Evidence From Parkinson’s Disease That the Superior Colliculus Couples Action and Perception

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ABSTRACT: Background: Action and perception should be coordinated for good visual-motor performance. The mechanism coupling action and perception may be a prominence map in the intermediate layer of the superior colliculus that modulates motor and attention/perceptual processes. This coordination comes with a cost: the misperception that briefly overlapping stimuli are separated in time. Our model predicts that abnormal intermediate layer of the superior colliculus inhibition, such as that arising from increased basal ganglia output, would affect the action and perception coupling, and it would worsen the misperception.

Objective: To test the prominence map model by measuring reaction times and perceptions in human intermediate layer of the superior colliculus dysfunction.

Methods: We measured the saccadic and perceptual reaction time changes and the percept for different temporal asynchronies between fixation point offset and peripheral target onset in Parkinson’s disease (PD).

Results: We found that increased basal ganglia inhibitory output to the intermediate layer of the superior colliculus prominence map disrupted the normal coupling of action and perception. With increasing temporal asynchronies, the PD perceptual reaction times increased approximately 3 times more than the increase of the saccadic reaction times. Also, PD subjects misperceive small overlaps as gaps for temporal asynchronies up to 3 times longer than controls. The results can be reproduced by an intermediate layer of the superior colliculus rostral-caudal gradient of inhibition.

Conclusion: These findings support the hypothesis that a prominence map in the intermediate layer of the superior colliculus couples action and perception through modulation of attention. A dysfunction of this network quantifies abnormal basal ganglia output and could underlie visual deficits, including common, yet poorly understood, misperceptions and visual-motor deficits of PD. © 2019 International Parkinson and Movement Disorder Society

Key Words: attention; eye movement; hallucination; priority; salience

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is computed by combining salience (the bottom-up, feature-dependent conspicuousness of the target) and priority (the top-down preference for a behavioral goal).2,7,9-11 Inter- and intralaminar connections within the superior colliculus (SC) and inputs to the SCi from the basal ganglia2,12 compare competitive locations, allowing the selection of the most prominent stimulus.13 Prominence determines both where and when to make the saccade and modulates the gain of the corresponding signal in the attention-perception system that gives rise to object perception. As a result, prominence influences both the time when the target of a saccade is identified and the time when that object is perceived. This temporally couples action and perception. This coupling, however, comes with a cost: a perceptual illusion. When the FP and TG compete, the prominence map requires more time to identify the target for the saccade and thus more time to perceive it. As a result, small overlaps are misperceived as gaps.

In this model, dysfunction of the SCi prominence map affects both the motor and attentional/perceptual processing, thereby decoupling action and perception. Such dysfunction could arise from a disruption of the neural network within the SCi and/or from abnormal external inputs, for example, increased SCi inhibition from the basal ganglia.

Parkinson’s disease (PD) is a neurodegenerative disorder caused by dopaminergic cell loss within the substantia nigra pars compacta, resulting in depletion of striatal dopamine and subsequent increased inhibitory output from the internal globus pallidus and SNr.14 The SNr targets the interneurons of the SCi,15,16 which are thus abnormally inhibited.

Besides motor manifestations, 90% of PD patients develop a variety of non-motor symptoms,17 commonly including visual deficits such as perceptual and attentional impairment and hallucinations,18-22 the pathophysiology of which is not well understood.19,23-26 The abnormal SCi inhibition in PD may contribute to these symptoms, given that our model predicts that PD patients would have abnormal temporal coupling of action and perception, which could cause misperceptions.

### Participants and Methods

#### Participants

Sixteen subjects were recruited by a movement disorders neurologist and met UK Brain Bank Criteria for PD diagnosis (Table 1).27 The study was approved by the NIH Institutional Review Board. All participants provided written informed consent before participation, but were naïve to the scientific purpose of the experiment.

All patients were assessed with the UPDRS part III in the off medication state before the experimental testing session (mean score: 32/72; range, 15/72–50/72) and H & Y staging (mean score: 2.2/5; range, 1.0/5–3.0/5). A neuro-ophthalmological examination before the experiment showed normal range of eye motion. Best-corrected visual acuity was evaluated using Snellen Charts (mean score: LogMAR 0.05; range, –0.10 to 0.30). Some patients had very mild refractive errors, but correction of vision was not necessary for recordings because targets were bright and high contrast. Patients were recorded when off medication for at least 12 hours.

Data from 2 subjects (7 and 9) were excluded, 1 because of equipment failure and 1 because he did not meet the protocol criteria. Data from the 12 healthy volunteers in our previous study,7 reanalyzed with the same methods used for PD patients, acted as the control (CT) group.

#### Visual Stimuli and Apparatus

Visual stimuli were red laser spots (3-mm diameter, 0.16-degree visual angle) projected onto a screen positioned 105 cm from the subject’s eye. Horizontal and vertical eye movements were recorded at 1 kHz from the right eye using an infrared iView X Hi-Speed tracker (SensoMotoric Instruments, Teltow, Germany). Only horizontal movements were analyzed. Viewing was binocular. The subject’s head was stabilized by a chin rest, a forehead rest, and a strap around the head. Each session began by calibrating the stimulus display and the eye monitor.

#### Statistical Analysis

Psychometric functions (PFs) were fitted (Psignifit version 4 software package for Matlab; The MathWorks, Inc., Natick, MA)28 to the perceptual responses (percent overlap responses as a function of TA) using Bayesian inference (Markov chain Monte Carlo method).29 The PF was a normally distributed, beta-binomial model with a single degree of freedom.28 The same function was used

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**TABLE 1.** Demographic and clinical data of patients with PD, with psychophysical thresholds in SP task

<table>
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<tr>
<th>PD</th>
<th>Age (Years)</th>
<th>Sex</th>
<th>UPDRS-III OFF</th>
<th>H &amp; Y</th>
<th>Threshold TA (ms)</th>
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for all subjects, to facilitate comparisons between patients and controls, leading to small (but insignificant) changes in fits for controls from those reported in the previous study. This model has five parameters: threshold (m), width (w), lapse rate (λ, upper asymptote), guess rate (γ, lower asymptote), and a scale factor (η) for the standard deviation of the normal distribution. The chance threshold occurs at \((γ + 0.5(1 − γ − λ))\), not 0.5.

Design

The experimental design was adapted from Saslow’s gap/overlap paradigm,6 with which he identified the gap effect in healthy subjects: with respect to the synchronous condition, saccadic reaction times are reduced when the FP disappears before the TG appears (gap) and are increased when the TG appears before the offset of the FP (overlap). The SCi is the main neural substrate of the gap effect: the FP onset or offset causes the maintenance or release, respectively, of the rostral neurons’ inhibition on the caudal saccade-related neurons, increasing or reducing the saccadic reaction times.30 In PD, saccadic reaction times have been reported after gaps,32-34 but it is not known whether reaction times increase for overlaps.

In our paradigm, a central FP with a duration randomly drawn from the set (1,100, 1,200, 1,300, 1,400, or 1,500 ms) turned on. Later, the TG appeared at an eccentric location (6 or 12 degrees, left or right, randomly; duration, 1,500 ms). The TG onset could follow the FP offset after a short time (TA < 0, gap), it could be simultaneous with the FP offset (TA = 0, synchronous), or it could precede the FP offset (TA > 0, overlap). Gap, synchronous, and overlap TAs were presented pseudorandomly during each session. We tested 13 TAs (0 and gap and overlap of 50, 100, 150, 200, 300, and 500 ms). The next trial started 200 ms after the TG was turned off. Subjects took part in three sessions, plus a brief practice session, in 1 day. Each TA was repeated approximately 13 to 18 times per session. Every 5 or 6 minutes, subjects were given 1 to 2 minutes of rest time; hydrating eye drops were provided, if requested.

Session One: Control Session, Saccade-Only Task (SO)

The subject was instructed to look at the FP and to maintain fixation until the appearance of the TG. Then, the subject was asked to make a saccade to the TG as quickly and accurately as possible. No perceptual report or button press was required.

Practice Session: A Brief Session to Ensure Appropriate Understanding of the Task

The paradigm was slowed down to make the task easier, and only large TAs (±200 and ±500) were used.

Session Two: Main Paradigm, Saccade + Perception Task (SP)

The subject held a two-button box. Instructions were as in the SO, but after looking at the TG, the subject was requested to push the bottom button (marked gap) to indicate that he or she saw a gap (perceiving a period when no lights were on) or the top button (marked overlap) to indicate the perception of an overlap (both lights on at the same time). The subject received no feedback about his or her performance. The button press did not terminate the trial, and subjects were instructed to fixate the TG until its disappearance. Further details are available in our study of control subjects.7

Session Three: Control Session, Perception-Only Task (PO)

The stimulus sequence was the same as in the previous session, but subjects were asked to maintain fixation on the FP, without making a saccade when the TG appeared. After TG onset, they pressed a button to indicate whether they perceived a gap or an overlap.

Data Analysis

Data were analyzed offline with algorithms developed by our laboratory in Matlab (The MathWorks, Inc.). Eye position data were obtained by smoothing the raw data with a Savitzky-Golay filter (golayfunction35 with order four and frame length 21). Eye velocity was obtained from raw eye position data with a Savitzky-Golay differentiating filter (order two and frame length 25). Saccades were detected when velocity crossed 0.3 times the standard deviation of the eye velocity trace during fixation. Saccade end was defined as when the velocity decreased below the same threshold. In the SP and SO sessions, peak velocity, amplitude (degrees of visual angle between saccade start and end), and duration (time between saccade start and end) were measured for the first saccade after TG onset. Only trials without artifacts (such as blinks or saturation of eye tracker signal) were analyzed. Trials were excluded whenever: the subject did not look at the FP at the start of the trial and did not maintain fixation within ±1 degree; the first saccade was made away from the target; saccadic latency was <80 or >500 ms; and peak speed was <25 or >800 degrees/sec. In the SP session, if the subject made no saccade after the TG onset, or did not press a button, or pressed the button before the saccade, the trial continued but was considered incorrect and was not analyzed. In the PO session, trials with saccades were ignored.

Saccadic and perception report (button press) reaction times were measured from TG onset. Whenever subjects pushed a button in the SP and PO sessions, both the perception (gap or overlap) and its time were recorded. If the subject pressed a button twice, or pressed first one button and then another, we took as valid only the first button
press after the TG. In the SP session, a valid response had to follow a valid saccade.

Each session was analyzed separately. For each TA, we calculated the average saccadic and perception reaction times and the number of gap and overlap responses. Data were first analyzed for each subject. We then analyzed the pool of all subjects. Correlation between patient characteristics (e.g., age) and thresholds was performed in Excel (Microsoft, Redmond, WA).

Model

The model used in this article has been described in complete detail previously. Briefly, the model combines a model of perception that accumulates evidence to a threshold with a model of attention that uses divisive normalization. The spatial prominence map in the SC provides the gain field for the normalization. Simulations were performed in Matlab (version R2018b; The MathWorks, Inc.).

Results

Saccadic and perceptual reaction times and perceptual transition thresholds were studied as a function of the TA. Saccadic and perceptual reaction time-change comparison slope and perceptual transition threshold were independent of TG direction and eccentricity and age of the subjects. Figure 1 shows an example of saccade and perception responses of 1 PD subject during the dual task.

Saccade and Perceptual Reaction Time Dependence on TA

Saccadic reaction times in PD are dependent upon TA in both SO and SP conditions (Fig. 2A, single subject; Fig. 2B, pool of all subjects). With respect to the synchronous condition, saccadic reaction times are reduced for gaps and increased for overlaps, demonstrating the gap effect in PD. On average, however, the range of the effect is smaller than normal: the time-change upon TA (longest – shortest saccadic reaction time) in CT was approximately 120 ms,\(^7\) consistent with the effect observed by Saslow\(^6\); in PD, instead, the range of the effect is <50 ms.

The perception report reaction time (button press) also depends on TA in PD, with gap reaction times shorter than overlap reaction times (Fig. 2C,D). Perceptual reaction times of PD patients appear longer than in controls (by \(~\sim 150\) ms), but the effect of the time-change is close to normal (for the pooled data, \(~\sim 200\) ms in PD vs. \(~\sim 150\) ms in CT). As previously discussed for our normal subjects,\(^7\) the perception report reaction time can be considered as resulting from two main components: the time to form the conscious perception and the time to plan and execute the button press (manual reaction time). Manual reaction times can be slightly shortened by a gap between stimuli, but they do not increase when there is an overlap.\(^3\) Therefore, we previously concluded that the perceptual report reaction time-change dependence upon TA (shape of the curve) is mainly attributable to a dependence upon TA of the time to build the perception. In PD, manual reaction time and movement execution are usually longer than normal.\(^3\) Our patients likely required a longer time to push the button, causing an

![Figure 1](image-url)
upward bias of the curve. Nonetheless, also in PD, the slope of the curve is attributed to the dependency upon TA of the time to build the perception.

When compared, the PD saccadic and perceptual reaction time changes look correlated, given that they both increase with increasing TAs, but they are not 1:1 coupled (i.e., they do not increase by the same amount after different TAs), as in healthy subjects (Fig. 2E,F).

F-tests reveal that the linear regression is only significant in the control case.

Perceptual Transition and Misperception of Small Overlaps as Gaps

In PD, the transition from perceiving a gap to perceiving an overlap is shifted to the right with respect to
controls (Fig. 3). Thus, PD patients cross the transition from gap to overlap significantly later than normal, which causes a misperception of even longer overlaps as gaps (overlaps up to ~191 ms are misperceived in PD vs. overlaps up to ~78 ms in CT). Because there was no third response option (e.g., “synchronous”) to exclude a bias toward responding “gap” at TA = 0, we looked at the data from subjects with a nonzero guess rate. The responses of these subjects did not deviate (above or below) that rate at TA = 0. Thus, we do not find any evidence for such a bias at synchrony.

We found no significant correlation between transition threshold and UPDRS-III score (R = 0.031; P = 0.917): lack of correlation could be attributed to the relatively uniform disability score of our patients’ sample or the inclusion in the UPDRS-III of symptoms that might have a heterogeneous pathological substrate. Neither was a correlation found between patients’ lapse rate (failure to say overlap for large TAs) and UPDRS-III score (R = −0.065; P = 0.826).

Simulations

In our previous study, we implemented a functional model of action and perception by combining models for perception and attention with a collicular map of prominence. This model simulates the time of perception of the FP and TG and therefore can determine the TA where the perceptual response occurred. In the model, a subject starts to consciously perceive a given stimulus when the object activity crosses above a threshold or stops perceiving it when the object activity crosses below that threshold. Prominence in the SCi modulates the gain of the object’s signal in the perceptual pathway and thus the time when the object-related activity crosses the threshold. Thus, our model simulates the time of perception of the FP and TG and therefore can determine the TA where the perceptual response occurred.

FIG. 3. Psychometric function for perceptual transition threshold between responding gap and overlap, as a function of TA. Dark lines are for the saccade + perception (SP) task, and light lines are for the perception-only (PO) task. (A) Single patient (PDS) with PD. Transition threshold in the SP task was 254 ms (95% confidence interval [170, 500]), with upper and lower asymptotes and standard deviation scale parameters (λ, γ, η): 0.29, 0.00, and 0.29. In the PO task, the threshold was 199 [155, 283] ms, with fit parameters 0.18, 0.00, and 0.30. Vertical lines indicate where the threshold for estimating the transition from responding “gap” to responding “overlap” occurred. The corresponding horizontal line indicates the threshold at (γ + 0.5(1 − γ − λ)). (B) Pooled patients with PD, compared to pooled controls. In the SP task, transition threshold for controls (CT) was 78 [64, 92] ms, with fit parameters 0.04, 0.07, and 0.10. In the PO task, transition was at 60 [35, 82] ms, with fit parameters 0.05, 0.05, and 0.17. For PD patients, in the SP task, the threshold was at 191 [138, 282] ms, with parameters 0.19, 0.10, and 0.10. In the PO task, the threshold was at 166 [127, 270] ms, with parameters 0.24, 0.12, and 0.10. Thus, transition thresholds for PD patients were ~109 ms longer than for controls. The upper asymptote in PD was much smaller than that of CT, and the lower asymptote was bigger than that of CT, indicating that PD patients had more lapses and guessed more often than controls. The standard deviation scale factors were similar in both PD and CT. Note that the transition from saying “gap” to saying “overlap” was also much more drawn out (lower slope). All of which suggests that making the discrimination between zero or two lights was more difficult for PD patients than for normal controls.
changes from gap to overlap. We can simulate the increased SNr inhibition on the SCi by modulating the prominence map. SNr inhibition could be uniformly increased across the SCi, causing a uniform decrease of prominence at both the caudal and rostral poles; alternatively, the inhibition might not be uniform, creating a rostral-caudal gradient in the prominence map.

The simulation in Figure 4A shows the activity on the perceptual map (the output of the model), Figure 4B shows output of the prominence layer, and lower panels (C,F) show the vision input. TG is the activity in the layer at the locus of the target (blue), and FP is the activity at the fixation point (magenta). An object enters conscious perception when it crosses above a varying threshold (black line) in (A). The maximum threshold level was adjusted so that the fixation point offset and target onset (TA = 78 ms) are perceived at the same TA as our normal subjects in Figure 3 (CT, dotted lines, rostral and caudal gain = 1.0) (A) Simulation of the model’s perception layer for rostral and caudal gains, FP:TG = 1.0:1.0 (control simulation, CT, dotted lines, perceptual transition threshold m = 78 ms) and FP:TG = 0.2:0.2 (PD simulation, solid lines, m = 58 ms). When the gain is uniformly lowered, the illusion of synchrony requires less overlap (m = 58 vs. 78 ms). (D) Same as in (A), but the ratio FP:TG = 1.5:0.2. Synchrony occurs much later in PD than in CT (m = 225 vs. 78 ms). (B,E) Output of the prominence layer of the model. This layer acts as a multiplicative modulator, so its value is normally 1 and deviates above and below 1 as lights turn on and off. There is reciprocal inhibition between rostral and caudal parts of the layer, which causes, for example, a dip at the TG locus (blue) and a rise at the FP locus (magenta) when the FP turns on. (E) Effect on threshold (m) when the gain of the rostral and caudal parts of the prominence map in the SC change. There is a mild effect of changing the caudal gain (along the abscissa), but a large effect of changing the rostral gain (along the ordinate). The rostral/caudal ratio of 1.5:0.2 matches the average of our subjects (Fig. 3, CT, m = 78; PD, m ~ 225 ms). SC rostral gains >1 presumably result from a loss of inhibition from the caudal SC, which is inhibited by the SNr. Panel (G) shows a diagram of the model; green blocks are the attention normalization part, purple blocks are the object perception blocks, and blue is the prominence map in the SC. Panel (H) shows a map of the threshold as a function of rostral and caudal gains. Later thresholds are in warmer colors. The red line indicates where rostral and caudal gains are equal. Above that line, the threshold increases markedly.

FIG. 4. Simulation. Effects of different rostral/caudal prominence gains on the perceptual transition in PD. We hypothesize that in PD the increased activity in the SNr causes excess inhibition in the SC. Upper panels (A,D) show the output of the perception layer of our model,7 middle panels (B,E) show output of the prominence layer, and lower panels (C,F) show the vision input. TG is the activity in the layer at the locus of the target (blue), and FP is the activity at the fixation point (magenta). An object enters conscious perception when it crosses above a varying threshold (black line) in (A). The maximum threshold level was adjusted so that the fixation point offset and target onset (TA = 78 ms) are perceived at the same TA as our normal subjects in Figure 3 (CT, dotted lines, rostral and caudal gain = 1.0). (A) Simulation of the model’s perception layer for rostral and caudal gains, FP:TG = 1.0:1.0 (control simulation, CT, dotted lines, perceptual transition threshold m = 78 ms) and FP:TG = 0.2:0.2 (PD simulation, solid lines, m = 58 ms). When the gain is uniformly lowered, the illusion of synchrony requires less overlap (m = 58 vs. 78 ms). (D) Same as in (A), but the ratio FP:TG = 1.5:0.2. Synchrony occurs much later in PD than in CT (m = 225 vs. 78 ms). (B,E) Output of the prominence layer of the model. This layer acts as a multiplicative modulator, so its value is normally 1 and deviates above and below 1 as lights turn on and off. There is reciprocal inhibition between rostral and caudal parts of the layer, which causes, for example, a dip at the TG locus (blue) and a rise at the FP locus (magenta) when the FP turns on. (E) Effect on threshold (m) when the gain of the rostral and caudal parts of the prominence map in the SC change. There is a mild effect of changing the caudal gain (along the abscissa), but a large effect of changing the rostral gain (along the ordinate). The rostral/caudal ratio of 1.5:0.2 matches the average of our subjects (Fig. 3, CT, m = 78; PD, m ~ 225 ms). SC rostral gains >1 presumably result from a loss of inhibition from the caudal SC, which is inhibited by the SNr. Panel (G) shows a diagram of the model; green blocks are the attention normalization part, purple blocks are the object perception blocks, and blue is the prominence map in the SC. Panel (H) shows a map of the threshold as a function of rostral and caudal gains. Later thresholds are in warmer colors. The red line indicates where rostral and caudal gains are equal. Above that line, the threshold increases markedly.
Also, because of the normal rostral-caudal reciprocal inhibition, when we decreased the gain in the caudal SCi, we also increased the gain of the rostral SCi; Figure 4H shows the threshold as a heatmap dependent upon the gain of the rostral and caudal inhibition to SCi. The red line shows equality of gains. The threshold only increases markedly when the rostral gain is greater than the caudal gain.

Discussion

In PD Action and Perception Are Temporally Correlated, but Not Coupled, and the Misperception Is Worse

In PD, as in controls, there is a dependency of saccadic and perceptual reaction times upon the TA between FP and TG (Saslow’s gap effect). However, whereas in healthy subjects the time-change of action and perception is 1:1 coupled (the 2 times increase or decrease by the same amount with TA), in PD action and perception are correlated, but not linearly related: for small increases of the saccadic reaction times, the perceptual reaction times increase much more (approximately 1:3). Also, PD subjects misperceive small overlaps as gaps for overlapping TAs approximately 3 times as long as in controls. The decoupling of action and perception might be explained if action could partly bypass the increased basal ganglia inhibition onto the SCi, whereas perception could not.

Reflexive saccades, as in our task, can be generated by direct projections from the parietal cortex onto saccade-related neurons in the deeper layers of the SC. This pathway is supposed to largely bypass the basal ganglia circuit, which explains why reflexive saccades are basically preserved in PD. Saccades generated through this pathway would be less modulated by the SCi. This also explains the reduced saccadic gap effect in PD, because the SCi is the main neural substrate for the gap effect.

In contrast, perception may not skip the increased basal ganglia inhibition on the SCi (the prominence map). Prominence still modulates the gain of the target’s signal in the perceptual pathway. As a result, in PD the prominence map takes longer to develop. Thus, action (skipping SCi) and perception (SCi dependent) become temporally decoupled.

Moreover, a different collicular rostral/caudal inhibition in PD explains the misperception of longer overlaps.

Impact of Action/Perception Temporal Decoupling and Misperception of Overlaps on the Visual Performance of PD Patients

Several visual deficits have been described in PD, but their neural substrates are not always clear. Increased basal ganglia inhibition on the SC, a crucial structure for both voluntary and reflexive saccades, might cause eye movement abnormalities.

Some of these abnormalities, such as the facilitation of small saccades, large square wave jerks, and abnormally fragmented saccades, suggest an increased SC rostral activity in PD. A nonuniform rostral-caudal inhibition might contribute to the hypokinetic status of PD, because not only movements would be inhibited, but also a fixational/foveal activity would be promoted. Indeed, our subjects were able to correctly maintain fixation in the PO task, where no saccades were required.

Eye-movement abnormalities might underlie some impaired behaviors in PD, such as visual search: patients scan smaller areas than normal subjects with fewer hypometric saccades and longer fixations. However, the saccadic abnormalities alone explain poorly many other visual deficits and symptoms reported by PD patients, such as attention and working memory dysfunctions, reading and driving difficulty, visual spatial deficits, and misinterpretation of peripheral stimuli. The loss of temporal coupling of action and perception that we have shown could affect many of the above-listed functions that require fine motor-visual coordination.

Moreover, whereas PD patients usually have normal visual acuity, dopaminergic depletion in the retina might cause decreased color discrimination and decreased retinal contrast sensitivity. However, a degraded foveal input alone is unlikely to be responsible for impaired higher-level visual functions, such as visual hallucinations and misperceptions, which affect most patients with PD by the onset of, or even before, the motor symptoms. The pathophysiological mechanism of visual hallucinations in PD is poorly understood, but attentional and perceptual processing impairments are proposed as possible underlying dysfunctions by most models of PD hallucinations. Given that prominence in our model modulates both the attentional and perceptual pathways, its impairment could contribute to the inability to recruit activation in the dorsal attention network and/or to the presence of an ambiguous percept, both of which have been hypothesized as responsible for misperceptions and hallucinations.

Earlier studies in PD have focused mainly on subjectively reported and/or qualitative hallucinations. The misperception that we report has additional benefits in that it can be easily quantified, and it arises from the dysfunction of a mechanism with a defined anatomical localization. Thus, in PD this misperception may represent a suitable parameter to assess disability and, because it results from the nigral-collicular inhibition, may also be able to quantify abnormal basal ganglia output.

In conclusion, this study supports our hypothesis that a mechanism in the SCi couples action and perception by modulating motor and attentional/perceptual processing. Increased basal ganglia inhibition of the SCi in PD may be responsible for decoupling action and perception and...
causing misperceptions as gaps of longer overlaps than normal. Action/perception decoupling and misperception in PD might affect complex functions that require fine action-perception coordination and attentional and perceptual processing.

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References


Author Roles

(1) Research Project: A. Conception, B. Designed and Performed the Experiments, C. Critical Review of Study Design; (2) Manuscript: A. Writing of the First Draft, B. Review and Critique; (3) Other: A. Recruited the Patients With Parkinson’s Disease, B. Collected the Patient’s Clinical and Demographic Data, C. Performed the Neurological Evaluations and UPDRS Assessments, D. Performed the Neuro-ophthalmological Evaluations, E. Provided the Eye Movement Laboratory, F. Analyzed the Data, G. Critical Review of the Data Interpretation.

E.P.: 1A, 1B, 1C, 2A, 2B, 3F, 3G
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L.M.O.: 1A, 1B, 1C, 2B, 3F, 3G

Financial Disclosures

Dr. Hallett may accrue revenue on US Patent #6,780,413 B2 (Issued: August 24, 2004): Immunotoxin (MAB-Ricin) for the treatment of focal movement disorders, and US Patent #7,407,478 (Issued: August 5, 2008): Coil for Magnetic Stimulation and methods for using the same (H-coil); in relation to the latter, he has received license fee payments from the NIH (from Brainsway) for licensing of this patent. He is on the Medical Advisory Boards of CALA Health and Brainsway. He is on the Editorial Board of approximately 15 journals and receives royalties and/or honoraria from publishing from Cambridge University Press, Oxford University Press, Springer, and Elsevier. Dr. Hallett’s research at the NIH is largely supported by the NIH Intramural Program. Supplemental research funds have been granted by Merz for treatment studies of focal hand dystonia, Allergan for studies of methods to inject botulinum toxins, Medtronic, Inc. for a study of DBS for dystonia, and CALA Health for studies of a device to suppress tremor. Dr. Vanegas has received teaching honoraria from Neurocrine and consultation compensation from AbbVie.