individual's nervous system. It has been proposed that these adjustments are mediated by error correction mechanisms that allow internal timekeepers (oscillations of neural populations) in the individual's brain to remain entrained (i.e., coupled) to the external sequence under such variable conditions.[19–22] One class of mechanism, phase correction, is an automatic process that adjusts the way in which the pulses generated by an internal timekeeper are aligned against events in the external sequence. Two types of phase correction have been identified, one triggered by abrupt timing perturbations and the other elicited in the context of regularly timed sequences and sequences containing continuous modulations of event timing.[14] The other mechanism, period correction, involves consciously controlled adjustments to the duration of timekeeper intervals and is invoked when the individual intentionally adapts to attended tempo changes in the sequence.[10]

Models of error correction assume that a proportion of each asynchrony between a tap and pacing tone is compensated for on a subsequent movement.[13,16] The effects of continuously applied (as opposed to abrupt) error correction on sensorimotor synchronization have been studied with adaptive pacing sequences that are programmed to implement varying degrees of phase and period correction.[21,23,24] This work has shown that moderate amounts of phase correction implemented by the adaptive pacing sequence (accounting for 0.25–0.50 of each asynchrony) increase the precision of sensorimotor synchronization by reducing the variability of asynchronies, whereas amounts of phase correction outside this range lead to poorer synchronization. Furthermore, synchronization with tempo-changing sequences is most precise with small amounts of period correction (0.5 or less) because larger amounts potentially lead to tempo drift.[7,24]

Temporal anticipation

Anticipatory mechanisms facilitate sensorimotor synchronization by allowing individuals to plan the timing of their movements with reference to predictions about the future time course of the pacing sequence. These predictions may evolve along two routes, one relying on automatic expectancies triggered by perceptual input and the other on effortful processes involving attention, working memory, and mental imagery.[25,26] Anticipation during sensorimotor synchronization has been studied using tasks that require an individual to tap along with pacing sequences that contain gradual tempo changes resembling those found in expressive music performance.[3,12,27,28] In these studies, the degree to which individuals predicted tempo changes was estimated based on the assumption that prediction is high to the extent that intertap intervals match (rather than lag behind) pacing sequence interonset intervals. It was found that the tendency to predict tempo changes was positively correlated with musical experience and that these individual differences were related to the accuracy and precision of synchronization with computer-controlled pacing sequences and sounds produced by another individual during dyadic finger tapping.[3]

Modeling and parameter estimation

The roles of temporal adaptation and anticipation in sensorimotor synchronization have traditionally been investigated separately. However, it has recently been proposed that a fuller understanding of individual differences in sensorimotor synchronization skill can be achieved by examining the way in which adaptation and anticipation mechanisms interact.[17] To this end, a combined adaptation and anticipation model (ADAM) has been developed in which (1) an adaptation module implements phase and/or period correction; (2) an anticipation module generates temporal predictions based on linear extrapolation of tempo changes in the pacing sequence; and (3) a joint module compares the output of the adaptation and anticipation modules and compensates for any discrepancy by implementing anticipatory error correction.[7] The model also includes two sources of noise, one that represents variability in a "timekeeper" in the central nervous system and the

other representing "motor" noise in the peripheral nervous system.[16,18]

Each process instantiated in ADAM is controlled by an independent parameter, and the value of these parameters can be estimated for a particular individual by fitting the model to asynchrony data from sensorimotor synchronization tasks.[14] ADAM was tested in a recent study that combined behavioral experimentation, computer simulations, and parameter estimation.[7] The results suggested that although adaptation mechanisms alone can account for synchronization with steady pacing sequences, synchronization with tempo-changing sequences is best accounted for by including anticipation in the model and, moreover, by activating links between adaptation and anticipation in the joint module.

Current aim

This study investigated the neural substrates of temporal adaptation and anticipation during sensorimotor synchronization. Neuroimaging work indicates that subcortico-thalamo-cortical networks subserving both processes overlap to some degree. Subcortical regions implicated in adaptive timing include the cerebellum and basal ganglia, and cortical regions include the premotor cortex, (pre-)supplementary motor area, sensorimotor cortex, superior temporal gyrus, and inferior frontal gyrus.[5,29–33] Temporal prediction has likewise been linked to these areas in addition to regions of the prefrontal cortex.[34–39]

Here we focus on subcortical contributions to temporal adaptation and anticipation by examining synchronization impairments in patients with lesions of the cerebellum and basal ganglia. Several theories addressing timing have proposed a division of labor between these structures.[26,34,40] In this view, the cerebellum is specialized for automatic event-based temporal processing at short timescales (up to tens of milliseconds), whereas the basal ganglia specialize in attention-dependent interval-based processing at longer timescales (from hundreds of milliseconds to seconds). Thus, it is possible that the cerebellum and basal ganglia are differentially implicated in automatic and attention-dependent components of temporal adaptation and anticipation. To test this, we administered sensorimotor synchronization tasks with adaptive and tempo-changing pacing sequences to patients and healthy controls and then assessed individual differences in task performance via parameter estimation using ADAM.

Methods

Participants

Fifteen patients (two female) with focal lesions of the basal ganglia (13 ± 5.32 years postlesion onset; Fig. 1A) and 11 patients (five female) with focal lesions of the cerebellum (4.5 ± 2.88 years postlesion onset; Fig. 1B) participated in the study. Median age was 60 years (range 37–82) for basal ganglia patients and 45 years (range 25–59) for cerebellar patients. The etiology of basal ganglia lesions included middle cerebral artery infarction and intracranial hemorrhage, and for cerebellar lesions etiologies included infarction, hemorrhage, aneurism, and resection. Healthy control individuals who matched each patient in terms of age, education, sex, and handedness also participated in the study. Patients and controls had comparably low amounts of musical training (apart from one basal ganglia patient who had formerly worked as a pianist). The study was approved by the local ethics committee of the University of Leipzig, and all individuals received financial compensation in return for participation.

Procedure

Participants performed two sensorimotor synchronization tasks that involved the use of a drumstick to tap in time with auditory pacing sequences. One task employed adaptive pacing sequences, and the other employed tempo-changing sequences. Participants tapped on the rubber surface of a MIDI percussion pad (Roland Handsonic HPD-10, Roland Corp. US, Los Angeles, CA, USA). Stimulus presentation and response registration were controlled by MaxMSP software running on a Windows PC. Stimulus sounds ("tones") were woodblock samples generated by a Roland SPD-S Sampling Pad and presented over headphones. Participants' taps did not trigger feedback sounds other than the drumstick impact on the rubber pad. Participants performed the tasks in a fixed order, starting with the adaptive task followed by the tempo-changing task. The tasks were performed first with the dominant hand followed by the nondominant hand (in all but three basal ganglia patients, who were unable to complete the task with the nondominant hand), with a rest break in between.

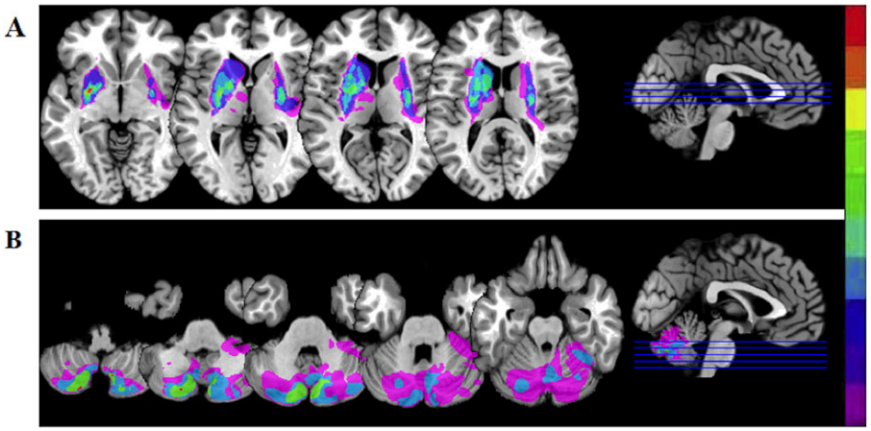

Figure 1. Structural overlay of basal ganglia lesions (A) and cerebellum lesions (B). Basal ganglia scale from 1 to 7 (locations of maximum overlap); slices *z* 63, 69, 75, 81. Cerebellum scale from 1 to 4, slices: 20, 26, 32, 38, 44. Purple shades are associated with minimal lesion overlap, and red shades reflect maximum lesion overlap.

Adaptive task

The adaptive task involved synchronizing with pacing sequences in which event timing was controlled by an algorithm that adjusted sequence interonset intervals in order to compensate for asynchronies between the participant's taps and pacing tones.[8,17,21] The amount of adaptation was governed by a phase correction parameter (αc), which represents the proportion of each asynchrony that is corrected for by the algorithm. Specifically, the onset time of the next tone was determined by calculating the most recent tap-tone asynchrony, multiplying it by αc, and adding the result to the timekeeper period to obtain the current tone interonset interval.

Four levels of adaptation were employed: $\alpha c = 0$, 0.3, 0.5, and 0.7 (with 0 representing an isochronous metronome and 0.7 representing an overly high amount of phase correction). Each of these adaptation conditions was presented in a separate block of 15 trials, with block order randomized. The pacing sequence in each trial had a base interonset interval of 600 ms and consisted of 25 tones. Participants were instructed to start tapping from the third tone onward and to synchronize their taps as accurately as possible with the pacing signal while maintaining the initial tempo. Participants completed one practice trial per condition before the experimental blocks.

Tempo-changing task

The pacing sequences in the tempo-changing task consisted of 68 tones. Sequence interonset intervals were set to a constant value of 600 ms for the first five tones and thereafter changed between 600 and 387 ms (following a sigmoidal function) over the course of five to nine intervals, alternating between acceleration and deceleration.[8] The rate and direction of tempo change were thus varied to produce 12 different pacing sequences. The 12 sequences were presented once in random order. Two randomly chosen sequences served as practice trials. Participants were instructed to start tapping with the third tone and to tap as accurately as possible in synchrony with the sequences.

Data analysis

In one set of analyses, the accuracy and precision of sensorimotor synchronization were assessed in both tasks by computing asynchronies between participants' taps and pacing tones (in such a way that negative values indicate that taps preceded tones). A small number (1.5%) of taps that were missed or fell outside a $\pm$200-ms asynchrony window were excluded from analysis. The mean asynchrony was calculated for each trial as an inverse measure of synchronization accuracy, and the standard deviation (SD) of asynchronies served as an inverse measure of precision. For data from the tempo-changing task, the degree to which participants predicted versus tracked (i.e., lagged behind) tempo changes was estimated by calculating the prediction/tracking ratio (PT-ratio).[3] The PT-ratio is computed by dividing the lag 0 by the lag 1 cross-correlation between intertap and interstimulus intervals. A value greater

than 1 indicates a stronger tendency to predict than to track tempo changes, whereas a value smaller than 1 indicates a stronger tendency to track than to predict. Measures of synchronization accuracy, precision, and prediction were entered into analyses of variance (ANOVAs) with group (patient or control) and region of interest (ROI: basal ganglia or cerebellum) as between-subject factors. For the adaptive task, the ANOVAs also included level of adaptation ($\alpha c = 0, 0.3, 0.5, 0.7$) as a within-subjects factor. The Greenhouse–Geisser correction was applied when the assumption of sphericity was violated.

In a second set of analyses, parameter estimates based on ADAM were obtained for data from the adaptation task and the tempo-changing task. For the adaptation task, the adaptation module of ADAM was fitted to the data from patients and controls. For the tempo-changing task, the full model (with the adaptation module, the anticipation module, and the joint module) was fitted to the data. Parameter estimates were computed using the bounded generalized least-squares method,[13,14] which solves standard generalized least-square regression problems by applying matrix algebra with empirically justified constraints.[41] Parameter estimates for the two tasks were analyzed in multivariate analyses of variance (MANOVAs) with group (patient or control) and ROI (basal ganglia or cerebellum) as between-subject factors. Finally, multiple regression analyses were conducted to find the combinations of model parameters that best predicted the precision of sensorimotor synchronization in the adaptation and tempo-changing tasks.

Results

Adaptive task

Measures of synchronization accuracy (mean asynchrony) and precision (SD of asynchronies) for the adaptive task, averaged across participants in each patient and control group, are shown in Figure 2A and B, respectively. The ANOVA on mean asynchrony data revealed no statistically significant main effects of group (patient versus control), ROI (basal ganglia versus cerebellum), or level of adaptation ($\alpha c = 0, 0.3, 0.5, 0.7$), and no significant interactions among these factors (all $P > 0.05$). The ANOVA on SD of asynchronies, by contrast, revealed significant main effects of group ($F_{(1,48)} = 9.58$, $P = 0.003$) and level of adaptation ($F_{(3,144)} = 11.34$, $P < 0.001$), but the ROI main effect and interaction effects were not significant. The main effect of group reflects less precise (more variable) sensorimotor synchronization in patients than healthy control participants. The effect of level of adaptation reflects a quadratic trend ($F_{(1,48)} = 34.78$, $P < 0.001$), indicating that synchronization was generally more precise at moderate than extreme levels of adaptation.

Estimated values for the phase correction (α), period correction (β), timekeeper noise, and motor noise parameters from the adaptation module of ADAM are shown in Table 1. The MANOVA on these parameter estimates yielded main effects of group (patient versus control) for timekeeper noise ($F_{(1,48)} = 7.44$, $P = 0.009$) and motor noise ($F_{(1,48)} = 8.47$, $P = 0.005$), reflecting the fact that both noise parameter values were higher in patients than controls. Parameter estimates were similar across ROI subgroups within each group, leading to nonsignificant ROI and group × ROI interaction effects (all $P > 0.05$). To test the relationship between model parameters and synchronization precision, we ran a multiple regression analysis that included all parameter estimates (and participant classifications as basal ganglia patient, cerebellar patient, or control) as predictors of SD of asynchronies. This analysis yielded a model with $R^2 = 0.98$, with timekeeper noise ($\beta = 0.77$, $P < 0.001$) and motor noise ($\beta = 0.23$, $P < 0.001$) emerging as the only significant unique predictors of synchronization precision.

Tempo-changing task

Measures of synchronization accuracy (mean asynchrony), precision (SD of asynchronies), and temporal prediction (PT-ratios) for the tempo-changing task, averaged across participants in each patient and control group, are shown in Figure 3A–C. The ANOVA on mean asynchrony data revealed no significant main effects of group, ROI, or level of adaptation and no significant interactions among these factors (all $P > 0.05$). The ANOVA on SD of asynchronies, however, yielded a significant main effect of group ($F_{(1,48)} = 14.50$, $P < 0.001$), indicating that patients were again less precise than control participants. No other effects were significant in this ANOVA. Similarly, there were no significant effects in the ANOVA on PT-ratios (all $P > 0.05$), which one-tailed *t*-tests revealed were significantly greater than 1 (indicating stronger prediction of tempo changes than tracking) for all participant groups (all $P < 0.05$).

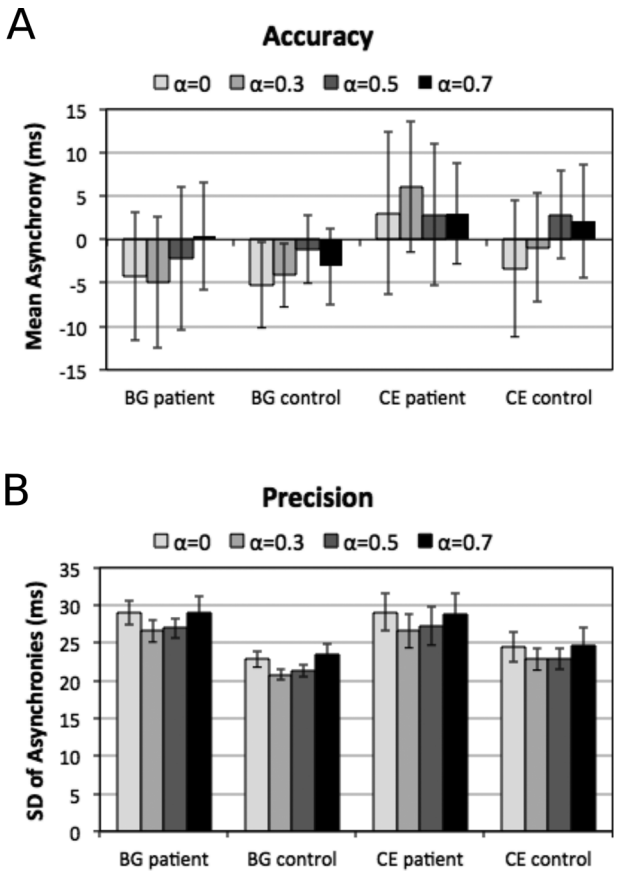

Figure 2. **Measures of sensorimotor synchronization in the adaptive task for basal ganglia (BG) and cerebellar (CE) patients and matched healthy controls: (A) synchronization accuracy indexed by mean asynchrony; and (B) synchronization precision indexed (inversely) by the standard deviation of asynchronies. Error bars indicate standard error of the mean.**

Estimated values for model parameters related to period correction (β, implemented in ADAM's adaptation module), temporal prediction (*m*, implemented in ADAM's anticipation module), anticipatory error correction (γ, implemented in ADAM's joint module), timekeeper noise, and motor noise are shown in Table 1. The MANOVA on these parameter estimates yielded a main effect of group (patient versus control) for temporal prediction ($F_{(1,48)} = 6.09$, $P = 0.017$) and timekeeper noise ($F_{(1,48)} = 9.55$, $P = 0.003$). Estimates of the temporal prediction parameter were lower in patients than controls, whereas timekeeper noise estimates were higher in patients than controls. There was also a significant main effect of ROI ($F_{(1,48)} = 5.73$, $P = 0.021$) on motor noise estimates, which were higher in cerebellar patients than in basal ganglia patients (whereas the corresponding difference for the matched control groups was not significant). No further effects were significant.

A multiple regression analysis conducted to test the relationship between model parameters and synchronization precision yielded a model with $R^2 = 0.98$, with temporal prediction ($\beta = -0.31$, $P < 0.001$), anticipatory error correction ($\beta = 0.16$, $P = 0.001$), and timekeeper noise ($\beta = 0.85$, $P < 0.001$) emerging as the only significant unique predictors of precision.

Discussion

The role of subcortical brain structures in temporal adaptation and anticipation was investigated by testing the abilities of patients with lesions of the cerebellum or basal ganglia on sensorimotor synchronization tasks that required drumming in time with adaptive and tempo-changing auditory pacing

Table 1. Average parameter estimates for the adaptive task (based on ADAM's adaptation module) and the tempo-changing task (based on ADAM with adaptation, anticipation, and joint modules) for basal ganglia and cerebellar patients and matched controls

Group	Adaptive task	Mean	SD	Tempo-changing task	Mean	SD
Basal ganglia patients	α	0.34	0.11	β	0.16	0.09
	β	0.40	0.08	γ	0.28	0.09
	Timekeeper noise	17.99	3.99	*m*	0.33	0.23
	Motor noise	7.98	2.04	Timekeeper noise	58.18	19.83
				Motor noise	2.14	1.71
Cerebellum patients	α	0.36	0.09	β	0.14	0.03
	β	0.40	0.05	γ	0.25	0.09
	Timekeeper noise	17.85	4.61	*m*	0.28	0.17
	Motor noise	8.65	3.54	Timekeeper noise	50.58	16.96
				Motor noise	5.54	4.76
Basal ganglia controls	α	0.34	0.08	β	0.16	0.08
	β	0.41	0.04	γ	0.26	0.06
	Timekeeper noise	14.60	2.00	*m*	0.45	0.13
	Motor noise	6.04	1.63	Timekeeper noise	42.21	9.48
				Motor noise	4.15	3.84
Cerebellum controls	α	0.34	0.11	β	0.14	0.05
	β	0.42	0.08	γ	0.25	0.05
	Timekeeper noise	15.54	3.36	*m*	0.41	0.16
	Motor noise	6.67	2.37	Timekeeper noise	41.23	7.65
				Motor noise	6.28	5.80

α, phase correction parameter; β, period correction; γ, anticipatory error correction; *m*, temporal prediction.

sequences. Patients generally performed as accurately as healthy control participants (in terms of mean asynchrony), but their precision was relatively low (i.e., variability of asynchronies was high). A computational model of adaptation and anticipation, ADAM,[7,17] was employed to uncover potential sources of individual differences in participants' behavior by estimating their use of error correction and temporal prediction as well as the amount of noise associated with central timekeeping and peripheral motor processes.

Model-based parameter estimates for the adaptive task (which required synchronization with sequences that implemented varying degrees of phase correction) indicated that levels of timekeeper and motor noise were high in patients relative to controls, although estimates of phase correction and period correction were commensurate across groups. Deficits in the precision of sensorimotor synchronization in cerebellar and basal ganglia patients were therefore attributable to variability in internal timekeeping processes as well as in motor programming and execution rather than to impairments of sensorimotor coupling mechanisms that underpin continuous temporal adaptation. Further evidence that continuous adaptation was intact in patients comes from the result that they benefited similarly to controls when synchronizing with sequences that employed moderate amounts of error correction.

The finding that parameter estimates did not differ reliably between the two patient groups (although estimates of motor noise were numerically higher in cerebellar than in basal ganglia patients) suggests that the adaptive task was neither attentionally nor motorically demanding. Indeed, synchronization at a steady tempo should not require large-scale updating of temporal predictions or motor programs.

Parameter estimates for the tempo-changing task revealed that the relatively low precision observed in patients when they were required to synchronize with sequences containing gradual accelerations and decelerations was related to impaired temporal

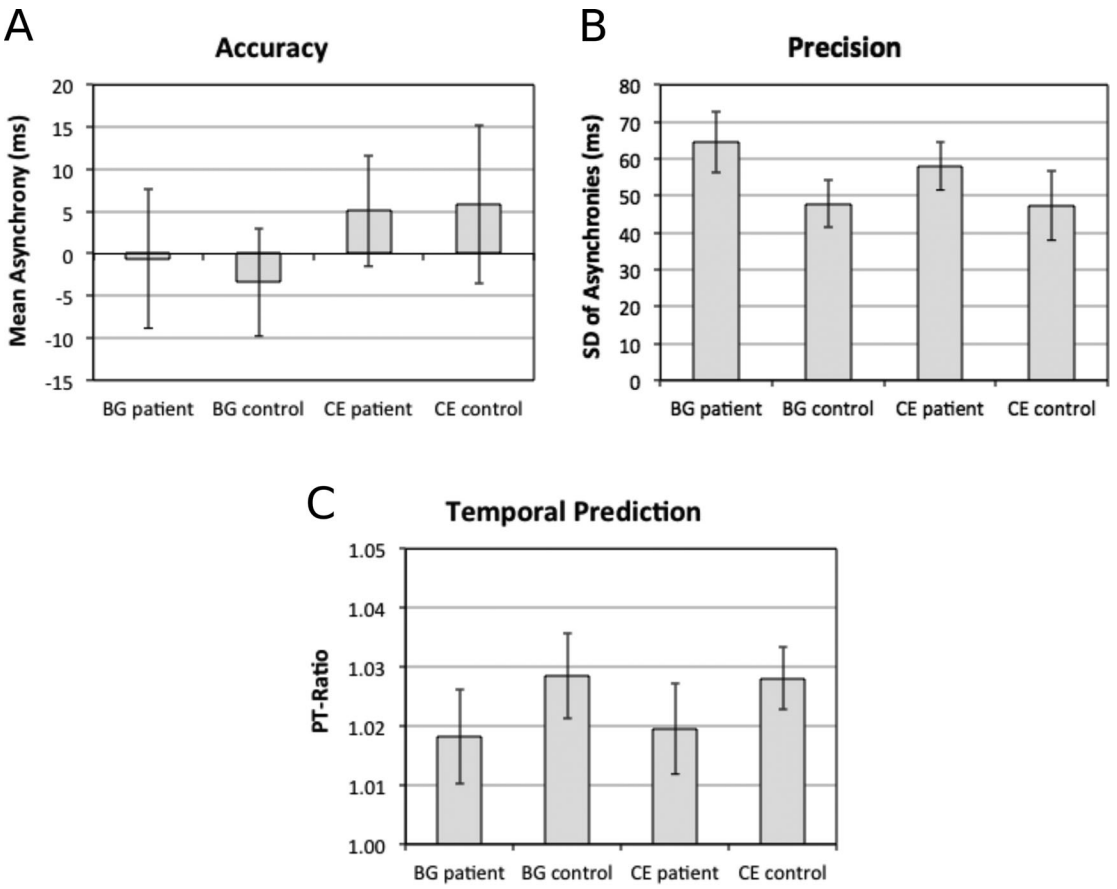

Figure 3. Measures of sensorimotor synchronization in the tempo-changing task for basal ganglia (BG) and cerebellar (CE) patients and matched healthy controls. (A) synchronization accuracy indexed by mean asynchrony; (B) synchronization precision indexed (inversely) by the standard deviation of asynchronies; and (C) temporal prediction indexed by the prediction/tracking ratio (PT-ratio). Error bars indicate standard error of the mean.

prediction and high timekeeper noise. Although our behavioral index of temporal prediction (the PT-ratio, based on cross correlations between intertap and interstimulus intervals) did not differ reliably between patients and controls (although it was numerically lower in patients), model-based parameter estimates of prediction were significantly lower in patients. Model-based estimates of prediction may be more sensitive than behavioral measures because these estimates partial out the effects of other processes, such as period correction and anticipatory error correction, which were apparently spared in patients.

The benefit of supplementing behavioral data with computational modeling is further highlighted by the finding that estimates of anticipatory error correction (based on the joint module in ADAM) accounted for interindividual variation in the precision of sensorimotor synchronization, although this relationship was independent of participant status as patient or control. Evidence for anticipatory error correction is noteworthy, as it supports the proposal that discrepancies between the timing of planned movements and anticipated pacing events can be compensated for in advance[7] via a process of internal modeling.[42,43]

The finding that timekeeper noise estimates were higher in patients than controls in the tempo-changing task is consistent with the results of the adaptive task. Lesions of the cerebellum or basal ganglia thus generally impair the precision of sensorimotor synchronization by increasing the variability of internal timekeeping processes. However, in contrast to the adaptive task, estimates of motor noise

were higher in cerebellar patients than basal ganglia patients for the tempo-changing task. This suggests that cerebellar patients may have been less successful at meeting the heightened demands associated with updating motor programs in order to maintain synchrony with tempo changes. Selective impairment to low-level motor processes is broadly consistent with proposals that the cerebellum is specialized for automatic processes at short timescales.[34,39,40]

In conclusion, this study provides evidence that lesions of the cerebellum and basal ganglia impair precision in sensorimotor synchronization by increasing the variability of timekeeper and motor processes as well as by reducing the ability to generate temporal predictions based on extrapolation from patterns of sequential tempo change. It should be noted, however, that the deficits displayed by cerebellar and basal ganglia patients were not severe enough to prevent accurate synchronization. This underscores the importance of investigating how these subcortical structures interact with a broader network of cortical regions to support both temporal adaptation[23] and anticipation.[37]

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Conflicts of interest

The authors declare no conflicts of interest.

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