REGULAR ARTICLE

Neurophysiological Evidence of Motor Preparation Dysfunction during Inner Speech and its Association with Auditory Verbal Hallucinations in Schizophrenia Spectrum Disorders

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Background and Hypothesis: Auditory verbal hallucinations (AVH) are hypothesized to result from failures in corollary discharge mechanisms to correctly predict self-initiated inner speech. However, the role of motor preparation in inner speech, during which sensorimotor predictions are formed, remains unclear. This study aimed to test the hypothesis by examining the relationship between AVH and an electrophysiological marker of action preparation: the contingent negative variation (CNV).
Study Design: Participants completed an electroencephalographic paradigm. In the Active condition, they imagined an inner syllable at a cued moment coinciding with the presentation of an audible syllable. In the Passive condition, participants passively listened to audible syllables. The amplitude of the late CNV preceding inner speech production was compared with that associated with passive listening across 3 groups: (1) schizophrenia spectrum patients with current AVH (SZAVH+, *n* = 58), (2) schizophrenia spectrum patients without current AVH (SZAVH-, *n* = 50), and (3) healthy controls (HC, *n* = 49).
Study Results: The HC group showed a more negative late CNV in the Active condition compared with the Passive condition. In contrast, the SZAVH+ and SZAVH- groups showed positive-going slow cortical potentials in both conditions, with less positivity in the Active condition in the former. This pattern significantly predicted AVH status.
Conclusions: These findings provide evidence of motor preparation dysfunction during inner speech in schizophrenia spectrum disorders. The distinct pattern of deficits observed in hallucinators may reflect imprecise corollary discharges theorized to underlie some AVH. Premovement neural indices may provide a novel window into abnormalities in prediction formation.

Key words: contingent negative variation; readiness potential; covert speech; corollary discharge; schizophrenia.

Introduction

Auditory verbal hallucinations (AVH) in schizophrenia have been suggested to arise from abnormalities in corollary discharge mechanisms related to inner speech[1–6]—a fully covert "action" sometimes argued to be our most complex motor act.[7] Specifically, the *forward model* suggests that when an action is initiated, a copy of the motor command is used to predict, and often attenuate, the resultant sensory consequences of the action (dubbed sensory suppression), thereby giving us the ability to self-monitor and attribute internally-generated percepts as own,[8–10] whereas failures in these processes are thought to underlie some of the characteristic symptoms of schizophrenia, including AVH.[11] More recently, neurophysiological studies have shown that inner speech, similar to overt actions, engages the corollary discharge mechanisms,[12–17] and that this inner speech-specific predictive process is uniquely disrupted in schizophrenia spectrum patients with current AVH.[18–20]

However, it is unclear whether AVH is related to motor preparation dysfunction during inner speech. A negative-going slow wave can be observed in electroencephalography (EEG) a few seconds prior to the execution of an action.[21] Depending on whether the action is voluntary (uncued) or is prompted (cued), the slow cortical potential is dubbed the readiness potential (RP) or the contingent negative variation (CNV).[21–25] The RP and CNV are both widely considered to reflect motor planning and preparation for an impending action, particularly the centroparietally-distributed late component that occurs around 500 ms before the action is performed.[26–28] However, the two components are not completely identical, as the late CNV also involves activities of anticipatory attention.[29–32] A growing number of studies have demonstrated the presence of these negative-going motor preparatory activities before the initiation of covert actions, such as motor and visual imagery.[33–37] Of relevance, our recent work showed that an inner speech-specific CNV, similar to that preceding overt speech, was present prior to the production of a fully covert (inner) syllable,[38,39] providing further evidence that inner speech may be conceived as "a kind of action" that similarly engages motor preparatory processes and the forward model.[13,40]

The relationship between various slow cortical potentials and their functional interpretations is complex and remains an active area of research.[41] Recent studies have reported modality-specific *sensory* RP during the prestimulus period, where no motor or cognitive response is required and when only a sensory stimulus is expected.[42,43] These passive slow cortical potentials, which originate from the respective sensory cortical regions, appear to reflect the brain's perceptual anticipation of an upcoming stimulus.[44,45] For example, the auditory positivity (aP) component in the auditory domain, which is a frontal positive-going slow wave with sources in the bilateral auditory cortices, has been observed starting about 800 ms before an expected sound onset.[42,46] The interaction between these sensory preparatory activities and motor-related signals, particularly for covert actions, is yet to be fully understood.

Given the impaired psychomotor functions commonly observed in schizophrenia,[47–51] it is unsurprising that research has consistently reported a reduction in RP[52–55] and CNV[56–60] in people with schizophrenia in comparison to healthy controls (HC). A recent meta-analysis found a large effect for CNV blunting in people with schizophrenia comparable to that of reaction time slowing, even after considering potential moderators and methodological confounds.[56] Together, these studies provide robust evidence of dysfunctional psychomotor processes in schizophrenia with regard to overt actions. However, empirical evidence has yet to establish the critical link—proposed by the forward model—between psychomotor dysfunction in the context of inner speech (a *covert* action) and AVH.

Furthermore, it has been suggested that neural activity preceding motor actions may be the instantiation of corollary discharges; that is, the predicted sensory consequences may be embedded in the motor preparatory activity along with motor commands.[61,62] Indeed, recent studies have shown that action-outcome pairings, compared with stand-alone actions, were preceded by a larger RP,[63–65] even when the outcome was imagined.[66] If AVH arises from erroneous feedforward mechanisms of inner speech, it may be reflected in abnormalities in premovement neural indices of inner speech, during which sensorimotor predictions are formed. Consistent with this idea, reductions of neural synchrony preceding overt speech[67] and button presses[68] have been observed in schizophrenia.

The present study aimed to test the hypothesis that AVH may be related to inner speech-specific motor preparation deficits in people with schizophrenia spectrum disorders using the same paradigm as Chung et al.[38] The present study adopted a 3-group design; namely, we compared individuals with a schizophrenia spectrum disorder with current AVH (SZAVH+), those without current AVH (SZAVH-), and HC, through which the theoretical association between abnormalities in inner speech preparation and AVH may be established. We hypothesized that while the HC participants would show a typical CNV (ie, a negative slow wave) preceding inner speech production, the SZAVH+ groups would exhibit CNV blunting (ie, a diminished negativity). The SZAVH- group would allow us to assess the extent to which this blunting is specific to patients currently experiencing AVH, or it is a more general trait marker of schizophrenia.

Methods

Participants

Three groups of adult participants were recruited for the study: SZAVH+, SZAVH-, and HC. To enhance

Table 1. Demographic and Clinical Information for the Study Participants

	Hallucinators (SZAVH+)	Non-hallucinators (SZAVH-)	Healthy controls (HC)	Between-group comparisons (*p*-value)
Number of participants	58	50	49	
Age (years)	42.47 (12.60)	40.66 (10.94)	42.96 (13.06)	.613
Sex	28F, 30M	19F, 31M	24F, 25M	.461
Education	1NP, 5P, 24S, 17D/T, 5B, 1M, 5NA	1P, 24S, 13D/T, 9B, 3NA	1P, 11S, 4D/T, 18B, 8M, 7NA	<.001*
Employment	24Y, 30N, 4NA	17Y, 29N, 4NA	35Y, 8N, 6NA	<.001*
Diagnosis (SCID-I/P)	51SCZ, 5SAD, 1SFD, 1PD-NOS	37SCZ, 7SAD, 4DD, 2BPD		.075
Past history of AVH	58Y	32Y, 17N, 1NA		<.001*
History of hospitalizations	35Y, 9N, 14NA	30Y, 11N, 9NA		.489
Antipsychotic medication class	3U, 46A, 5A + T, 4NA	3U, 40A, 2T, 5NA		.137
Olanzapine equivalent (mg/day)	18.34 (11.29)[a]	12.12 (10.08)[b]		.008*
PANSS P3	4.22 (0.92)	1.24 (0.63)		<.001*
PANSS positive	18.69 (5.55)	12.98 (5.48)		<.001*
PANSS negative	12.74 (5.32)	16.50 (8.15)		.007*
PANSS general psychopathology	28.22 (7.27)	28.30 (9.50)		.963
PANSS total	59.66 (15.63)	57.78 (19.99)		.593
PSYRATS AH	23.36 (7.00)[c]	0 (0)		<.001*
PSYRATS D	11.99 (6.94)[d]	4.91 (7.05)[e]		<.001*
PSYRATS total	35.52 (11.88)[d]	4.91 (7.05)[e]		<.001*
Handedness	6L, 43R, 2A, 7NA	1L, 44R, 2A, 3NA	2L, 38R, 3A, 6NA	.331
Location	25AU, 33HK	21AU, 29HK	19AU, 30HK	.898

Mean and standard deviation are presented where appropriate. Education: NP, no primary school; P, primary school; S, secondary school; D/T, diploma or trade school; B, bachelor's degree; M, master's degree; NA, not available. Employment, Past history of AVH, History of hospitalization: Y, yes; N, no; NA, not available. Diagnosis: SCZ, schizophrenia; SAD, schizoaffective disorder; DD, delusional disorder; SFD, schizophreniform disorder; BPD, brief psychotic disorder; PD-NOS, psychotic disorder NOS. Antipsychotic medication class: U, unmedicated; A, atypical antipsychotic; T, typical antipsychotic; NA, not available. SCID, Structured Clinical Interview for DSM-IV Axis I Disorders. PANSS, Positive and Negative Syndrome Scale. PSYRATS AH, Psychotic Symptom Rating Scales Auditory Hallucinations Scale. PSYRATS D, Psychotic Symptom Rating Scales Delusions Scale. Handedness: L, left; R, right; A, ambidextrous; NA, not available. Location: AU, Australia; HK, Hong Kong. Chi-squared, *t*-test, and ANOVAs were used as appropriate, and statistically significant comparisons (*P* < .05) are indicated with asterisks*. [a]*n* = 48. [b]*n* = 40. [c]*n* = 57. [d]*n* = 55. [e]*n* = 45.

the participant sample size and boost statistical power, recruitment and testing were conducted at 2 separate locations: the Westmead Institute for Medical Research (WIMR), Sydney, Australia, and the Department of Psychology, the Chinese University of Hong Kong (CUHK), Hong Kong. Table 1 provides a detailed breakdown of the participant sample by group and testing site. Although the experimental and EEG data analysis protocols were identical across both sites, the EEG hardware used was different. Given the within-subjects design, this hardware variation was not expected to pose an issue. Nevertheless, testing site was included as a nuisance covariate in the statistical analysis to account for potential variability (see *Statistical Analysis*). The study sample was shared with another study that reported on the poststimulus event-related potentials (ERPs)[20]—specifically, the auditory N1, P2, and P3—which performed different preprocessing steps and analyses.

For the SZAVH+ and SZAVH- participants, inclusion criteria were a diagnosis of a schizophrenia spectrum disorder (88 schizophrenia, 12 schizoaffective disorder, 4 delusional disorder, 1 schizophreniform disorder, 2 brief psychotic disorder, 1 psychotic disorder not otherwise

specified) based on the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P),[69] and an age of 18 or above. The presence of AVH was defined by a score of 3 or above (from a score of 1-7) on item P3 hallucinatory behavior of the Positive and Negative Syndrome Scale (PANSS),[70] with follow-up on the Psychotic Symptoms Rating Scales (PSYRATS)[71] to confirm that the hallucinations were auditory-verbal in nature. Most participants in the SZAVH- group scored 2 or below on the PANSS P3 item; 3 participants in this group received a P3 score of 3, but follow-up revealed that the hallucinations were not in the auditory domain. HC participants had no present or past diagnosis of an Axis I disorder (based on SCID-I/P), and were age- and sex-matched to the clinical groups as best as possible. There were no differences in age and sex between the groups (see Table 1). All interviews were conducted by trained psychiatrists, clinical psychologists, or research assistants.

SZAVH+ and SZAVH- participants were referred by clinicians from collaborating hospitals in Sydney, Australia, and the New Territories East Cluster and Kowloon West Cluster, Hong Kong. HC participants were recruited by advertisements and word-of-mouth. Exclusion criteria

for all groups included a history of alcohol or substance dependence within the past year (except nicotine dependence), a history of a significant medical or neurological illness, drug-induced or organic psychosis, and intellectual disability.

In total, 58 SZAVH+, 51 SZAVH-, and 49 HC participants took part in the study. One participant was excluded from the analysis due to excessive artifacts in the EEG recording, defined as generating ≤25% of usable epochs in one or more conditions. The final sample consisted of 58 SZAVH+, 50 SZAVH-, and 49 HC participants. All participants self-reported to have normal or corrected-to-normal vision and hearing. The study was approved by the Western Sydney Local Health District Human Research Ethics Committee (AU RED HREC/13/WMEAD/423), the Joint CUHK–New Territories East Cluster Clinical Research Ethics Committee (2020.477), and the Kowloon West Cluster Research Ethics Committee (KW/EX-21-038(157-03)), and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent.

The demographic and clinical details of the study participants are summarized in Table 1. Symptom severity ratings for the SZAVH+ and SZAVH- participants were obtained by trained raters administering the PANSS[70] and PSYRATS[71] during semi-structured interviews. Handedness[72] was also assessed in all participants.
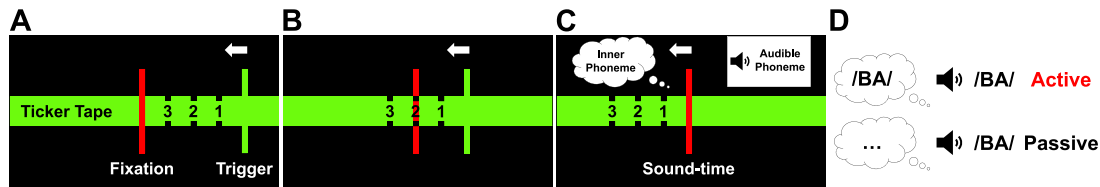
Inner Speech Paradigm

Participants completed our inner speech paradigm, summarized below and described in detail previously.[12,13,38] On each experimental trial, participants viewed a countdown animation on a computer monitor (24-inch, 1920 × 1080 pixels, 60 Hz). The animation began with a thick green horizontal line (the ticker tape) positioned centrally on the screen, a red vertical line (the fixation line) at the center of the screen, and a green vertical line (the trigger line) located on the far right, as depicted in Figure 1. Participants were instructed to maintain their gaze on the stationary fixation line throughout the trial. The ticker tape, along with the embedded trigger line, then moved leftward across the screen at a constant speed of 6.5 °/s. The ticker tape featured tick marks that crossed the fixation line 3, 2, and 1 s before reaching the trigger line. After 3.75 s, the trigger line intersected with the fixation line, a moment which was designated as the "sound-time", and continued to move beyond. At the sound-time, an *audible syllable* /BA/ or /BI/ was delivered to the participants' headphones (Sennheiser HD201 in Australia and Focusrite Scarlett HP60 MKII in Hong Kong), produced by a male speaker, lasting about 200 ms and with an intensity of 70 decibels sound pressure level (dB SPL). Each audible syllable (ie, /BA/ and /BI/) was presented in 50% of the trials within each trial block in a randomized order. Consequently, the specific syllable heard at the sound-time was unpredictable.

The experiment consisted of 12 blocks, each containing 24 trials. In two-thirds of these blocks, participants were instructed to silently articulate an *inner syllable* /BA/ or /BI/ at the sound-time by imagining themselves physically producing the syllable, engaging their articulatory organs (ie, mouth, tongue, larynx, etc.) without any actual movement—the *Active* condition. They were always asked to imagine the same inner syllable throughout a block. In the remaining third of the blocks, participants were required only to listen to the syllable without imagining anything specific—the *Passive* condition (see Figure 1D). Each of the 3 block types (imagine /BA/ blocks, imagine /BI/ blocks, passive blocks) was presented once in a randomized sequence before any block type was repeated, resulting in 4 repetitions of each block type. Stimulus presentation was controlled by custom MATLAB (MathWorks) scripts using the Psychophysics Toolbox extensions.[73,74]

After each trial, participants rated their subjective performance for that trial on a 5-point Likert scale. Specifically, participants were given the following instructions for the "imagine /BA/" trials: *"After each trial, you will be asked to rate how well you were able to imagine yourself producing the sound 'BA' on a scale of 1 (didn't imagine 'BA' at all) to 5 (imagined 'BA' perfectly)"*. For the "imagine /BI/" trials, the instructions were identical except that "BA" was replaced with "BI". For the passive trials, participants were asked: *"After each trial, you will be asked to rate how well you were able to listen to the sound, on a scale of 1 (didn't listen to it at all) to 5 (listened to it perfectly)"*. The "imagine /BA/" and "imagine /BI/" trials were combined to form the Active trials. In the Active condition, there was an average score of 4.34 (SD = 0.69) in SZAVH+, 4.38 (SD = 0.66) in SZAVH-, and 4.58 (SD = 0.55) in HC. In the Passive condition, there was an average score of 4.44 (SD = 0.65) in SZAVH+, 4.55 (SD = 0.62) in SZAVH-, and 4.48 (SD = 1.11) in HC. A two-way ANOVA (controlling for *Location*) revealed no significant main effect of *Group* on the trial ratings for the Active condition, $F(2,151) = 1.808$, $P = .168$, $\omega^2_p = 0.010$. Similarly, a two-way ANOVA (controlling for *Location*) revealed no significant main effect of *Group* on the trial ratings for the Passive condition, $F(2,151) = 0.500$, $P = .607$, $\omega^2_p < 0.001$.

EEG Acquisition and Preprocessing

Electroencephalographic data were acquired from 64 Ag/AgCl electrodes positioned according to the extended 10-20 system using a Biosemi ActiveTwo system (www.biosemi.com), referenced online to CMS/DRL for participants in Australia. A 64-channel ANT EEGO system (www.ant-neuro.com/), referenced online to CPz, was used for participants in Hong Kong. In both sites, electro-oculogram (EOG) data was measured by placing electrodes on the outer canthi of both eyes, and above and below the left eye, to reflect horizontal (HEOG) and

Figure 1. A schematic of the experimental protocol. A. Participants were asked to maintain their gaze on the fixation line positioned centrally on the screen. B. After a delay of 1-2 s, the trigger line began moving leftward across the screen at a constant speed of 6.5°/s, starting from the far-right edge. C. After 3.75 s, the trigger line aligned with the fixation line—a moment referred to as the "sound-time". At this precise moment, 2 simultaneous events occurred. D. In the active trials, participants were instructed to silently articulate a predefined syllable (either /BA/ or /BI/) in their heads. In the passive trials, participants were instructed to passively listen to the audible syllable. At the same time, an audible syllable (either /BA/ or /BI/), produced by a male speaker at a volume of 70 dB SPL, was delivered to the participants' headphones. Each audible syllable (/BA/ and /BI/) was presented in 50% of the trials for each trial block in a randomized order. After the sound-time, the trigger line continued its trajectory for an additional 1 s, marking the end of the trial

vertical (VEOG) eye movements, respectively, as well as blinks. Additional electrodes were placed on the bridge of the nose above the face mask (consistent with local COVID-19 masking policies), and on the left and right mastoid. The sampling rate was 2048 Hz for both systems.

The data were pre-processed and analyzed using BrainVision Analyzer (Brain Products GmbH). The EEG data were re-referenced offline to the average of the mastoid electrodes. Data were first notch filtered at 50 Hz, and then band-pass filtered from 0.1 to 30 Hz using a phase-shift free Butterworth filter (12 dB/Oct slope) (see Supplementary Material for the results obtained when using a 1 Hz high-pass filter). Spherical spline interpolation was applied to bad channels.[75] Epochs were extracted from -2500 to 100 ms, relative to the audible syllable onset, and baseline-corrected to the mean voltage from -2500 to -2000 ms. All epochs were corrected for eye-movement artifacts, using the technique described in Gratton et al.[76] and any epochs with signals exceeding peak-to-peak amplitudes of 200 μV for any channel was excluded.

In the Active condition, there were an average of 162.91 (SD = 38.87) useable epochs in SZAVH+, 156.32 (SD = 43.89) useable epochs in SZAVH-, and 179.31 (SD = 30.13) useable epochs in HC. In the Passive condition, there were an average of 82.24 (SD = 19.06) useable epochs in SZAVH+, 79.88 (SD = 19.94) useable epochs in SZAVH-, and 88.18 (SD = 16.25) useable epochs in HC. A two-way ANOVA (controlling for *Location*) revealed a significant main effect of *Group* on the number of useable epochs in the Active condition, $F(2,151) = 5.189$, $P = .007$, $\omega^2_p = 0.051$. Post-hoc comparisons showed that the number of usable epochs in HC was higher than in SZAVH-, $t(151) = 3.156$, $P_{holm} = .006$, $d = 0.647$, 95% CI [8.832, 38.397]. However, there was no difference between HC and SZAVH+, $t(151) = 2.221$, $P_{holm} = .056$, $d = 0.439$, 95% CI [1.771, 30.279], and between SZAVH+ and SZAVH-, $t(151) = 1.065$, $P_{holm} = .288$, $d = 0.208$, 95% CI [-6.487, 21.666]. In contrast, a two-way ANOVA (controlling for *Location*) revealed no significant main effect of *Group* on the number of useable epochs in the Passive condition, $F(2,151) = 2.682$, $P = .072$, $\omega^2_p = 0.021$.

The amplitude of the late CNV, observed before S2 (the "sound-time" in this case) in a traditional S1-S2 (ie, a warning stimulus followed by an imperative stimulus) design of CNV studies,[31,32] was the primary dependent variable. The late CNV was defined as the mean amplitude in the 500-ms time window preceding the sound onset. This time window was selected based on visual inspection of the ERPs and voltage maps, and to be consistent with our previous study.[38] While speech preparatory activity was found to be maximal at electrode Cz,[77,78] analyses were conducted collapsing across neighboring midline electrodes FCz and CPz to improve reliability. The results remained unchanged when analyses were restricted to electrode Cz. The late CNV amplitudes in the Active ("Active CNV") and Passive ("Passive CNV") conditions were examined for each group.

Voltage and current source density (CSD) maps were used to show scalp topography. CSD is a surface Laplacian technique that uses a spherical spline algorithm to compute estimates of radial current flow leaving (sinks; positive values) and entering (sources; negative values) the scalp based on scalp-recorded potentials.[79] These reference-free estimates provide a clearer mapping of the direction, location and intensity of current generators that underlie an ERP topography, reducing the negative impact of volume conduction.

Statistical Analysis

Data visualization was performed using JASP[80] and ERPLAB.[81] The late CNVs were analyzed with a mixed ANOVA using JASP 0.95.0.0.[82] The between-subjects factor was *Group* (3 levels: SZAVH+, SZAVH-, and HC). The within-subjects factor was *Condition* (2 levels: Active and Passive). The between-subjects factor *Location* (2 levels: Australia and Hong Kong) was also entered as a nuisance covariate. Partial omega squared was reported, which is considered a less biased estimate of the effect size.[83] In the case of a significant *Condition* × *Group* interaction, post-hoc pairwise comparisons conditional on each of the interaction terms using estimated marginal means were used to unpack the simple main effects. One-sample *t*-tests (two-tailed) were performed for the Active

CNV and Passive CNV in each group to test whether their amplitudes were significantly different from zero. The Holm-Bonferroni method was applied to correct for multiple comparison testing.

Within the patient groups, given the violation of normality, an exploratory Kendall's tau-b partial correlation, controlling for Passive CNV, was conducted to examine the relationship between Active CNV and severity of AVH as measured by the PSYRATS Auditory Hallucinations Scale (PSYRATS AH). Controlling for Passive CNV by including it as a covariate removes shared variance between the 2 experimental conditions, isolating the unique contribution of inner speech-related motor readiness in the Active CNV to AVH severity, independent of baseline neural activity. The relationships between Active CNV (after controlling for Passive CNV) and the following symptom severity measures were also examined: delusions (PSYRATS Delusions Scale; PSYRATS D), positive symptoms (PANSS Positive Subscale; PANSS positive), negative symptoms (PANSS Negative Subscale; PANSS negative), and general psychopathology (PANSS General Psychopathology Subscale; PANSS general psychopathology).

To complement these findings and assess the predictive role of motor readiness, 3 binomial logistic regression models were conducted with AVH status (presence of hallucinations: 1 = yes; 0 = no) as the dependent variable. The first model included Active CNV as the sole predictor, while the second model included Passive CNV as a covariate. PSYRATS D and PANSS negative were added as covariates in the third model.

Results

[Figure 2](#) shows the results of the late CNV analyses for the 2 experimental conditions (Active in red, Passive in black), for the HC group ([Figure 2(i)](#)), the SZAVH+ group ([Figure 2(ii)](#)) and the SZAVH- group ([Figure 2(iii)](#)). All results were averaged across electrodes FCz, Cz, and CPz, which were the 3 electrodes at which the inner speech-specific late CNV was previously found to be maximal.[38] In these figures, Subpanel A shows the grand average prestimulus slow wave waveforms for the 2 conditions; Subpanel B shows the voltage and CSD maps for the 2 conditions; Subpanel C shows the raincloud plots of each participant's mean late CNV amplitude for the 2 conditions. The figures revealed distinctive topographic distributions and morphological features between the 3 groups, with medial-central negative-going slow potentials in the HC group, and medial-frontal positive-going slow potentials in the SZAVH+ and SZAVH- groups.

An omnibus ANOVA revealed a significant main effect of *Condition*, $F(1,151) = 11.384$, $P < .001$, $\omega^2_p = 0.008$, and a significant *Condition × Group* interaction, $F(2,151) = 4.3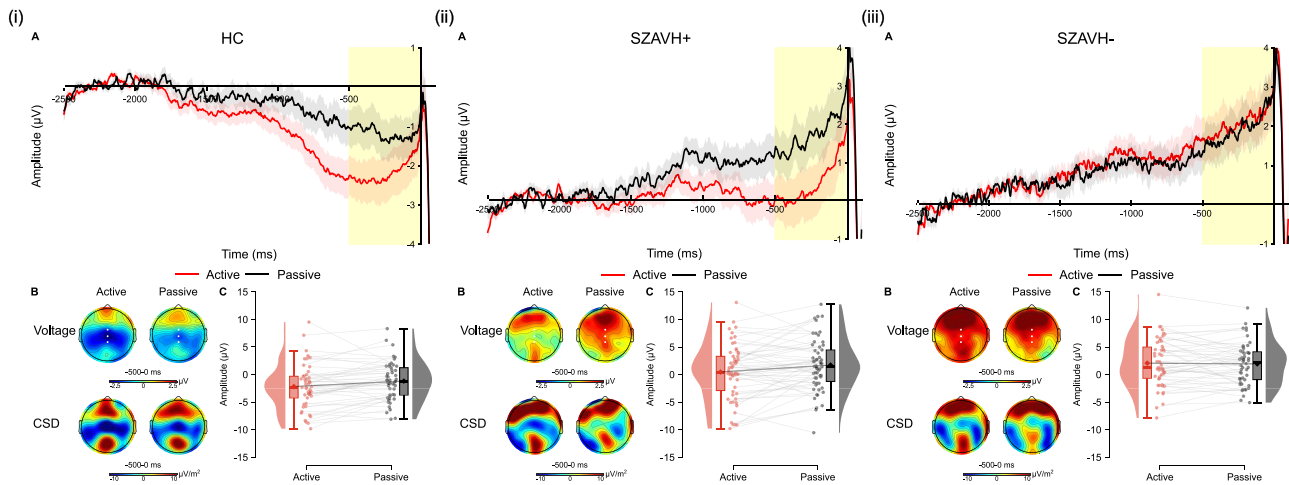84$, $P = .014$, $\omega^2_p = 0.005$, after controlling for *Location*, which indicates that the pattern of the late CNV amplitude across conditions differed between the 3 groups. In the Supplementary Material, we reported the effect of *Location*, *Age*, and *Sex* on the statistical analysis.

The Late CNV: Within-Group Comparisons

For the HC group, a post-hoc comparison (controlling for *Location*) showed that the late CNV amplitude was significantly more negative in the *Active* condition than the *Passive* condition, $t(151) = 2.620$, $P_{holm} = .019$, $d = 0.262$, 95% CI [-1.935, -0.271]. One-sample *t*-tests revealed that the late CNV amplitude was significantly more negative than the baseline in both the *Active* condition, $t(48) = 3.765$, $P_{holm} = .001$, $d = 0.538$, 95% CI [-3.331, -1.012], and in the *Passive* condition, $t(48) = 2.356$, $P_{holm} = .023$, $d = 0.337$, 95% CI [-2.195, -0.174]. The *Condition × Location* interaction was not significant, $F(1,47) = 2.156$, $P = .149$, $\omega^2_p = 0.002$, and neither was the main effect of *Location*, $F(1,47) = 0.974$, $P = .329$, $\omega^2_p < 0.001$.

For the SZAVH+ group, a post-hoc comparison (controlling for *Location*) showed that the late CNV amplitude was significantly less positive in the *Active* condition than the *Passive* condition, $t(151) = 3.754$, $P_{holm} < .001$, $d = 0.339$, 95% CI [-2.182, -0.677]. One-sample *t*-tests revealed that the late CNV amplitude was significantly more positive than the baseline in the *Passive* condition, $t(57) = 2.831$, $P_{holm} = .013$, $d = 0.372$, 95% CI [0.506, 2.951], but not in the *Active* condition, $t(57) = 0.707$, $P_{holm} = .483$, $d = 0.093$, 95% CI [-0.850, 1.778]. There was a significant *Condition × Location* interaction, $F(1,56) = 7.484$, $P = .008$, $\omega^2_p = 0.013$, which was driven by the late CNV amplitude being less positive in the *Active* compared with *Passive* conditions for participants in Australia, $t(56) = 3.981$, $P_{holm} < .001$, $d = 0.543$, 95% CI [-3.947, -1.304], but not for participants in Hong Kong, $t(56) = 0.407$, $P_{holm} = .686$, $d = 0.048$, 95% CI [-1.384, 0.916].

For the SZAVH- group, a post-hoc comparison (controlling for *Location*) showed no significant difference between the *Active* and *Passive* conditions, $t(151) = 0.406$, $P_{holm} = .686$, $d = 0.040$, 95% CI [-0.646, 0.980]. One-sample *t*-tests revealed that the late CNV amplitude was significantly more positive than the baseline in both the *Active* condition, $t(49) = 3.362$, $P_{holm} = .003$, $d = 0.475$, 95% CI [0.834, 3.314], and the *Passive* condition, $t(49) = 3.569$, $P_{holm} = .002$, $d = 0.505$, 95% CI [0.847, 3.031]. Within the SZAVH- group, a repeated measures ANOVA (controlling for *Location*) comparing those with ($n = 32$) and without ($n = 17$) a past history of AVH did not find a significant main effect of *Condition*, $F(1,45) = 0.008$, $P = .927$, $\omega^2_p < 0.001$, a main effect of *AVH-History*, $F(1,45) = 0.189$, $P = .666$, $\omega^2_p < 0.001$, or a *Condition × AVH-History* interaction, $F(1,45) = 1.589$, $P = .214$, $\omega^2_p = 0.002$, which suggest that past AVH experience did not affect the pattern of

Figure 2. Analysis of the prestimulus cortical potentials for the active and passive conditions, for the 3 participant groups (HC, SZAVH+, SZAVH-). **Panel (i)** shows the waveform data for the HC group ($n = 49$), **panel (ii)** shows the waveform data for the SZAVH+ group ($n = 58$), and **panel (iii)** shows the waveform data for the SZAVH- group ($n = 50$). There are 3 subpanels within panels (i)-(iii). **Subpanel A.** Waveforms showing the event-related potentials during the pre-stimulus period averaged across electrodes FCz, Cz, and CPz in the active condition (red line) and passive condition (black line). The shaded areas represent the standard error of the mean. The yellow bar shows the late CNV time window (-500 to 0 ms). The waveforms are shown collapsed across trials with audible syllable /BA/ and /BI/ at stimulus onset (0 ms), and the waveform for the active condition is shown collapsed across inner syllable /BA/ and /BI/ during the inner speech task. **Subpanel B.** Voltage and current source density (CSD) maps are plotted separately for each condition; white dots illustrate the electrodes used in the analysis. **Subpanel C.** Raincloud plots for each condition showing half a violin (density) and a boxplot with the horizontal stripe representing the median and the diamond shape representing the mean amplitude of the late CNV; each dot represents 1 participant's mean amplitude averaged across trials
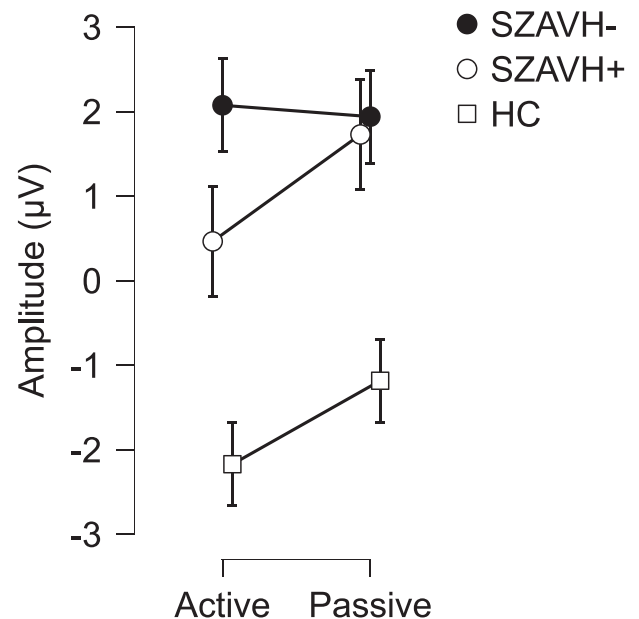
results in SZAVH- patients. There was a main effect of *Location*, F(1,48) = 9.565, $P = .003$, $\omega^2_p = 0.146$, which was driven by the participants in Australia having a more positive late CNV amplitude compared with participants in Hong Kong.

An exploratory repeated measures ANOVA (controlling for *Location*) parsing the Active condition by syllables (ie, imagining /BA/ trials and imagining /BI/ trials) did not find a significant main effect of *Syllable*, $F(1,150) = 1.800$, $P = .182$, $\omega^2_p = 0.001$, or a *Syllable × Group* interaction, $F(2,150) = 1.602$, $P = .205$, $\omega^2_p = 0.001$, which suggest that there was no difference in inner speech-related motor readiness based on linguistic and phonetic processing.
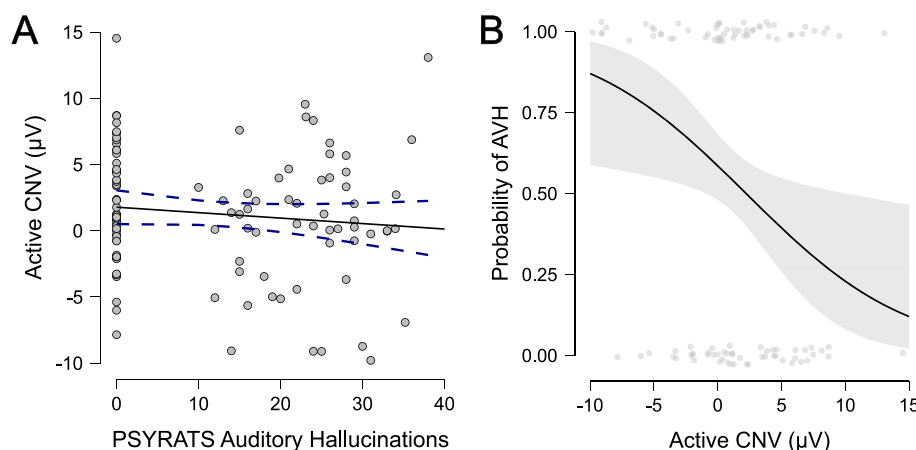
The Late CNV: Between-Group Comparisons

Figure 3 shows the mean late CNV amplitudes for the 2 conditions in each participant group. In the *Active* condition, post-hoc comparisons showed that the late CNV amplitude in HC was more negative than in SZAVH+, $t(151) = 3.124$, $P_{holm} = .004$, $d = 0.648$, 95% CI [-4.459, -1.004], and SZAVH-, $t(151) = 5.173$, $P_{holm} < .001$, $d = 1.112$, 95% CI [-6.481, -2.898]. Additionally, the amplitude in SZAVH+ was less positive than in SZAVH-, $t(151) = 2.268$, $P_{holm} = .025$, $d = 0.464$, 95% CI [-3.664, -0.252].

In the *Passive* condition, post-hoc comparisons showed that the late CNV amplitude in HC was more negative than in SZAVH+, $t(151) = 3.867$, $P_{holm} < .001$, $d = 0.725$, 95% CI [-4.620, -1.496], and SZAVH-, $t(151) = 4.170$,

Figure 3. Descriptive plot summarizing the late CNV amplitude data. Mean amplitude of the late CNV for the active and passive conditions, plotted separately for the SZAVH- (empty circles), SZAVH+ (filled circles), and HC (empty squares) groups. The error bars represent the normalized 95% CIs for the within-subjects effect based on Morey[84]

$P_{holm} < .001$, $d = 0.811$, 95% CI [-5.040, -1.799]. However, there was no difference between SZAVH+ and SZAVH-, $t(151) = 0.463$, $P_{holm} = .644$, $d = 0.086$, 95% CI [-1.905, 1.181].

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Figure 4. Results of correlation and logistic regression analysis between the active CNV and severity/presence of AVH in the patient groups. Both plots showing the results of participants in the SZAVH+ and SZAVH- groups only, after controlling for the amplitude of the passive CNV. **A.** A scatterplot showing the correlation between amplitude of the active CNV and total score on the PSYRATS auditory hallucinations scale. The solid line represents the line of best fit, while the dotted lines represent the 95% CI. **B.** An estimate plot showing the logistic regression curve between amplitude of the active CNV and probability of AVH, where 1 represents presence of AVH and 0 represents absence of AVH. The shaded area represents the 95% CI

Correlation with AVH Severity and Predicting AVH Status

Within the patient groups, a Kendall's tau-b partial correlation, controlling for Passive CNV, revealed no significant relationship between Active CNV and *PSYRATS AH*, $\tau b_{partial}(107) = -0.088$, $P = .181$, indicating no monotonic association between the inner speech-related motor readiness and severity of AVH (see Fig. 4A). Similarly, there was no significant relationship between Active CNV and the following clinical variables: *PSYRATS D*, $\tau b_{partial}(100) = -0.013$, $P = .853$, *PANSS positive*, $\tau b_{partial}(108) = -0.035$, $P = .596$, *PANSS negative*, $\tau b_{partial}(108) = 0.080$, $P = .224$, and *PANSS general psychopathology*, $\tau b_{partial}(108) = 0.072$, $P = .270$.

The logistic regression model, with AVH status (yes/no) as the dependent variable and Active CNV as the predictor, was not significant, $\chi^2(1) = 3.149$, $P = .076$, Nagelkerke's $R^2 = 0.038$. However, after including Passive CNV as a covariate, the overall model approached significance, $\chi^2(2) = 5.881$, $P = .053$, Nagelkerke's $R^2 = 0.071$, which indicates an improved model fit over the null model. As shown in Fig. 4B, Active CNV was a significant predictor of AVH status, $B = -0.156$, SE = 0.068, Wald = 5.313, $P = .021$, OR = 0.856, 95% CI [-0.288, -0.023], suggesting that for every unit decrease in Active CNV (ie, more negative amplitude), the odds of AVH presence increased by 14% (1 - 0.86 = 0.14). In contrast, Passive CNV was not a significant predictor, $B = 0.118$, SE = 0.073, Wald = 2.607, $P = .106$, OR = 1.125, 95% CI [-0.025, 0.261]. The inclusion of the HC group in the sample returned a non-significant result for the Active CNV, $B = -0.083$, SE = 0.059, Wald = 1.995, $P = .158$, OR = 0.921, 95% CI [-0.198, 0.032].

The addition of *PSYRATS D* and *PANSS negative* to the model as covariates among patients significantly improved model fit, $\chi^2(4) = 37.560$, $P < .001$, Nagelkerke's $R^2 = 0.419$. Importantly, Active CNV remained a significant predictor, $B = -0.179$, SE = 0.087, Wald = 4.220, $P = .040$, OR = 0.836, 95% CI [-0.350, -0.008], indicating that Active CNV independently predicted AVH status after controlling for the effects of Passive CNV, delusions, and negative symptoms.

With regards to the potential impact of antipsychotic medications on the inner speech-related motor readiness in the patient groups: Kendall's tau-b partial correlation between Active CNV and olanzapine-equivalent medication dosage, controlling for Passive CNV, was non-significant, $\tau b_{partial}(88) = -0.085$, $P = .245$. Similarly, there was no significant correlation between olanzapine-equivalent medication dosage and the amplitude of Active CNV, $\tau b(88) = -0.004$, $P = .960$, or Passive CNV, $\tau b(88) = 0.114$, $P = .116$.

Discussion

The key results can be summarized as follows: the HC participants exhibited the expected inner speech-specific motor preparatory activity preceding the production of inner speech; that is, they exhibited a more negative late CNV amplitude when they were preparing to produce an inner syllable, when compared with sitting passively, replicating the results of our previous study.[38,39] In contrast, patients with schizophrenia spectrum disorders did not exhibit this normal pattern of motor preparatory activity before inner speech production. Contrary to our hypothesis that patients with current auditory verbal hallucinations (SZAVH+) would show greater blunting of the late CNV compared with those without hallucinations (SZAVH-), both groups exhibited positive-going slow cortical potentials in both the Active

and Passive conditions. Unexpectedly, in SZAVH+ patients, the amplitude in the Active condition was significantly less positive than in the Passive condition, suggesting some degree of preserved inner speech-related motor preparatory activity, similar to the pattern in HC but with reversed polarity. In contrast, SZAVH- patients showed no difference between the conditions. Moreover, more negative Active CNV was associated with the presence of AVH in patients, after controlling for the severity of delusions and negative symptoms. Importantly, the 2 patient groups were matched in the severity of illness as estimated by the PANSS total score and history of hospitalizations. Taken together, these results provide evidence that, beyond overt actions, patients with schizophrenia spectrum disorders show abnormalities in motor preparatory mechanisms associated with inner speech production, and that a distinct pattern of such deficits may be related to AVH characteristic of this class of disorders.

A novel finding in this study is the prestimulus frontal positivity observed in the patient groups, which distinctly differs from traditional action-preparatory negativities like the RP and CNV in both polarity and scalp topography. Given its morphology, scalp topography, and identified cortical sources (notably, the involvement of the bilateral temporal cortices evident in the CSD maps in Figure 2(ii) and (iii), comparable to previous studies[42,44]), we propose this positive-going slow wave reflects a sensory RP—specifically, the aP. Crucially, the aP is typically masked at the vertex (near electrode Cz) by the stronger negative-going RP or CNV during movement-related tasks in healthy participants,[44,85] which is consistent with the results observed in our HC group. Consequently, we speculate that the prominent aP observed in the patient groups, even in the inner speech condition, may in fact be a consequence of a markedly blunted late CNV, allowing the aP to "break through". By contrast, previous research on motor preparation in schizophrenia has focused on overt actions (eg, overt speech), where the CNV is inherently larger than for inner speech,[38] such that even a blunted CNV in patients may still be sufficiently large to prevent "breakthrough" of the aP. That is, the pattern observed here for inner speech may have been masked by the focus on overt action in prior research. Alternatively, the inverted polarity may simply represent a general disease marker, a possibility that warrants further investigation.

Action-outcome prediction is thought to be embedded in the premovement activity,[61] which provides a unique window to probe the forward model *prior to* the fulfillment of sensorimotor prediction. Consistent with reports of a blunted RP in patients with schizophrenia during button-press-for-beep tasks,[61,86] our finding of a blunted late CNV in the inner speech condition in both patient groups demonstrates that deficits in action-outcome prediction may similarly occur during inner speech as early as

the motor preparation stage, providing empirical evidence to the long-held hypothesis of impaired corollary discharges during inner speech in schizophrenia. Specifically, these weak predictions may cause the normally attenuated inner speech-related sensations to attain salience as if they were externally generated, leading to misattribution to an external source and ultimately manifesting as AVH.[40] Indeed, weaker lateralized RP has been associated with reduced cortical suppression to self-generated tones.[61]

The observed blunting of CNV in patients with schizophrenia spectrum disorders, regardless of AVH status, suggests that impaired motor preparation of inner speech may be a trait marker of these disorders, rather than specific to AVH. Crucially, however, a distinct pattern emerged within the patient groups: SZAVH+ exhibited a more negative Active CNV compared with Passive CNV, indicating heightened motor preparatory activity in the inner speech condition, as evidenced by medial-central activation resembling the RP in the CSD map (see Figure 2(ii)). This specific pattern was absent in SZAVH-. Furthermore, statistical analyses isolating this inner speech-related motor readiness in the Active CNV within patients revealed it uniquely predicted the presence of AVH, with greater negativity associated with a higher likelihood of AVH. These findings thus raise the intriguing possibility that SZAVH+ may generate not only *weak* sensorimotor predictions, which is similarly implicated in SZAVH-, but also *noisy*, imprecise predictions, potentially leading to increased prediction errors within the monitoring system.[87] Consistent with this idea, a recent study[88] found that while SZAVH- and HC showed enhanced cortical responses to target syllables during speech preparation, SZAVH+ exhibited enhancement to non-target syllables, indicating imprecise corollary discharge that erroneously sensitizes neighboring auditory units around the sensory target of the motor act. It is possible that these distinct abnormalities during prediction formation may lead to different sensory processing outcomes to inner speech in hallucinating and non-hallucinating patients, as recently implicated.[20]

It is important to consider the current findings in light of another influential account of hallucinations, namely the "strong priors" theory, which posits that hallucinations arise when expectations are given undue weighting, overriding contradictory sensory evidence and ultimately causing the brain to perceive non-existent stimuli.[89,90] This apparent paradox—of weak and strong priors simultaneously contributing to hallucinations—might be reconciled if they are considered to work in tandem within an integrated framework, with suggestions pointing to operations at different hierarchical levels[87] or in parallel dual-hierarchies.[91] For example, Leptourgos and Corlett's account,[91] which has some empirical support,[92] suggests that failure of corollary discharge in the ego-centric system (leading to diminished *feeling* of agency) results in strong prediction error signals flooding the allo-centric

system. This system, responsible for *judgment* of agency, then receives overwhelming bottom-up signals, causing it to compensate by increasing the precision of its high-level priors, which subsequently results in the experience of hallucinations and their attribution to external agents. Extending this model, our results further suggest that low-level prediction errors, perhaps due to noisy and imprecise prediction during inner speech preparation (ie, as observed in SZAVH+ but not SZAVH-), might need to be substantial *enough* to drive this compensatory enhancement of priors, and thereby may potentially serve as a state marker of AVH.

Given the covert nature of the inner speech task, we acknowledge that an objective measure of participant collaboration is not readily available. However, we have several lines of evidence that make non-cooperation an unlikely explanation for the smaller CNV in the patient groups. Firstly, participants across all groups reported high subjective performance ratings for both the Active and Passive conditions, suggesting overall engagement with the task. Secondly, if participants, particularly those in the patient groups, were simply not cooperating, we would expect no systematic difference between the Active and Passive conditions within these groups (as they would just be sitting passively), and there is no reason to believe SZAVH+ and SZAVH- patients would approach the tasks differently. Therefore, the fact that the SZAVH+ group showed a less positive slow wave in the inner speech condition compared with the SZAVH- group argues against a general lack of cooperation as the underlying cause of our results.

Our findings highlight the value of using a subgroup symptom-focused approach over the traditional "correlational" approach to investigate neural mechanisms that underlie different aspects of deficits in psychotic disorders,[40,93] with the latter often failing to identify relationships between degree of neurophysiological abnormalities and symptom severity.[94,95] Indeed, we did not find a correlation between the inner speech-related motor readiness and a multidimensional measure of AVH severity. Interestingly, though, it uniquely predicted the presence or absence of AVH, suggesting that it may function as a marker of AVH status rather than severity; that is, it is possible that neural markers may not necessarily correlate linearly with symptom severity, but reflect underlying neural disruptions specific to the presence of the symptom—AVH in our case. Understanding the extent to which the inner speech theory and its associated neural mechanisms can account for different AVH subtypes,[96] as well as how the (in)ability of people to experience inner speech could relate to AVH experiences,[97,98] would be a worthwhile pursuit of future research.

Finally, our results relating to the CNV in the Passive condition are noteworthy. The CNV is thought to contain both motoric and nonmotoric components (see Chung et al.[38] for a more detailed discussion).[21] Specifically, the stimulus preceding negativity (SPN) is a nonmotor anticipatory slow potential that occurs prior to stimuli that carry motivationally important information,[99] and it has been suggested that the SPN sums with the RP to produce the CNV.[32,100] Therefore, while there was no preparation to an act in the passive condition, the visual countdown leading to a task-relevant auditory probe was sufficient to generate a negative-going slow wave in healthy participants as previously demonstrated,[38] providing us with a unique window to evaluate the nonmotoric anticipatory processes in schizophrenia. Consistent with previous findings,[101] we found that the Passive CNV was significantly diminished in the patient groups compared with HC, potentially leading to the breakthrough of the aP similar to that observed in the Active condition. However, no difference was found between the SZAVH+ and SZAVH- groups, indicating that disturbances in the underlying anticipatory processes may reflect a broader characteristic of schizophrenia spectrum disorders, rather than one specifically tied to the experience of AVH. Together, our results showed that disrupted motoric and nonmotoric expectation-related processes may reflect distinct aspects of disturbances in schizophrenia.

In conclusion, the present study revealed abnormalities in the late CNV preceding the production of inner speech in individuals with schizophrenia spectrum disorders. Both hallucinating and non-hallucinating patients exhibited markedly blunted motoric CNV during inner speech preparation compared with HC, which in turn led to the unmasking of a sensory RP, indicating dysfunctions in the motor preparatory mechanisms specific to inner speech, and weak sensorimotor predictions. Crucially, the presence of an inner speech-related motor readiness unique to patients with current AVH may reflect the instantiation of noisy and imprecise corollary discharges in the hallucinating brain. As such, our findings provide empirical data in support of a mechanism that has long been proposed to be implicated in the formation of AVH, and which lies at the heart of the atypical inner speech monitoring theory of AVH. Overall, our findings suggest that abnormalities in the formation of corollary discharges may be reflected in neural alterations as early as during motor planning and preparation, underpinning the potential of using motor preparatory indices like the CNV and RP as neurophysiological biomarkers for the identification and monitoring of covert symptoms.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin.

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Conflicts of Interest

Dr Harris has received consultancy fees from Boehringer Ingelheim. He is a director of Mind Australia, a leading non-government organisation. All other authors declare no competing interests.

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