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Mind and body: Psychophysiological profiles of instructional and motivational self-talk

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Abstract

Self-talk is a psychological skill that benefits motor performance by controlling and organizing performers' thoughts. While the behavioral effects of self-talk are clear, research on the mechanisms underpinning the effects of different modes of self-talk is sparse. To address this issue, we propose and test a psychophysiological model of the effects of self-talk on motor performance. Forty golf novices practiced a golf putting task while using either instructional or motivational self-talk preceding each putt. We measured performance (radial error), technique (club kinematics and muscle activity), cardiac activity (heart-rate and event-related heart-rate change), as well as electroencephalographic alpha power and connectivity in a randomized (group: instructional self-talk, motivational self-talk) experimental design. Instructional self-talk promoted superior technique and was associated with greater parietal alpha power and weaker connectivity between frontal and parietal electrodes and all other scalp sites, possibly indicative of increased top-down control of action. These findings provide initial evidence for an information-processing mechanism underlying the benefits of instructional self-talk. They also cast doubt on the validity of left-frontotemporal connectivity as a measure of verbal-analytic processing during motor tasks. Motivational self-talk led to increased heart-rate and reduced event-related heart rate variability, suggesting an effort-based mechanism to explain the benefits of motivational self-talk. Our study represents the most complete multi-measure investigation of self-talk to date. We hope that our psychophysiological model of self-talk will encourage researchers to move beyond the exclusive reliance on behavioral and self-report measures to discover the mechanisms underlying the benefits of self-talk for performance.

KEYWORDS

conscious processing, frontoparietal network, high-alpha, Method < EEG, motor performance, verbal processing

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1 | INTRODUCTION

Our stream of thoughts can be accompanied by covert verbalizations known as self-talk. Self-talk acts as an accelerator to thinking and understanding (Vygotsky, 1978s), and is especially prevalent during the acquisition of motor skills (Hardy, Gammage, & Hall, 2001; Masters, Polman, & Hammond, 1993). It is common for performers to recite *instructional self-talk* to guide the steps for successful skill execution (Hardy, Comoutos, & Hatzigeorgiadis, 2018). In addition, to regulate arousal, support confidence, and motivational drive, individuals may also verbalize a series of self-motivating statements (*motivational self-talk*; Hardy et al., 2018). Meta-analytic evidence indicates that both instructional and motivational self-talk benefit performance (Hatzigeorgiadis, Zourbanos, Galanis, & Theodorakis, 2011). However, for motor tasks placing a premium on precision, instructional self-talk seems to have a relative advantage over motivational self-talk (Theodorakis, Weinberg, Natsis, Douma, & Kazakas, 2000), as shown by behavioral metadata revealing larger performance benefits ($d_{\text{instructional}} = .83$ vs. $d_{\text{motivational}} = .22$; Hatzigeorgiadis et al., 2011) and more consistent movement kinematics (Abdoli, Hardy, Riyahi, & Farsani, 2018).

While the effects of self-talk on motor performance and skill execution are well understood at the behavioral level, the self-talk literature lacks both data and a guiding model highlighting detailed multifaceted mechanistic pathways, such as neurophysiological adaptations that explain the processes beyond performance-related markers of skill outcome (e.g., missed or holed golf putts). To address this shortcoming, we present and test the first psychophysiological model of self-talk and motor skill execution. This model is grounded on a unique integration of both self-talk and psychophysiological literatures. For instance, we draw from Hardy, Tod, and Oliver's (2009) self-talk framework highlighting the broad cognitive (e.g., information processing), motivational (e.g., increased effort), behavioral (e.g., superior technical kinematics/form), and affective (e.g., anxiety control) mediatory pathways. Precise psychophysiological predictions of our model are based on evidence from investigations of related cognitive constructs, such as conscious motor processing (e.g., Masters & Maxwell, 2008) and motivation (e.g., Harmon-Jones, Gable, Peterson, 2010). Given their different effects on performance, we propose that instructional and motivational self-talk have a different neurophysiological basis and, therefore, influence the motor skill control via different psychophysiological mechanisms.

1.1 | Instructional self-talk

In guiding the steps for successful skill execution by steering individuals' attention to the correct stimuli at appropriate

moments (Theodorakis et al., 2000), instructional self-talk represents a conscious top-down process acting on the motor system (Hardy, 2006). Operationally, this appears comparable to the conscious processing of movement-related instructions (Mullen & Hardy, 2010). Indeed, like instructional self-talk, training regimes fostering the conscious processing of movements can produce faster skill acquisition at the initial stages of motor learning, compared to less explicit forms of training (e.g., Hardy, Mullen, & Jones, 1996; Masters, 1992). Therefore, we hypothesize that compared to motivational self-talk the use of instructional self-talk will, at the initial stages of learning, result in better technique. In the case of golf putting, this could manifest as greater forearm muscle activity at and immediately after the putter-ball impact to reflect acceleration through the swing, and reduced lateral clubhead acceleration to reflect reduced risk of putts being pushed or pulled wide of the hole (Cooke, Kavussanu, McIntyre, & Ring, 2010). These variables distinguish experts from novices (Cooke et al., 2014). Accordingly, we expect instructional self-talk will promote greater forearm muscular activation around impact and reduced lateral clubhead acceleration compared to motivational self-talk.

Like conscious motor processing, instructional self-talk may also provoke distinct patterns of neural activity that help explain its behavioral outcomes. Several electroencephalographic (EEG) studies of motor performance have associated EEG alpha activity with conscious motor processing (Hatfield et al., 2013; Masters & Maxwell, 2008; Zhu, Poolton, Wilson, Maxwell, & Masters, 2011). Alpha has an inhibitory function, whereby greater alpha power (i.e., the magnitude of alpha activity) indicates greater inhibition and lower alpha power indicates a greater release from inhibition (Klimesch, Sauseng & Hanslmayr, 2007). Alpha connectivity is another EEG-derived index representing cortico-cortical communication, whereby highly synchronous activity between sites reflects strong connectivity and less-synchronous activity reflects weaker connectivity (Lachaux, Rodriguez, Marinier, & Varela, 1999). During the final moments of preparation for action, individuals deemed more likely to plan and control movements consciously (e.g., beginners; individuals scoring high in trait movement-related self-consciousness) showed comparatively lower left-temporal (e.g., T7) alpha power and stronger alpha connectivity between left-temporal (e.g., T7) and the frontal (e.g., Fz) channels than their less likely counterparts (Hatfield et al., 2013; Gallicchio, Cooke, & Ring, 2017, 2016; Zhu et al., 2011). Taken together, these results associated conscious motor processing with a relative increase in activity (release from inhibition) over the left-temporal region, and an increased communication between left-temporal and frontal regions of the cortex. Considering the conceptual overlap between instructional self-talk and conscious motor processing (Hatfield et al., 2013), we hypothesize that the use of instructional self-talk (compared to motivational self-talk)

will be characterized by decreased left-temporal alpha power and increased left-frontotemporal alpha connectivity during movement preparation.

Finally, additional predictions concerning the neural basis of instructional self-talk were made based on neuropsychological models of feedback and feedforward motor control (Ashe, Lungu, Basford, & Lu, 2006; Babiloni et al., 2011; Murata & Ishida, 2007). These models suggest that the top-down (feedforward) control of actions is linked to the activity of the frontoparietal network, a circuit involving frontal and parietal regions. The main function of the frontoparietal network is to integrate frontally generated decisions and action plans with parietally generated multi-modal representations based on an integration of visual and somatosensory information (see Ashe et al., 2006; Murata & Ishida, 2007). When greater top-down control is attained, the network is driven more by frontal regions, while when less control is required, execution becomes more sensory-driven and the balance of the network is shifted toward parietal regions (Ashe et al., 2006).

Although this theorizing has yet to be directly tested with EEG data, re-examination of previously published evidence provides a foundation for a series of hypotheses. For example, Cooke and colleagues (2015) revealed that cortical activity over frontal and central regions increased (i.e., less alpha power) in the trials following golf putting movement errors, reflecting increased top-down control to correct the error. Moreover, Gentili and colleagues (2015) reported a progressive isolation of frontal sites (i.e., weaker frontal connectivity), as participants switched from a bottom-up (feedback based) to a top-down (feedforward based) control of movement. Verbalizing movement instructions and engaging in conscious motor processing is an example of top-down control (Hardy, 2006). Accordingly, following Ashe and colleagues' frontoparietal network model, we hypothesize that the use of instructional self-talk will encourage top-down control characterized by less frontal and greater parietal alpha power, alongside reduced frontoparietal connectivity. This reflects preferential utilization of conscious instructions (i.e., top-down) over more subtle visual and somatosensory (i.e., bottom-up) information.

1.2 | Motivational self-talk

Motivational self-talk is thought to improve the performance by nurturing confidence (Hardy, 2006) and creating psychological activation states that can support increased effort and behavioral persistency (cf., Bandura, 1997; Theodorakis et al., 2000). Nevertheless, nearly all the data concerning motivational self-talk have been treated it as though it is a unidimensional construct. Corroborating our multidimensional stance, Hardy et al.'s (2001) qualitative results revealed that

motivational self-talk is comprised three aspects: arousal, drive, and mastery functions. Motivational arousal self-talk is concerned with athletes' use of self-talk to psych themselves up and regulate their arousal levels. As a result, the researchers proposed that arousal oriented self-talk is more relevant for the competition setting characterized by pressure and stress than more emotionally neutral contexts (such as the acquisition of a fine motor skill in the laboratory). Motivational drive self-talk aids the individual to keep striving toward his/her goals with the necessary determination and effort by maintaining motivation and effort levels via self-statements that among other things provide encouragement. Lastly and of greatest relevance for the present study, motivational mastery self-talk helps the individual to remain focused on the task at hand (e.g., by clearing his mind of mistakes he has committed), bolsters self-confidence, and facilitates effective coping in challenging situations (e.g., when confronted with repeated negative mastery experiences at the early stages of motor learning). This type of enhanced motivational state epitomized by increased confidence, effort, and behavioral persistency, closely resembles what is known as approach motivation enabling action toward a goal and is linked to the so-called behavioral activation system (Gray, 1994). In contrast, the behavioral inhibition system, which is associated with avoidance-motivation, promotes the situational avoidance as well as increased the attention toward aversive stimuli (Gray, 1994). To distinguish between approach and avoidance motivation, it is common practice to compute asymmetry scores based on frontal EEG alpha power: relatively greater left-frontal asymmetry characterizes approach motivation, while relatively greater right-frontal activation characterizes avoidance motivation (e.g., Harmon-Jones et al., 2010). Due to the confidence supportive nature of motivational mastery self-talk (Hardy, 2006), it is likely that the use of such self-talk encourages approach rather than avoidance motivation. Thus, we predicted that using motivational mastery self-talk would be characterized by a relative increase in left-frontal cortical activity compared to instructional self-talk.

As motivational mastery self-talk, used in conjunction with non-physically demanding tasks such as fine motor skills, should influence individuals' attempts to succeed, the cognitive resources allocated to the task, and their overall effort and persistence; this form of self-talk will likely be characterized by cardiovascular indices of increased mental effort. For example, a large body of research has associated increases in mental effort with an increase in heart rate, and reduction in heart rate variability (Mulder, 1992; Wilson, Smith, & Holmes, 2007). Accordingly, we predicted that our motivational self-talk would elicit greater heart rate and less variability in heart rate during the final seconds of motor preparation (i.e., event-related heart rate; Cooke et al., 2014), when compared to instructional self-talk.

1.3 | The present study

This study investigated a psychophysiological model distinguishing instructional and motivational self-talk and their differential effects on motor skill execution. We instructed novice golfers to putt golf balls under either instructional self-talk or motivational self-talk. On the one hand, we hypothesized that by increasing top-down control of the motor system, instructional self-talk would be characterized by EMG and kinematic indices of better technique (greater forearm activation, and a slower, straighter swing), greater left-temporal activation to indicate increased conscious processing, and a frontoparietal network function shifted toward frontal rather than parietal sites to reflect more top-down feedforward control. On the other hand, we hypothesized that by fostering approach motivation, motivational self-talk would be characterized by left-frontal asymmetry indicative of approach motivation, and cardiovascular changes indicative of increased mental effort.

2 | METHOD

2.1 | Participants

Participants were 40 (19 male, 21 female), right-handed (Edinburgh Handedness Inventory $\geq +70$, Oldfield, 1971), golfing novices, aged 26.97 ($SD = 4.40$) years. Participants were randomly allocated to an instructional self-talk or a motivational self-talk group (between-participant factor) and completed 80 trials of a golf-putting task. We used novices and a golf-putting task based on meta-analytic evidence showing largest effects sizes for self-talk manipulations involving novel tasks with fine-motor requirements (Hatzigeorgiadis et al., 2011) and because EEG can be recorded while putting (e.g., Cooke et al., 2014). Our sample size was powered at .80 to detect large between participant effects ($\eta_p^2 = .17$ in a randomized analysis of variance (ANOVA) and $d = .80$ in between group t tests) at the 5% level of significance. All participants had normal/corrected vision, refrained from consuming alcohol, drugs (24 hr before), and caffeine (3 hr before), and reported more than 6 hr of sleep during the night preceding their participation. Participants provided informed consent before taking part and were paid £10 upon completion. The protocol was approved by the local research ethics committee.

2.2 | Experimental task

Participants were asked to putt golf balls (diameter = 4.27 cm) on an artificial flat putting surface to a target—adhesive paper marker (diameter = 6 mm)—at a distance of 2.46 m, using a

blade-style putter (length 90 cm). Participants were instructed to putt at their own pace as accurately as possible in order to get the final position of each ball “as close as possible to the target”. They were additionally instructed to rehearse an instructional or motivational self-talk cue (see Supplementary Material) immediately before executing the swing.

2.3 | Procedures

Participants completed a 2-hr testing session. Following instruction and instrumentation they sat and rested with eyes-closed for 1 min followed by eyes-open for 1 min while EEG was recorded to later adjust frequency bands to the individual alpha peak (IAF, see Bazanova & Vernon, 2014;Corcoran, Alday, Schlesewsky, & Bornkessel-Schlesewsky, 2018). The experiment then comprised three phases: (a) 10-putt familiarization task; (b) self-talk training; (c) putting task phase.

2.3.1 | 10-putt familiarization task

As participants were novices and unfamiliar with the laboratory setting, they performed 10 putts to familiarize with the experimental conditions.

2.3.2 | Self-talk training

Given that Hatzigeorgiadis and colleagues' (2011) metadata supports the inclusion of a self-talk training phase, our participants practiced the self-talk instructions while performing an aiming motor task. A pre-recorded podcast was used to explain either instructional or motivational self-talk (see Supplementary Material). Participants then completed 20 trials of a mini basketball free-throw task. In the first 10 trials they were asked to practice the skill without any additional instruction. In the second 10 trials, they rehearsed a self-talk cue corresponding to the group they were assigned to before each attempt. The use of this task ensured that all participants had experience using self-talk during a precision motor task that is dissimilar to our experimental task (cf. Hatzigeorgiadis, Zourbanos, Mpoumpaki, & Theodorakis, 2009).

2.3.3 | Putting task phase

Next, participants were told that they would begin the main golf-putting phase of the experiment. Participants were assigned a group-specific cue for the golf-putting task: instructional cue “feet still—wrists locked—arms through”; motivational cue “come on, I can do this”. We assigned cues to increase within-group consistency (in terms of content

and length) in the use of covert-verbalizations in preparation for each putt. We developed the self-talk cues based on previous protocols (Hardy, 2006; Hardy, Begley, & Blanchfield, 2015; Theodorakis et al., 2000), golf-coaching manuals, and pilot testing (see Supplemental Material). We instructed participants to silently say their assigned cue in their mind before every putt. They then completed a total of 80 putts with a 2-min break at the mid-point. We reminded participants about their self-talk cue every five putts during this phase of the experiment.

2.4 | Physiological data

2.4.1 | EEG data

EEG was recorded from 32 active electrodes at Fp1, Fp2, AF3, AF4, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO3, PO4, O1, Oz, and O2 (10–20 system, Jasper, 1958). In addition, active electrodes were positioned on each mastoid, at the outer canthus and below each eye to record vertical and horizontal electrooculogram. All channels were recorded in monopolar. The signals were sampled at 1,024 Hz, with no online filter, using an ActiveTwo amplifier (Biosemi, The Netherlands). Electrode offset was kept below 15 mV. TTL triggers were sent to the BioSemi system to identify swing-onset, identified by the putter head being moved away from and thereby breaking an infrared beam controlled by an optical sensor (S51-PA 2-C10PK, Datasensor, Monte San Pietro, Italy) and a microphone (NT1, Rode, Silverwater, Australia) connected to a mixing desk (Club 2000, Studiomaster, Leighton Buzzard, UK), which detected the putter-to-ball contacts. These signals were recorded using both Actiview (BioSemi) and Spike2 Software (CED-2).

Offline signal processing was performed using EEGLAB (Delorme & Makeig, 2004), ERPLAB (Lopez-Calderon & Luck, 2014), and bespoke scripts in MATLAB (Mathworks Inc., USA). Data were downsampled to 250 Hz, re-referenced to the average of all 32 EEG channels (no bad channels were identified), and filtered from 0.1 to 30 Hz (Butterworth, 12dB/40roll-off order2 non-causal). In line with previous research (e.g., Cooke et al., 2014; Hatfield et al., 2013) data were extracted from -4000 ms to $+1,000$ ms relative to swing-onset, and centered around the average voltages between -200 ms and 0 ms. Epochs were visually inspected and rejected if they contained gross artifacts. The number of epochs retained was 78.37 ($SD = 3.14$). Independent component analysis (ICA) weights were obtained through the RunICA informax algorithm (Makeig, Bell, Jung, & Sejnowski, 1996) running on these same EEG data (32 channels, yielding the same number of independent components): they were high-pass filtered to 1 Hz (FIR [finite impulse response] filter,

filter order 826) and concatenated across all trials within each participant. Then ICA weights were applied to the original 0.1–30 Hz filtered signals, and artifactual components (e.g., eye or muscle related) flagged by automated procedures (SASICA plugin; Chaumon, Bishop, Busch, 2015) and then, visually inspected were manually rejected.

Time-frequency analysis

Time-frequency analysis was applied by convolving the Fast-Fourier Transform (FFT) power spectrum of each EEG artifact-free epoch with a family of complex Morlet wavelets, defined as a Gaussian-windowed complex sine wave: $e^{i2\pi ft} e^{-t^2/2\sigma^2}$; where t is time, f is frequency bin, which increased from 4 to 30 Hz in 30 logarithmically steps, and σ defines the width of each frequency band (set to $\text{cycles}/2\pi f$, with cycles ranging from 3 and 6), and then, taking the inverse FFT to obtain the analytic signal z . From the convolution we obtained: (a) estimates of instantaneous power (squared magnitude of the analytic signal); and (b) phase (phase angle of the analytic signal) which was then used to compute inter-site connectivity.

Individual alpha frequency

Following the approach advocated by Bazanova and Vernon (2014), the individual frequency bands were calculated based on the individual alpha peak (IAF, Klimesch, 1999). IAF was calculated with the IAF toolbox (Corcoran et al., 2018) based on a 60s segment of eyes-closed EEG recording taken before the beginning of the task, which was processed in the same way as task-related data (excluding the epoching). The mean IAF was 9.92 ± 1.17 for the instructional group and $9.92 \pm .83$ for the motivational group.

Power

Changes in instantaneous power were calculated from the complex signal for each frequency bin (f) as the squared magnitude of the result of the convolution defined as Z_t (power time series: $p_t = \text{real}(z_t)^2 + \text{imag}(z_t)^2$).

For the analysis of power and connectivity, we focused on the IAF-adjusted high-alpha band (i.e., IAF to IAF + 2 Hz) since it is more sensitive than other frequency bands to task-related changes (Babiloni et al., 2011). Crucially, no baseline normalization was employed. Following the approach of Gallicchio and colleagues (Gallicchio et al., 2017) to control for skewness and interindividual differences, trial-averaged absolute alpha power was median-scaled log transformed, whereby values for each participant were scaled by the median of all values (electrode \times points \times block matrix) per each wavelet (representing a frequency bin) within that participant, and then, subjected to a 10-log_{10} transformation. Power was then averaged across IAF-adjusted frequency bands, and five 1 s time bins relative to movement initiation (bin1: -4 s to -3 s; bin2: -3 s to -2 s; bin3: -2 s to -1 s; bin4: -1 s to 0 s; bin5: 0 s to $+1$ s).

For the analysis of frontal asymmetry, we focused on the IAF-adjusted low-alpha (i.e., IAF-2Hz to IAF; Davidson, Ekman, Saron, Senulis, & Friesen, 1990). Asymmetry scores [$\log_e(\text{right}) - \log_e(\text{left})$] were computed for each time bin at medial frontal (F3, F4) regions (Coan, Allen, & Harmon-Jones, 1992001). Raw power values were for the calculation used as normalization and control for skewness is afforded by the natural log transformation and the subtraction (Davidson et al., 1990). Since more alpha power indicates less cortical activity, positive asymmetry scores indicate greater relative left-frontal activation, and negative asymmetry scores indicate greater relative right-frontal activation.

Connectivity

Functional connectivity between sites was computed in terms of inter-site phase clustering (ISPC) based on the phase angle time series, $\phi_t = \text{imag}(z_t)^2 / \text{real}(z_t)^2$. This measure was preferred to others (e.g., magnitude squared coherence) because it is independent of absolute power variations (Cohen, 2014; Lachaux et al., 1999). ISPC_{trials} measures consistency of phase angle differences at specific time points across trials and is calculated with the following formula: $\text{ISPC}_{xy}(f) = \left| n^{-1} \sum_{t=1}^n e^{i(\theta_x(tf) - \theta_y(tf))} \right|$; where n is the number of trials, i is the imaginary operator, θ_x and θ_y are the phase angles of the recorded signal at two different scalp locations, t is trial, and f is the frequency bin, $e^{i(\theta_x(tf) - \theta_y(tf))}$ is the complex vector with magnitude, $n^{-1} \sum_{t=1}^n (\cdot)$ denotes averaging over trials (for ISPC_{trials}), and $|\cdot|$ is the magnitude of the averaged vector (Cohen, 2014; Lachaux, Rodriguez, Martinerie, & Varela, 1999). The resulting ISPC is a real number between 0 (no functional connection) and 1 (perfect functional connection). ISPC values were Z-transformed (i.e., inverse hyperbolic tangent) to ensure normal distribution before statistical analyses were performed (Halliday et al., 1995).

2.4.2 | Cardiac activity

Cardiac activity was derived from an electrocardiogram (ECG) obtained using three single-use silver/silver chloride spot electrodes (BlueSensor SP, Ambu, Cambridgeshire, UK) placed on the clavicles and the lowest left rib. The ECG signal was amplified (Bagnoli-4, Delsys, Boston, MA), filtered (1–100 Hz), and digitized at 2,500 Hz with 16-bit resolution (CED Power 1,401, Cambridge Electronic Design, Cambridge, UK) using Spike2 software (Cambridge Electronic Design).

The ECG signal was then used to compute the event-related instantaneous heart rate time series. Typically, in golf-putting research, there is a distinct event-related heart rate variability profile characterized by a deceleration in heart rate during the 6 s prior to movement, and an acceleration in heart rate during the 6 s post-movement (e.g., Cooke

et al., 2014; Neumann & Thomas, 2008). Event-related heart rate in the final moments pre-movement until the putter-ball impact (typically around 1 s post movement initiation) are of particular interest because more pronounced heart rate deceleration (i.e., greater event-related heart rate variability) has been associated with increased automaticity and superior performance (e.g., Neumann & Thomas, 2008). Based on previous research (e.g., Cooke et al., 2014) the continuous time series was first segmented into epochs from -6 s to $+6$ s relative to swing-onset, and then, voltages were centered by means of baseline subtraction (i.e., the mean value of the whole epoch was subtracted from each point of the time series). Instantaneous heart rate was derived from the intervals between successive R-wave peaks (R–R intervals) of the ECG in each epoch. Data were processed as follows: (a) a filter in the frequency domain was applied to remove slow frequencies; (b) an initial set of probable R-wave peaks were identified; (c) segments containing artifacts were identified based on extreme values and were interpolated; (d) the minimum distance between peaks was identified and used to optimize the R-wave peaks identifier; (e) the identified R-wave peaks were manually reviewed and confirmed/adjusted as necessary; (f) the accepted R-wave peaks were used for the calculation of the R–R intervals; and (g) instantaneous heart rate (beats per minute) was calculated as $60,000 / (\text{R–R interval})$. Each epoch was then split into thirteen 1s time bins and the nearest instantaneous heart rate value was assigned to each bin. Absolute heart rate was calculated by taking the average heart rate across all bins. Event-related variability in heart rate was calculated by computing a difference score between heart rate in the earliest seconds preceding the swing (bin1: -6 ; bin2: -5 , bin3: -4), where heart rate is typically greatest and the value of the heart rate in the second after the movement initiation (bin007: $+1$), were maximal bradycardia is normally achieved (Cooke et al., 2014). Accordingly, a larger difference score indicates greater event-related heart rate variability (greater rate of change during the event-period) and was expected for the instructional self-talk group. A smaller difference score indicates less event-related heart rate variability (smaller rate of change during the event-period) and was expected for the motivational self-talk group.

2.4.3 | Muscle activity

Muscle activity was derived from an electromyogram (EMG) measured using a differential surface electrode (DE 2.1, Delsys) affixed to the *extensor carpi radialis* and the *flexor carpi ulnaris* of the left arm, and a ground electrode (BlueSensor SP, Ambu, Cambridgeshire, UK) on the left collarbone. These muscles were chosen based on previous research implicating them in the putting stroke of right-handed golfers (e.g., Cooke et al., 2010). The EMG signal was amplified (Bagnoli-4, Delsys), filtered

(20–450 Hz), and digitized at 2,500 Hz with 16-bit resolution (CED Power 1,401) using Spike2 software.

The event-related activity of the flexor and extensor muscles was then calculated via the following steps: (a) the continuous data time series was rectified, (b) continuous data were segmented into epochs from -6 s to $+6$ s relative to swing-onset; (c) voltages were centered by means of baseline subtraction (i.e., the mean value of the whole epoch was subtracted from each point of the time series); and (d) each epoch was split into 500 ms time bins and the average voltage for each bin was calculated (e.g., muscle activity for 6 s before was calculated as the mean activity between 6.25 and 5.75 s prior to movement; see Cooke et al., 2014, 2015).

2.5 | Behavioral data

2.5.1 | Performance outcome

Participants' performance was evaluated in terms of angle error (degrees), length error (cm), and radial error (cm), which, respectively yield measures of directional accuracy, force accuracy, and a combination of direction and force. These measures were computed for each putt using a camera system (Neumann & Thomas, 2008) and averaged (geometric mean, Gallicchio et al., 2017) to yield measures for each block.

2.5.2 | Movement kinematics

Technique was assessed by means of movement kinematics using a triaxial accelerometer (LIS3L06AL, ST Microelectronics, Geneva, Switzerland). Acceleration on the X , Y , and Z axes corresponded to lateral, vertical, and back-and-forth movement of the clubhead, and assessed clubhead orientation, clubhead height, and impact velocity, respectively. The signals were conditioned by a be-spoke buffer amplifier with a frequency response of DC to 15 Hz. Both accelerometer and amplifier were mounted in a 39 mm \times 20 mm \times 15 mm plastic housing secured to the rear of the putter head. To compute kinematic variables, we scored acceleration for each putt from the onset of the downswing phase of the putting stroke until the point of ball contact (e.g., Cooke, Kavussanu, McIntyre, Boardley, & Ring, 2011; Cooke et al., 2010). Specifically, we calculated average acceleration for the X , Y , and Z axes.

2.6 | Statistical analyses

Performance data were analyzed using independent samples t tests to compare the two groups (instructional, motivational).

Instantaneous heart rate and muscle activity were analyzed using Group \times Bin (2×13 ; $-6, -5, -4, -3, -2, -1, 0, +1, +2, +3, +4, +5, +6$) ANOVAs. Power was analyzed by a Group \times Site \times Bin (bin1: -4 s to -3 s; bin2: -3 s to -2 s; bin3: -2 s to -1 s; bin4: -1 s to 0 s; bin5: 0 s to $+1$ s) ANOVA followed by separate ANOVAs at specific sites based on the effects that emerged. Based on our study aims, the factor site included the following channel subsets: frontal (Fz, F3, F4, F7, F8), central (Cz, C3, C4), parietal (Pz, P3, P4), occipital (Oz, O1, O2), and temporal (T7, T8).

Connectivity was analyzed with separate Group \times Pair \times Bin ANOVAs to explore how the frontal-midline (Fz) and the parietal-midline (Pz) were, respectively connected with the other electrodes considered (Fz, C3, C4, Cz, C3, C4, Pz, P3, P4, Oz, O1, O2, T7, T8). Moreover, Group \times Bin ANOVA conducted for the T7-Fz pair based on our a priori hypothesis. Frontal asymmetry scores were analyzed through Group \times Bin ANOVAs for the frontal (F3, F4) channel pair. The bin factor is recommended in studies of self-paced aiming movements in order to account for phasic shifts in power and connectivity during preparation for action (Cooke et al., 2014).

Significant main effects and interactions were probed by separate ANOVAs for each group, or bin, using polynomial trend analyses. The multivariate method of reporting results was adopted as it minimizes the risk of violating sphericity and compound symmetry assumptions in repeated measures ANOVA (Vasey & Thayer, 1987). Effect size is reported with Cohen's d (t tests) and partial eta-squared (η_p^2 ; ANOVAs) with values of $d = 0.20$ and $\eta_p^2 = 0.01$ indicating small effects, $d = 0.50$ and $\eta_p^2 = 0.06$ indicating medium effects, and $d = 0.80$ and $\eta_p^2 = 0.14$ indicating large effects (Cohen, 1988).

3 | RESULTS

3.1 | Cortical activity

3.1.1 | Power

The Group \times Site \times Bin mixed-model ANOVA, conducted to obtain a general picture of the power profiles, revealed significant main effects of site, $F(15, 24) = 126.56$, $p < .001$, $\eta_p^2 = .99$, and bin, $F(4, 35) = 4.54$, $p = .005$, $\eta_p^2 = .34$, no effect for group, $F(1, 38) = 3.35$, $p = .075$, $\eta_p^2 = .81$, and a significant group \times site interaction, $F(15, 24) = 2.95$, $p = .009$, $\eta_p^2 = .65$. Event-related changes in power during the preparatory period were evident (main effect of bin, quadratic trend $p = .001$, $\eta_p^2 = .28$), with a decrease in the 2 s before and in the second after swing-onset. Moreover, we also observed a specific topographic distribution with highest power at occipital (Oz, O1, O2), intermediate at temporal (F7, F8, T7, T8) and then, frontal (Fz, F3, F4), reduced at parietal (Pz, P3, P4), and lowest at central sites (Cz, C3,

C4) (see Supplementary Material Figure S3). However, of most interest was the group \times site interaction. Separate ANOVAs performed for each channel revealed significant group differences at Pz and P4, F 's (1,38) = 8.97–12.02, p 's < .01, η_p^2 's = .19–.24, characterized by more power for the instructional group (Pz M = .59, SD = 1.20; P4 M = 0.26, SD = 1.30) than the motivational group (Pz M = -2.07, SD = 2.04; P4 M = -1.25, SD = 1.17). There were no significant group effects at any other sites. Effects are summarized in Figure 1a.

In sum, power analyses tended to show (a) a well-defined topographical distribution of alpha activity (see Supplementary Material Figure S3), with highest power at occipital electrodes, intermediate power at temporal and frontal electrodes, relatively low power at parietal electrodes, and lowest power at central electrodes; (b) a swing-onset related decrease in power; (c) greater power at parietal sites in the instructional group than the motivational group (Figure 1a).

3.1.2 | Connectivity

We conducted separate analyses to specifically assess the connectivity array with center of mass frontal-midline (Fz), and the parietal-midline (Pz).

Fz connectivity

The Group \times Pair \times Bin mixed-model ANOVA assessing Fz connectivity revealed a main effect of pair, $F(9, 30) = 120.21$, $p < .001$, $\eta_p^2 = .97$, with strongest connectivity at frontooccipital (Fz-Oz, Fz-O1, Fz-O2) and frontoparietal pairs (Fz-P4, Fz-P3, Fz-Pz), intermediate at frontocentral pairs (Fz-C3, Fz-C4), and lowest at lateral frontotemporal pairs (Fz-T7, Fz-T8). Moreover, the analysis revealed a main effect of bin, $F(4, 35)$

= 8.18, $p = .001$, $\eta_p^2 = .48$ (quadratic trend $p = .001$, $\eta_p^2 = .28$, increase-decrease-increase) characterized by a decrease in connectivity (at bin 4) followed by an increase prior to swing onset (at bin 5). Of most interest, there was also a main effect of group, $F(1, 38) = 11.81$, $p = .001$, $\eta_p^2 = .24$, whereby connectivity was stronger in the motivational ($M = .52$, $SD = .12$) than in the instructional ($M = .44$, $SD = .08$) self-talk group. The Group \times Bin mixed-model ANOVA for left-frontotemporal connectivity (T7-Fz), conducted based on our a priori hypotheses, confirmed the main effect of group, $F(1, 38) = 6.97$, $p = .012$, $\eta_p^2 = .15$, whereby connectivity was stronger for the motivational ($M = .26$, $SD = .10$) than the instructional ($M = .20$, $SD = .04$) self-talk group.

Pz connectivity

The Group \times Pair \times Bin mixed-model ANOVA examining Pz connectivity revealed a main effect of pair, $F(8, 31) = 151.51$, $p < .001$, $\eta_p^2 = .97$, whereby connectivity was highest at frontoparietal, and parietooccipital pairs (Pz-F3, Fz-Pz, Pz-F4, Pz-O1, Pz-O2), intermediate at parietotemporal pairs (Pz-T7, Pz-T8) and lowest at parietocentral pairs (Pz-C3, Pz-C4). Importantly, it also confirmed the same main effect of group, $F(1, 28) = 5.67$, $p = .022$, $\eta_p^2 = .13$ (motivational $M = .42$, $SD = .11$ > instructional $M = .36$, $SD = .08$ self-talk) as was observed for Fz connectivity. However, no effect of bin emerged.

In sum, the connectivity analyses revealed (a) strongest interconnections between frontooccipital and frontoparietal pairs (see Supplementary Material Figure S4); (b) an event-related decrease in Fz connectivity prior to swing-onset; (c) group differences characterized by stronger connectivity in the motivational group at frontal (including the left-frontotemporal pair) and parietal midline pairs (Figure 1b).

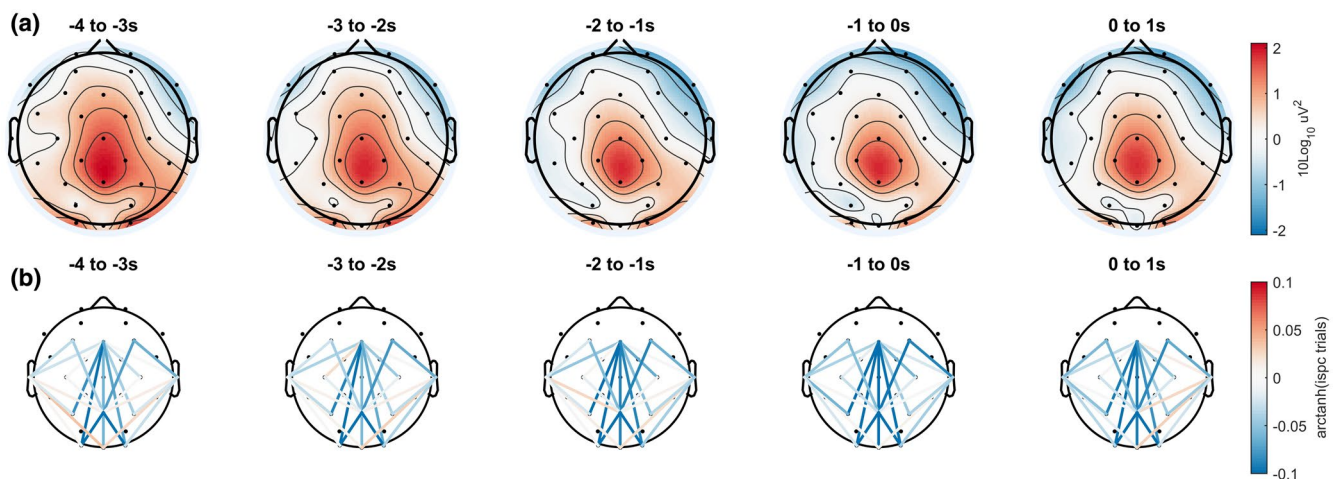


FIGURE 1 Instructional—motivational group differences: (a) topographic distribution of median scaled power; (b) topographic connectivity network of atanh(ISPCtrials). Each column represents time bin (-4 to -3, -3 to -2, -2 to -1, -1 to 0, 0 to +1). Red colors indicate higher values for instructional and blue colors indicate higher values for motivational self-talk groups

3.1.3 | Frontal asymmetry

The Group \times Bin mixed-model ANOVA failed to reveal any significant effects for F3–F4 asymmetry. Although non-significant, the means were in the expected direction (motivational self-talk $M = .03$, indicating asymmetry toward the left hemisphere; instructional self-talk $M = -.06$, indicating asymmetry toward the right hemisphere).

3.2 | Physiological data

3.2.1 | Cardiac activity

The Group \times Bin mixed-model ANOVA revealed main effects of bin, $F(12, 27) = 5.55$, $p = .001$, $\eta_p^2 = .71$, best described by a cubic trend ($p < .001$, $\eta_p^2 = .56$), whereby heart rate began decelerating 2 s prior to swing initiation, the strongest bradycardia occurred during movement execution, before heart rate returned progressively to baseline values in the following seconds. The targeted event-related heart rate change analysis was performed by comparing the magnitude of deceleration in the two groups, we calculated difference scores between the average heart rate in the seconds preceding the deceleration (bin001: -6 ; bin002: -5 , bin003: -4) and the value of the heart rate in the second when deceleration was maximal (bin007: $+1$). These scores were analyzed with a two-way randomized ANOVA which revealed a marginal effect of group, $F(1, 38) = 3.31$, $p = .076$, $\eta_p^2 = .08$, whereby the difference score was larger (i.e., more event-related heart rate variability) in the instructional group ($M = 8.06$ beats) compared to the motivational group ($M = 4.75$ beats). These results are displayed in Figure 2a.

3.2.2 | Muscle activity

For the extensor muscle, the Group \times Bin mixed-model ANOVA revealed a main effect of bin, $F(12, 27) = 4.04$, $p = .001$, $\eta_p^2 = .64$, which was best described by a quadratic trend ($p < .001$, $\eta_p^2 = .40$). Muscle activity increased 1 s before swing-onset, peaked during swing-execution, and returned to baseline thereafter. No main effect of group or Group \times Bin interaction were observed. These results are displayed in Figure 2b.

For the flexor muscle, the same analysis revealed a main effect of bin, $F(12, 27) = 2.53$, $p = .022$, $\eta_p^2 = .53$, best described by a quadratic trend ($p < .001$, $\eta_p^2 = .33$), and a main effect of group, $F(1, 38) = 4.92$, $p = .033$, $\eta_p^2 = .11$. Overall, muscle activity increased relative to the swing (i.e., increase in the second preceding swing onset, peak during movement, and return to baseline thereafter) and was higher in the instructional group. These results are displayed in Figure 2c.

3.3 | Behavioral data

3.3.1 | Performance outcome

The independent samples t test failed to reveal any group differences. Means are summarized in Table 1.

3.3.2 | Movement kinematics

The independent samples t test revealed group differences for X (lateral) axis acceleration, $t(38) = -2.70$, $p = .011$, $d = .83$, whereby acceleration was smaller for the instructional group. Means are summarized in Table 1.

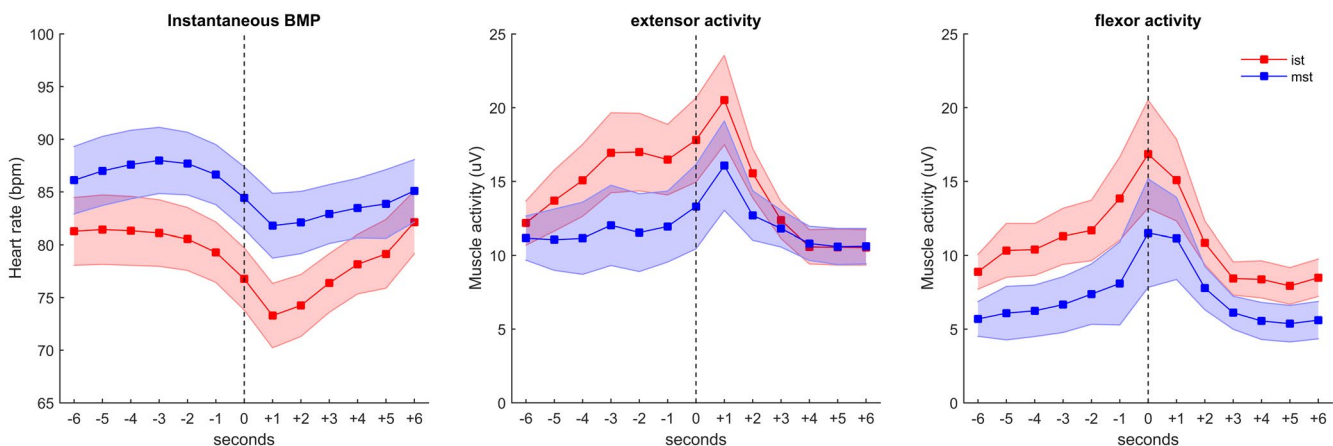


FIGURE 2 The figures represent the Bin \times Group effects for cardiac activity and muscle activity. The x-axis represents seconds around swing-onset (time = 0 s); the y-axis represents (a) instantaneous heart-rate; (b) extensor muscle activation; (c) flexor muscle activation. Red = Instructional self-talk; Blue = Motivational self-talk. Shading indicates standard error of the means

TABLE 1 Mean (*SD*) of performance and kinematic measures per group (i.e., instructional and motivational)

Measure (range)	Mean (<i>SD</i>)
<i>Radial error (cm)</i>	
Instructional	28.15 (6.14)
Motivational	29.55 (6.09)
<i>Angle error (degrees)</i>	
Instructional	0.65 (0.26)
Motivational	0.74 (0.21)
<i>Length error (cm)</i>	
Instructional	25.19 (7.37)
Motivational	26.35 (6.41)
<i>Lateral (x-axis) acceleration (ms⁻²)</i>	
Instructional	0.33 (0.14)
Motivational	0.47 (0.19)
<i>Vertical (y-axis) acceleration (ms⁻²)</i>	
Instructional	0.45 (0.15)
Motivational	0.45 (0.17)
<i>Back-and-Forth (z-axis) acceleration (ms⁻²)</i>	
Instructional	2.42 (1.17)
Motivational	3.06 (1.77)

4 | DISCUSSION

The present study is the first to introduce and test a psychophysiological model of self-talk highlighting the distinctive features of instructional and motivational self-talk as it pertains to motor skill execution. The effective integration of multiple data sources (e.g., behavioral, cardiac, muscular, and neural) and contemporarily analyzed (e.g., scalp level time-frequency power and connectivity analysis via wavelet convolution; individual alpha peak adjustment) data afforded us a comprehensive appreciation of how instructional and motivational self-talk might facilitate the motor performance. Some predictions of the model were supported while a few were questioned. Each prediction is discussed and suggestions for refinement of our model in light of our findings are considered in the sections below.

4.1 | Instructional self-talk

We hypothesized that instructional self-talk would benefit motor closed-skill execution via increased top-down control of action (Hardy, 2006). Our kinematic results supported this prediction. Participants using instructional self-talk developed better technique, indexed by reduced lateral clubhead acceleration, compared to their motivational self-talk counterparts. Although there were no statistically significant effects on performance, inspection of the means reveals that

there was also a tendency for members of the instructional group to perform better than members of the motivational group, indexed by smaller radial, angular, and length errors. Participants using instructional self-talk also tended to display greater muscle activity, but since this spanned all epochs rather than being localized to epochs around the moment of impact, it provides only partial support for our muscle activity hypothesis. Rather than reflecting good technique in the form of accelerating through the ball, it is possible that the elevated muscle activity that characterized the instructional self-talk group is simply a further reflection of their top-down control, and their adoption of an internal focus on the mechanics of their swing (e.g., Zachry, Wulf, Mercer, & Bezodis, 2005). Accordingly, muscle activity could be reconceptualized in our model as a variable reflecting conscious control, rather than as an index of technique. If considered as an index of conscious control of movements, our muscle activity findings (i.e., greater for instructional than motivational self-talk) are consistent with what our model would predict for a fine-motor skill.

Based on findings from the conscious processing literature (Gallicchio et al., 2016; Hatfield et al., 2013; Zhu et al., 2011), we also expected participants in the instructional group to be characterized by greater left-temporal activity. Our results did not support this hypothesis. No group differences or group-related interactions emerged for left-temporal power. Further, and in direct contrast to our hypothesis, left-frontotemporal was significantly lower among participants using instructional self-talk compared to those using motivational self-talk. These findings could indicate that instructional self-talk is not associated with conscious motor processing, but since conscious motor processing involves the use of explicit instructions to guide movement, this seems unlikely. An alternative possibility is that left-temporal *power* may reflect self-talk frequency rather than self-talk content. In the current study, while the self-talk content employed by the instructional and motivational groups was clearly different, their self-talk frequency was the same. Previous studies advocating left-temporal power as a neural index of conscious motor processing are mainly based on expert versus novice comparisons or comparisons of explicit versus implicit training methods which could elicit different self-talk frequencies. Future studies should further investigate the relative effects of semantic content versus self-talk frequency on left-temporal power to shed light on this pressing issue.

Self-talk frequency cannot account for our finding of lower left-frontotemporal *connectivity* in the instructional self-talk group. It should be noted that connectivity between frontal electrodes and all other sites was lower in members of the instructional self-talk group. In other words, there was nothing distinct about left-frontotemporal connectivity compared to any other form of frontal connectivity in our experiment.

These findings cast some doubt on the validity of left-frontotemporal connectivity as an index of conscious motor processing and are at odds with previous literature in the area. However, many of the previous studies endorsing left-frontotemporal measures did so via expert-novice comparisons (e.g., Gallicchio et al., 2016) or by comparing groups who were high versus low in dispositional reinvestment (e.g., Zhu et al., 2011). Accordingly, other between-group differences (e.g., experience, personality) may account for some of the effects in previous studies. The validity of T7-Fz connectivity as an index of verbal-analytic processing has also been questioned in a recent study by Parr, Gallicchio, Harrison, Johnen, and Wood (2019). Rather than reflecting conscious motor processing, it seems on the basis of our results that all connectivity between frontal and other sites could reflect the extent to which sensory (e.g., auditory, visual, perceptual) processes influence frontally-generated action plans. By having a clear instructional focus, members of our instructional self-talk group may have been able to block, to an extent, communication from other cortex sites from interfering with motor planning. Further elaboration of this idea is provided in reference to our next hypothesis.

Finally, based on neuroscience literature examining feedback and feedforward motor control (Ashe et al., 2006), we hypothesized that participants using instructional self-talk would be characterized by less frontal and more parietal alpha power, alongside reduced frontal and parietal connectivity. This hypothesis was partially supported. Specifically, the instructional group tended to be characterized by greater parietal alpha power (suggesting diminished sensorimotor processing). Moreover, it showed weaker connectivity between frontal sites and the rest of the sites examined, including the parietal ones. This suggests reduced integration of frontal plans with different types of information, such as perceptual-sensorimotor, visual and verbal information. However, no statistically significant alpha power differences were observed at frontal sites. These findings provide some evidence to suggest that instructional self-talk encouraged a reduced relative weight of parietal processing in the frontoparietal network, indicative of more top-down control of action (Ashe et al., 2006). However, as group differences in connectivity were widespread rather than localized to frontal-parietal electrode pairs, our results are not fully in line with Ashe and colleagues' (2006) frontoparietal model. Instead, our data suggest reduced parietal activity and reduced connectivity between frontal and all other sites appear the most promising neurophysiological signatures of instructional self-talk and could explain the performance benefits of this self-talk modality (Hardy, 2006).

4.2 | Motivational self-talk

We expected motivational self-talk to influence EEG frontal asymmetry and cardiac activity. First, based on theories of

approach and avoidance motivation, we hypothesized that motivational self-talk would encourage approach motivation, characterized by a relative increase in left-frontal cortical activity (Harmon-Jones et al., 2010). Results failed to support this hypothesis. There was no group main effect for frontal asymmetry. It is possible that the lack of group differences was due to instructional self-talk also encouraging approach motivation to some extent; future studies measuring frontal asymmetry could compare instructional, motivational and no self-talk groups to test this possibility. Additionally, given that some research (e.g., Hardy et al., 2015) indicates that the beneficial role of instructional self-talk is less pronounced, while the effect of motivational self-talk is more distinct for skilled performers, it is possible that any effects of motivational self-talk on frontal asymmetry would manifest more clearly when utilized by experts. Alternatively, the influence of motivational self-talk on EEG frontal asymmetry might be clearer if researchers incorporated gross motor skills into their studies, for which this type of self-talk has been demonstrated to be more effective (Hatzigeorgiadis et al., 2011).

Our second hypothesis was that motivational self-talk would elicit greater heart rate and less event-related heart rate variability, when compared to instructional self-talk. This was based on the premise that motivational self-talk would increase effort, and the compelling literature associating increased effort with greater heart rate and reduced heart rate variability (Mulder, 1992; Wilson et al., 2007). Results partially supported this hypothesis. Members of the motivational self-talk group displayed a trend for higher heart rates and showed significantly less variability in heart rate during the 6 s preceding putts when compared to their instructional counterparts. While these cardiovascular effects of motivational self-talk did little to aid performance and kinematics during the current fine-motor task, they might be very helpful for gross motor tasks (Hardy, 2006; Hatzigeorgiadis et al., 2011); future research should examine this suggestion.

4.3 | Limitations and future directions

Our results should be interpreted in light of some methodological limitations. First, the current study did not contain a no-self talk control group. However, there is already a body of literature investigating the psychophysiological profiles of novice golfers who did not receive any self-talk intervention (e.g., Gallicchio et al., 2017); our results can be compared to these extant findings. Second, we did not use an irrelevant self-talk control group. However, we believe that the simple engagement in covert verbal activity, was, to some extent, controlled for by having both groups engage in self-talk. Nonetheless, given the current encouraging findings we see value in a future investigation which would specifically target this question. Third, since we tested novice golfers, and given the short acquisition phase,

we believe that a replication of the present investigation with expert golfers could improve our understanding of the effects of self-talk on skills that have already been acquired and consolidated (cf. Tod, Hardy, & Oliver, 2011). Fourth, we concede that the sample size was relatively small. Our sample size was larger than those adopted in previous EEG and motor performance studies (e.g., Cooke et al., 2014; Gallicchio et al., 2017; Gentili et al., 2015; Hatfield et al., 2013; Zhu et al., 2011), and it was sufficiently powered to detect a number of main and interaction effects as detailed above. Notwithstanding, it may be beneficial for future studies to replicate and extend our experiment with a larger population. Fifth, we recognize that our ISPC (also known as phase locking value) approach to assessing connectivity provides a consolidated measure of connectivity (Gallicchio et al., 2016, 2017; Parr et al., 2019), but does not provide information on connectivity direction. Future studies would do well to apply alternative techniques, such as Granger Causality (Cohen, 2014), which can provide insights on directionality. Such techniques are rarely applied in the brain and motor performance literature. Given our theorizing and partial support for a frontoparietal model of connectivity, we hope researchers now see a mandate for examining directionality to further test this model in future brain and motor performance studies.

Finally, we concede that the current study tested only a small portion of what is considered the most comprehensive and up-to-date self-talk taxonomy (Latinjak, Hatzigeorgiadis, Comoutos, & Hardy, 2019). In brief, Latinjak and colleagues (2019) distinguish between naturally happening verbalization (organic self-talk) and predetermined verbalizations based on a specific intervention plan (strategic self-talk). Verbalizations are also distinguished based on whether they have been assigned or self-developed and on their timing in relation to the skill (prior, during, after). The instructional or motivational function of self-talk can also be sub-divided (e.g., instructional skill focus, instructional strategy, motivational arousal, motivational mastery, motivational drive). The current study speaks to strategic, assigned, prior-to-skill, instructional skill focus and motivational mastery self-talk in the context of fine motor skills only. Future endeavors should explore the full ramification of this taxonomy and expand the currently presented psychophysiological model of self-talk.

4.4 | Conclusion

By employing a multi-measure approach (e.g., EEG, ECG, EMG, kinematics, and detailed execution parameters), the current study is the first to develop and test a mechanistic psychophysiological model of instructional and motivational self-talk and their effects on closed-skill motor performance. In partial support of the model, instructional self-talk was associated with less parietal activation and less connectivity between frontal and parietal electrodes and all other sites,

which resulted in better technique and a nonsignificant trend for better performance. This finding provides some support for an information-processing mechanism for the benefits of instructional self-talk and provides the first evidence for a neurophysiological signature of instructional self-talk. Moreover, motivational self-talk triggered a cardiovascular response (higher heart rate and reduced event-related heart rate variability) which suggests a mental-effort-based mechanism for the benefits of motivational self-talk. Finally, our results cast doubt on the validity of left-temporal EEG measures as reliable indices of verbal-analytic processes during motor preparation. In all, the study represents the most thorough self-talk investigation to date, both in detail and range of data collected (e.g., EEG, ECG, EMG, kinematics, and detailed execution parameters). Importantly, had we limited our analysis to the common behavioral measures obtained in the self-talk literature, our key mechanistic findings would have gone undetected. We hope that our development of a psychophysiological model of self-talk alongside the encouraging data to support the model inspires other researchers to follow and move beyond a reliance on behavioral and self-report data collection methods.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

TABLE S1 Putting rules

TABLE S3 Sequence of rules

FIGURE S3 Topographic distribution of median scaled power in the instructional (panel A) and motivational (panel B) group. Each column represents time bin (−4 to −3, −3 to −2, −2 to −1, −1 to 0, 0 to +1 s)

FIGURE S4 Topographic distribution of atanh(ISPC_{trials}) in the instructional (panel A) and motivational (panel B) group. Each row represents a block (B1, B2), each column represents time bin (−4 to −3, −3 to −2, −2 to −1, −1 to 0, 0 to +1 s)

FIGURE S5 Distribution of connectivity values. Blue: instructional group; Red: motivational group. The left panel shows ISPC across trials, whereas the right panel shows the imaginary part of ISPC across trials

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