

INTRODUCTION

Parkinson disease (PD) is a neurodegenerative disease that affects the basal ganglia, which is involved with the selection and implementation of action sequences. Investigations suggest an extension of deficits to the speech motor and linguistic systems.

Comparison of findings from studies examining voice onset time (VOT) in the speech of individuals with PD are mixed. Some studies have found significantly longer VOTs for voiced stops in parkinsonian speech [1], while others have found VOTs for voiceless stops to be significantly shorter than control speakers [2]. Still others have found no differences in VOT durations between individuals with PD and controls [3]. These data suggest that VOTs are neither systematically delayed nor systematically advanced in parkinsonian speech, relative to control speakers.

The VOTs for voiced and voiceless stops are expected to differ for both individuals with PD and controls. However, inefficiencies in the sequencing or implementation of the voicing gesture may result in less VOT contrast (i.e., smaller difference) between voiced and voiceless cognates [4-6]. The current study examined VOT voicing contrast in individuals with PD and controls.

METHODS

Participants: Eighteen individuals, 9 patients with PD and 9 neurologically healthy controls, participated as speakers in the current investigation. For the current investigation, the speakers with PD were considered to be in the OFF medication state (mean withdrawal time = 12.05 hrs; Range: 9.5 to 16 hrs).

Speech Task: Each speaker read a corpus of stimuli designed to evaluate VOT. Participants produced the carrier phrase, "CVp again", with the "C" representing all 6 stop consonants, /p, t, k, b, d, g/, and the "V" representing the four corner vowels, /i, ae, a, u/. The "CVp" combinations were randomized and the protocol was completed twice resulting in a total of 48 tokens per participant.

Measures: The raw VOT was measured as the time interval from the burst release of the stop consonant to the onset of voicing. In cases of pre-voicing for a voiced stop, the VOT was measured as the interval of time from the onset of voicing to the burst release and the value was entered as a negative VOT.

Statistics: Because a each participant produced a number of tokens, a linear mixed model analysis was used to evaluate group differences in the VOT duration. To account for within participant variability, the specified model included both fixed and random effects. Fixed factors included group, as a between-subjects factor, and voicing, as a within-subjects factor, along with the group X voicing interaction. The individual participants and each observation were modeled as random effects, making the final model a random-intercept, random-slope model. Therefore, the model accounted for the within participant variability in VOT, as well as the variability in the voiced-voiceless contrast for each participant.

RESULTS

Significant main effect of voicing cognate [$F(1,745)=732.67$; $p<0.0001$]

- VOTs for voiceless stops were on significantly longer than voiced stops [Estimate=96 ms; $p<0.0001$].

Trend for main effect of group [$F(1,16)=3.283$; $p=0.09$]

- Fixed Effects revealed VOTs were on significantly longer for PD than controls [Estimate=39 ms; $p=0.001$].

Significant group X voicing interaction [$F(1,745)=56.54$; $p<0.0001$]

- Individuals in the PD group exhibited a smaller degree of difference in the VOT of voiced and voiceless cognates [Estimate= -42 ms difference; $p<0.0001$].
- Percent overlap and standardized difference between the voiceless and voiced VOT durations were calculated for each participant.
 - Participants in the PD group exhibited significantly less difference between the VOTs of voiced and voiceless cognates.

Percent Overlap in VOT ($p=0.02$):

PD Mean=49% (SD=30%) | Control Mean=18% (SD=20%)

Standardized Difference in VOT ($p=0.001$):

PD Mean=1.1 (SD=0.3) | Control Mean=1.5 (SD=0.2)

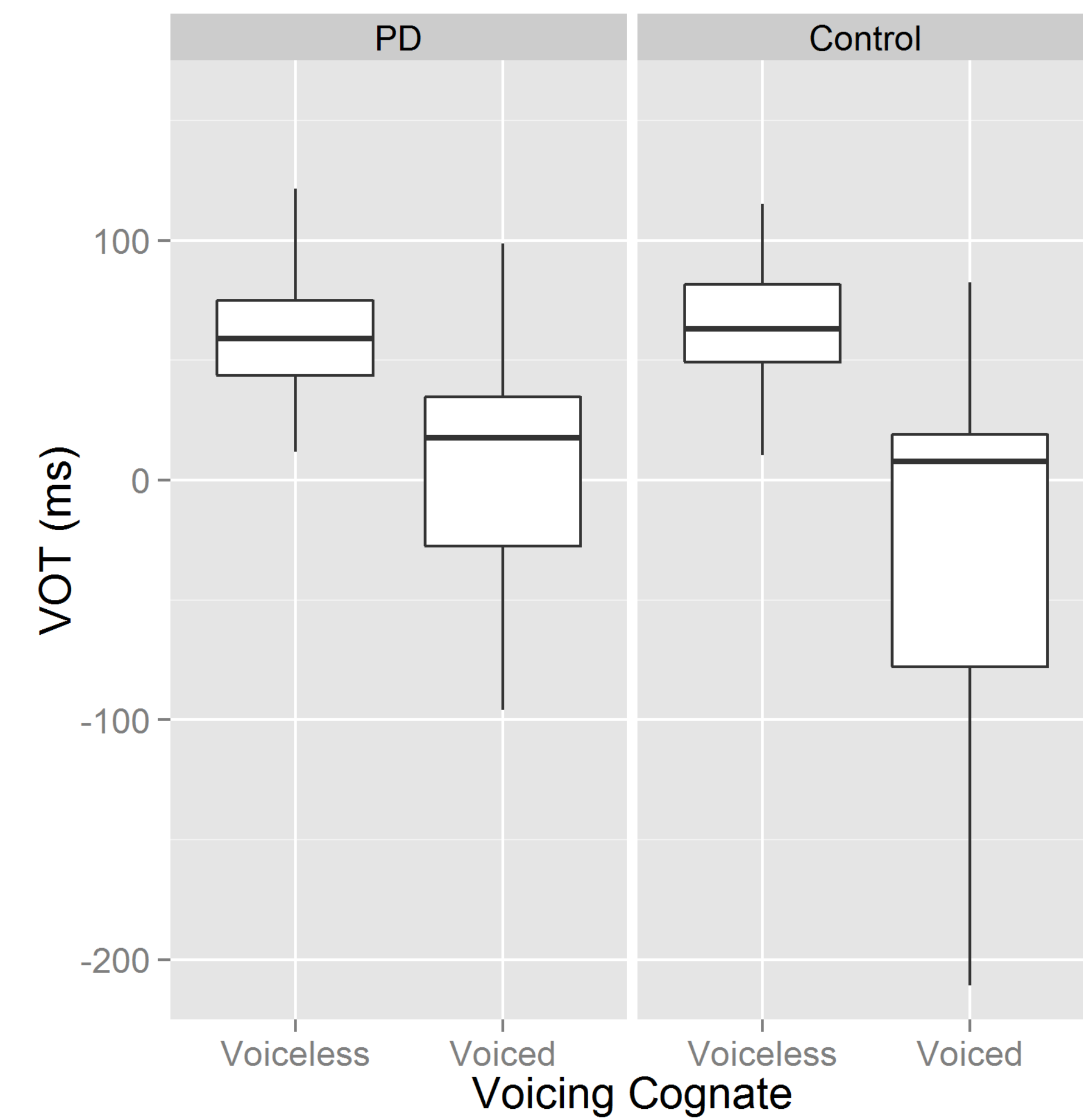


Figure 1. Box plot of all voice onset time (VOT) values produced by participants in the Parkinson disease (PD) and control groups.

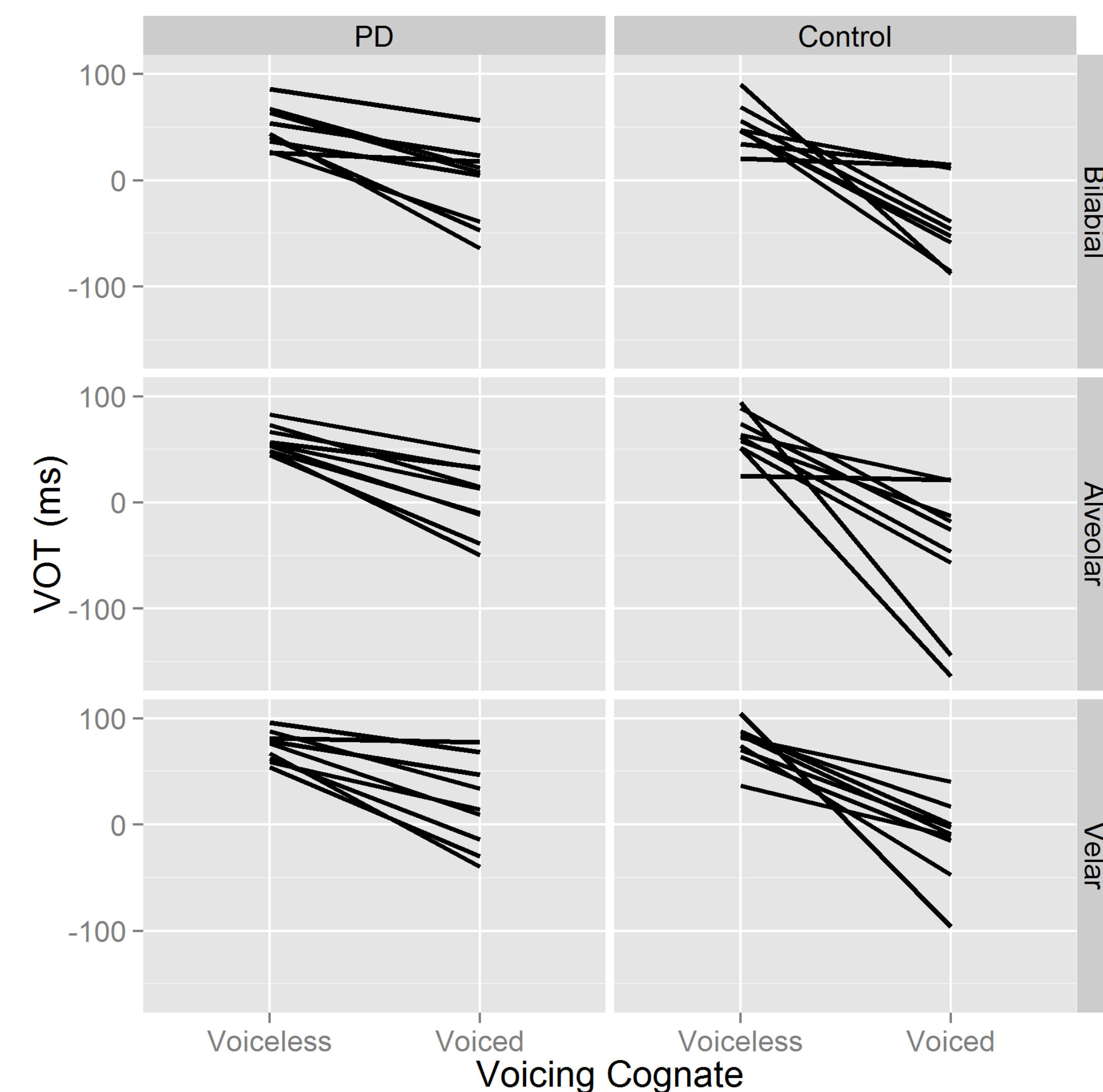


Figure 4. Voice onset times (VOT) for each participant by place of articulation showing the degree of change in VOT between voiced and voiceless cognates.

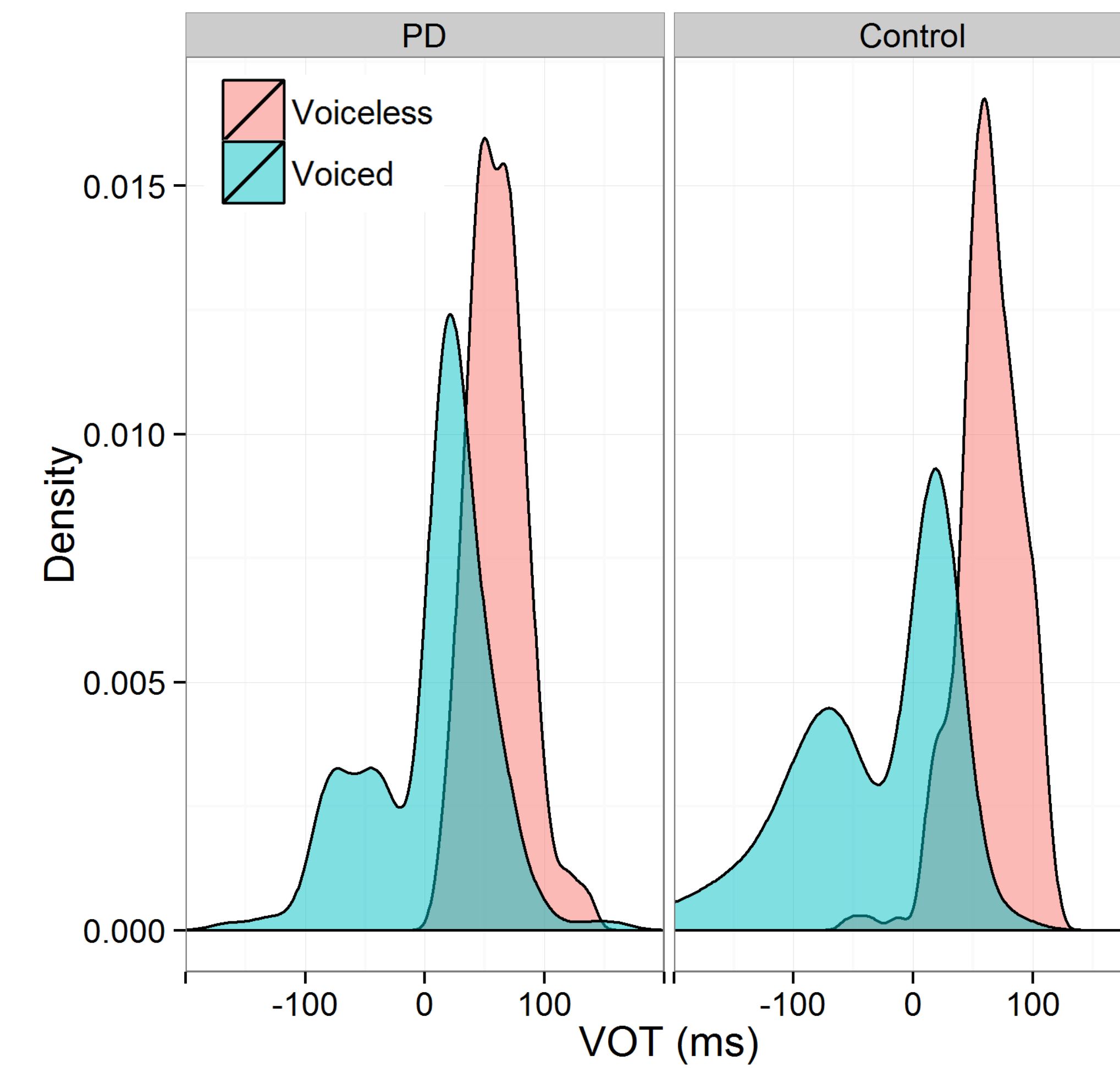


Figure 2. Distributions showing the degree of VOT overlap between voiced (blue) and voiceless (red) stops for the Parkinson disease (PD) group (left) and control group (right).

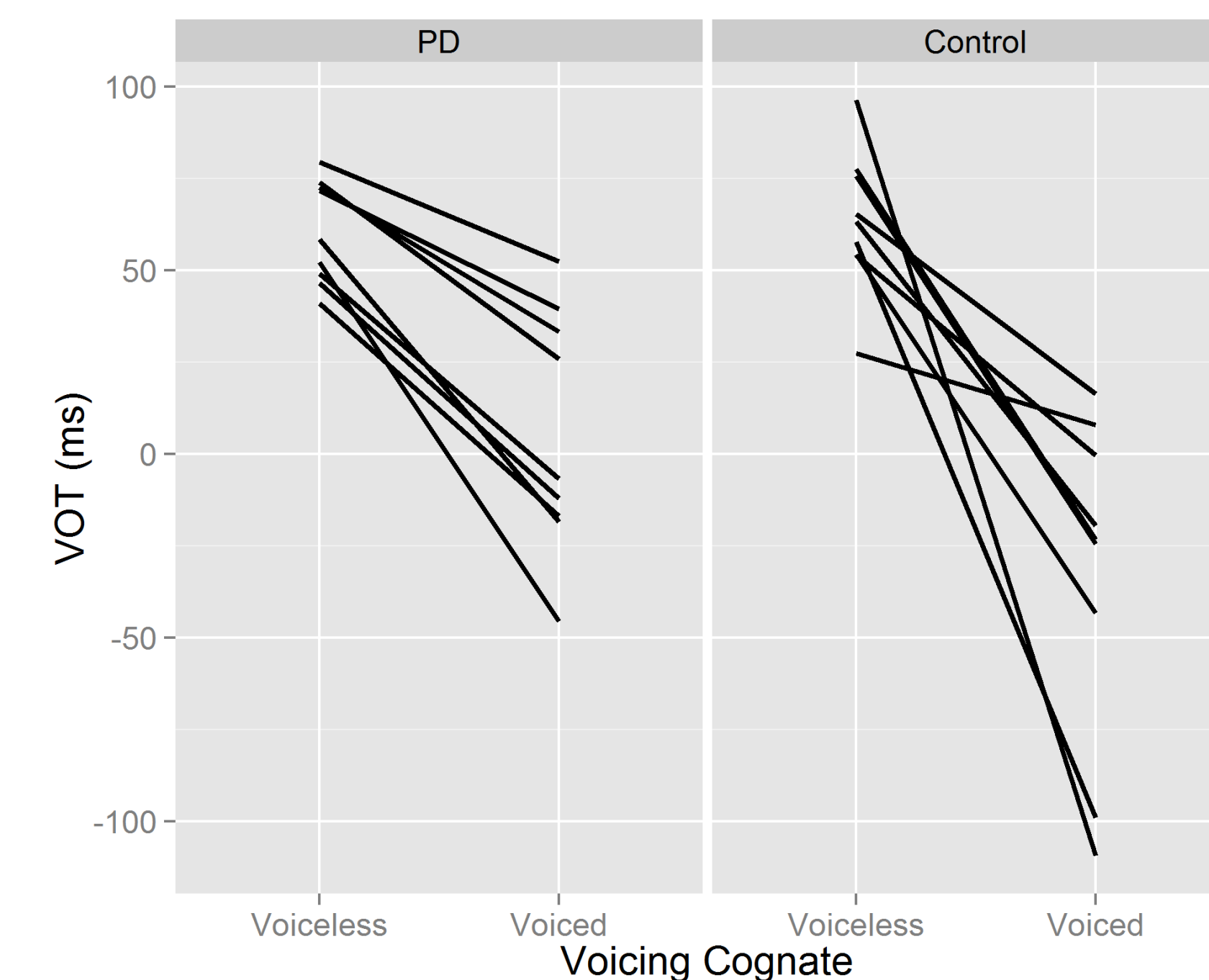


Figure 5. Fixed effect estimates showing the degree of change in the voice onset time (VOT) between voiced and voiceless cognates for each participant.

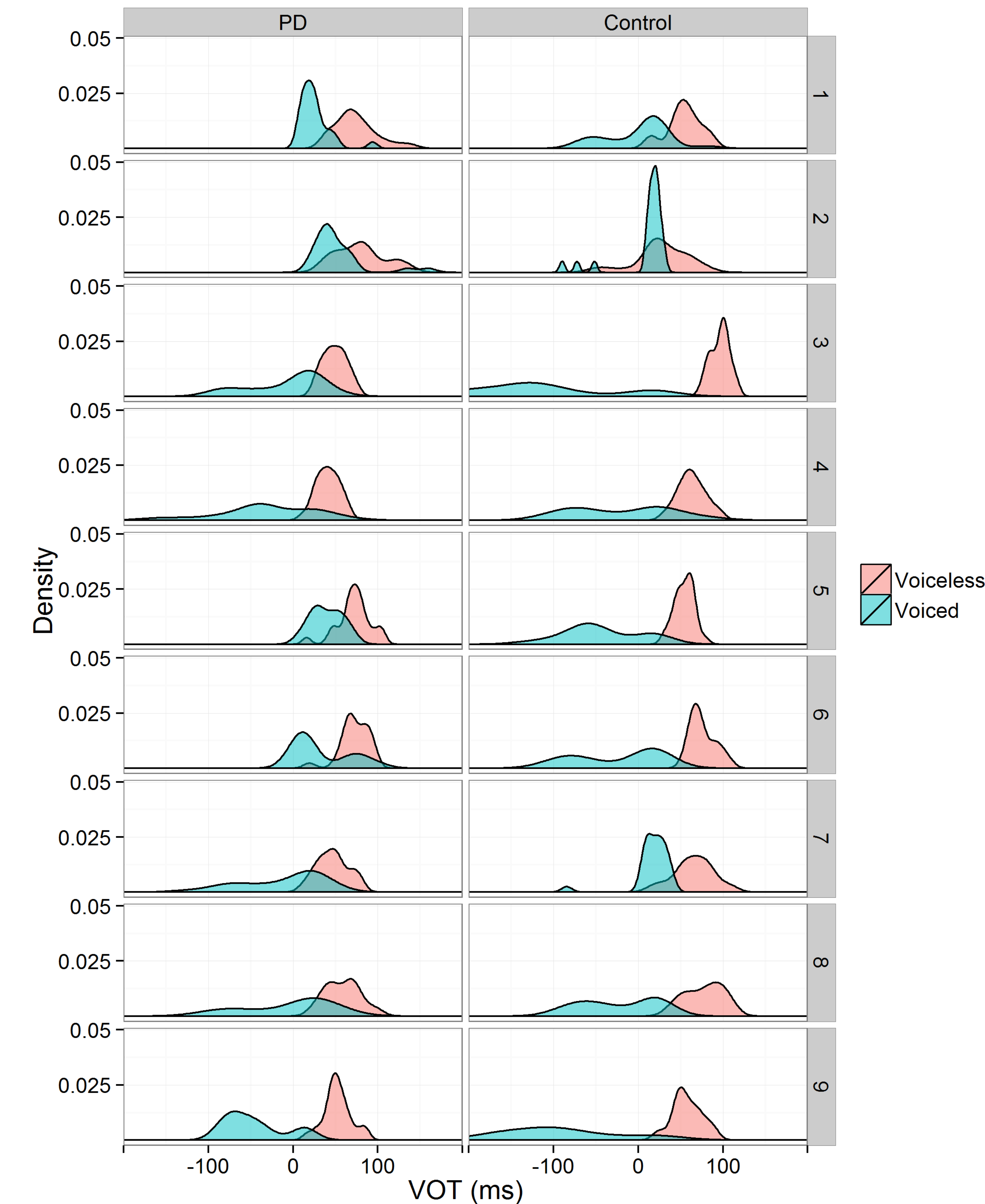


Figure 3. Individual voice onset times (VOT) distributions for participants in the Parkinson disease (PD) group (left panes) and control group (right panes) showing the degree of overlap between voiced (blue distributions) and voiceless (red distributions) stops.

CONCLUSIONS

The current study examined the effect of Parkinson disease (PD) on voice onset time (VOT) with specific emphasis on voicing contrast. These data show that while individuals with PD do exhibit significant differences in VOT between voiced and voiceless stops, the temporal contrast between voicing cognates is significantly less than healthy controls. In light of the current results, two hypothesis seem plausible. First, the decreased VOT contrast may result from hypokinesia or undershoot of the abduction-adduction system, reflecting production deficits. For this hypothesis, decrease range of motion of the adduction gesture would result in less temporal differentiation of voiced and voiceless stops. Alternatively, it is possible that the lesser VOT contrast may arise from deviant contrast enhancement affecting temporal organization of the voicing gesture, reflecting higher order deficits with implementation of speech motor plans. For this hypothesis, decreased contrast enhancement of the voicing action would likely result from dopamine-related dysfunction of the cortico-basal ganglia circuits.

References

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