

The Yips in Golf: Multimodal Evidence for Two Subtypes

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ABSTRACT

STINEAR, C. M., J. P. COXON, M. K. FLEMING, V. K. LIM, H. PRAPAVESSIS, and W. D. BYBLOW. The Yips in Golf: Multimodal Evidence for Two Subtypes. *Med. Sci. Sports Exerc.*, Vol. 38, No. 11, pp. 1980–1989, 2006. **Purpose:** To determine whether a model of two subtypes of yips is supported by evidence from a range of physiological, behavioral, and psychological measures. **Methods:** Fifteen golfers who experience yips symptoms while putting (mean age 58.1 yr, SD 13.6 yr), and nine golfers with no yips symptoms (mean age 39.6 yr, SD 19.3 yr) were recruited. Participants completed a golf history questionnaire to determine their playing experience and the nature of any yips symptoms experienced. In experiment 1, participants performed a putting task while electromyographic data were recorded from the forearm flexors and extensors and biceps brachii, bilaterally. The task was performed in two sessions, under low-pressure and high-pressure experimental conditions. The high-pressure condition was intended to increase anxiety through the use of a monetary incentive, video-taping of performance, and the presence of a confederate who provided negative feedback. Participants' state of anxiety was assessed using a questionnaire before each of the experimental sessions. In experiment 2, participants completed a task that required the inhibition of an anticipated response. Their accuracy and ability to inhibit their response was determined. **Results:** The golfers who experienced yips could be categorized according to whether they reported mainly movement-related symptoms (Type I) or anxiety-related symptoms (Type II). The Type I group exhibited greater muscle activity during putting and greater errors and less inhibition of the anticipated response task. The Type II group exhibited greater changes in cognitive anxiety and normal performance of the anticipated response task. **Conclusion:** This study provides evidence in support of two yips subtypes. Type I is related to impaired movement initiation and execution, whereas Type II is related to performance anxiety. **Key Words:** FOCAL DYSTONIA, ANXIETY, ELECTROMYOGRAPHY, INHIBITION

The yips was first defined as a motor phenomenon affecting golfers. It consists of involuntary movements during the performance of shots requiring fine motor control, such as chipping and putting (27). This definition has more recently been expanded to encompass an etiological continuum, anchored by a neurological basis (focal dystonia) at one end and a psychological basis (performance anxiety) at the other (33,34). The yips are characterized by excessive, involuntary muscle activity, resulting in unwanted movement or an inability to initiate movement. In sport, the yips are mainly reported by golfers, though the psychological characteristics of a small group of cricket bowlers who experience the yips have also recently been reported (3).

A recent review of the yips in golf described two subtypes of yips (33). These subtypes were defined by identifying themes in golfers descriptions of their yips experiences.

Those whose descriptions had a dystonia theme were classified as Type I yips, and those whose descriptions had a performance anxiety or “choking” theme were classified as Type II yips (33). An intermediate group was also identified; members of this group had experienced a mixture of Type I and Type II symptoms (33).

Type I yips has many characteristics in common with other forms of focal dystonia (33). The symptoms of upper-limb focal dystonias are dominated by involuntary contractions of the hand and forearm musculature, resulting in awkward, uncoordinated movements of the wrist and/or fingers. These symptoms are caused by the inappropriate cocontraction of antagonists and agonists, which is often associated with excessive levels of muscle activity (14). Symptoms are triggered by the performance of a skilled motor task that has been performed repetitively over the preceding months or years, such as writing, playing a musical instrument, or putting a golf ball. Multiple lines of investigation support the hypothesis that focal dystonias are associated with impaired inhibitory function at multiple levels of the central nervous system (12,13,17,35,37,39), which may stem from basal ganglia dysfunction (18,42). Like other forms of focal dystonia, Type I yips usually forces the sufferer to retrain him- or herself to perform the task differently, because an effective rehabilitation strategy has not yet been developed (33). Similarly, the symptoms of focal hand dystonia and Type I yips can sometimes be temporarily alleviated by the use of sensory tricks such as

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wearing a glove on the affected hand or using a different grip (2,23,25,33).

Type II yips golfers experience “choking” (33), where severe performance anxiety impairs the preparation and execution of movement, degrading performance. Generally, the combination of distraction by the specific competitive context and an increase in arousal and self-awareness increases performance anxiety and decreases confidence, which in turn increases the likelihood of suboptimal performance (33). The most anxiety-provoking situations for yips affected golfers include (i) leading the competition; (ii) tricky putts (putts with more than one change in inclination); (iii) playing against specific competitors; and (iv) the need to make easy putts (38). Most authors are in agreement that yips symptoms are exacerbated by heightened anxiety (3,30,33,34), though there remains some debate concerning whether excessive anxiety is sufficient to trigger yips symptoms (30,33).

Most studies of the yips have used questionnaires to explore the symptoms, clinical history, and sport experience of the participants (3,27,30,33,34). The model proposed by Smith et al. (33) was developed based on questionnaires completed by 70 golfers who had experienced yips symptoms. One of the limitations of this study is a potential response bias, because only 39% of invited participants (both with and without yips symptoms) completed and returned the questionnaires. A further limitation is that the respondents’ descriptions of their symptoms are subjective, and the model may therefore not accurately describe the range of physical manifestations of the yips. Two other studies report kinematic and electromyographic data, which indicate that golfers affected by yips have higher levels of forearm-muscle activation and exert greater grip force on the putter than unaffected golfers (1,34). However, neither categorized the yips-affected golfers as Type I or Type II, using the model proposed by Smith et al. (33), and both had small sample sizes (10 and 4 yips-affected golfers, respectively). Although these studies provide objective evidence of the nature of yips symptoms, a systematic exploration of the model described by Smith et al. (33) has yet to be conducted.

The purpose of the present study was to test the model put forward by Smith et al. (33), which describes the underlying causes of the yips as a continuum between focal dystonia and choking. As described by Smith et al. (33), golfers with yips were categorized as Type I or Type II, according to their descriptions of their symptoms. Based on this categorization of yips-affected golfers, our general predictions were that Type I golfers would differ from Type II and control golfers on physiological measures of muscle activity and behavioral measures that are sensitive to focal dystonia. We also predicted that Type II golfers would differ from Type I and control golfers on measures of anxiety and that their putting performance would be more adversely affected by experimental conditions that heighten performance anxiety. To test these general predictions, two experiments were conducted, as described below.

In experiment 1, we measured a range of psychological, physiological, and performance variables in golfers with and without yips symptoms as they performed a putting task under two experimental conditions. One condition was designed to minimize performance anxiety, and the other was designed to increase it. The high-pressure experimental condition included the use of a monetary reward for accurate putting performance, videotaping participants as they putted, and the presence of a confederate who was identified as a sport psychologist specializing in elite golf performance. The monetary reward started at \$75 and was reduced by \$5 for every putt that was missed. Most participants missed more than 15 of the 40 putts performed under the high-pressure condition, which removed the monetary reward for the remainder of the putts under this condition. For experiment 1, we hypothesized that

1. Type I golfers would exhibit greater levels of upper-limb muscle activation during putting than Type II and control golfers.
2. Type II golfers’ performance would be impaired to a greater extent under the high-pressure experimental condition than would putting performance of the Type I and control golfers.
3. Putting performance would be more accurate once the possibility of monetary reward was removed, for all groups.
4. Type II golfers would have higher cognitive-state anxiety scores than Type I and control golfers.

Experiment 2 was also designed to explore the model put forward by Smith et al. (33) by using a behavioral measure that is sensitive to focal hand dystonia. Our general prediction was that the performance of this task by Type I golfers would be significantly different from that of Type II and control golfers. Previous work in our laboratory has demonstrated that focal-hand dystonia is associated with an impaired ability to inhibit a planned response (36). Briefly, participants observe a clock face that has a sweep hand, which completes one revolution in 1 s. Their task is to stop the sweep hand at a target point in its revolution by releasing a key (32). On some trials, the sweep hand stops before the target, in which case the participant should not release the key, that is, inhibit the anticipated response. As the sweep hand stops closer to the target, participants are more likely to initiate the response and inadvertently release the key. Even when dystonic participants successfully inhibit their response and keep the key depressed, they are more likely to exhibit a partial burst of EMG activity in the agonist muscle (36). This indicates that they are less effective at preventing unwanted muscle activation. This task is therefore a useful behavioral measure of inhibitory function, which is sensitive to dystonia. All participants performed this task in a second experiment. For experiment 2, we hypothesized that on successful trials, Type I golfers would exhibit more partial EMG bursts than Type II and control golfers.

If the evidence from these two experiments supports their respective hypotheses, it will lend general support to the continuum model proposed by Smith et al. (33).

EXPERIMENT 1

Method

Participants. Twenty-two male golfers and two female golfers volunteered for this study. All golfers except one were right-handed by self-report, and age ranged from 18 to 75 yr (mean age 51.8 yr). Nine reported no problems with putting, and the remaining 15 reported yips-like symptoms while putting. The mean handicap of all participants at the time of testing was 11.45 (range 0–29). All golfers gave their written informed consent in accordance with the Declaration of Helsinki.

Participants who reported no difficulties with putting were assigned to the control group. As described by Smith et al. (33), golfers with yips were categorized as Type I or Type II, according to their descriptions of their symptoms. Specifically, participants who reported putting difficulties related to muscle tension and/or uncontrolled movement were assigned to the Type I yips group, whereas those who reported difficulties related to stress and/or anxiety were assigned to the Type II yips group. The golfers who had experienced problems with putting reported having tried a number of strategies to overcome these. Their strategies included relaxation (four golfers), exercise (three), listening to music (one), slow breathing (one), consulting a sport psychologist (two), changing their grip (five), changing their stance and/or setup (five), and changing to a putter of a different length (one). None of the golfers who had experienced problems during putting had used medications to relieve their symptoms.

Physiological measure (electromyography). Surface electromyography (EMG) was recorded from the flexor carpi radialis (FCR), extensor carpi radialis (ECR), and biceps brachii (BB) bilaterally, using 10-mm-diameter surface Ag/AgCl Hydrosport electrodes (Physiometrix, North Billerica, MA) and following standard skin-preparation techniques. Ground electrodes were positioned over the lateral epicondyle of each humerus. Signals were amplified using Grass P511AC amplifiers (Grass Instrument Division, West Warwick, RI). The EMG data were bandpass filtered at 30–1000 Hz, sampled at 1 kHz with a 16-bit National Instruments A/D acquisition system and software (National Instruments, Austin, TX), and stored for offline analysis. Two maximum voluntary contractions (MVC) were recorded from each muscle under isometric conditions. The larger peak root mean square (rms) value of the two recordings from each muscle was used to normalize subsequent recordings during putting.

Performance measure (putting task). Participants were given the opportunity to practice putting on the artificial surface using their own putter. The artificial putting surface (Proline Sporting Products Pty Ltd.) had four holes (numbered 1–4) and six traps. Participants were instructed that for a putt to be considered successful, the ball had to enter and remain in the experimenter-identified hole. A custom-built optical trigger was embedded in the putting surface. It triggered the EMG collection system to record a single trial when the ball was putted off it. The

trigger was flush with the artificial surface and provided a standard starting position for each putt. The distance from the trigger to the holes ranged from 2.2 to 2.5 m. For each putt, EMG data were collected for 4 s, with 3 s pretrigger and 1 s posttrigger (Fig. 1).

Psychological measure (state anxiety). Before the start of the putting task, participants completed the Competitive State Anxiety Inventory (26). The CSAI-2 is a multidimensional state scale measuring respondents' feelings and thoughts about competition at a given point in time. The scale takes approximately 5 min to complete and consists of three nine-item subscales that assess cognitive anxiety (the immediate conscious awareness of unpleasant feelings (worry) about oneself or external stimuli), somatic anxiety (the immediate awareness of bodily symptoms of the autonomic nervous system, such as heart rate, shortness of breath, tense muscles, or clammy hands), and self-confidence (the degree of certainty that athletes feel about their ability to be successful). Each CSAI-2 item is rated on a four-point scale anchored at extremes by not at all (1) and extremely (4). Large scores indicate higher levels of state anxiety and self-confidence. Studies investigating the psychometric properties of the CSAI-2 have supported the three subscales (e.g., Cronbach's alpha coefficients range between 0.79 and 0.90) as sport-specific measures of state anxiety (26).

Procedure. Once participants had practiced the golf task, they were informed of the experimental procedure. Participants experienced either the high- or low-pressure condition first, and the order was counterbalanced across participants. In the low-pressure condition, participants were simply told that a researcher would tell them which of

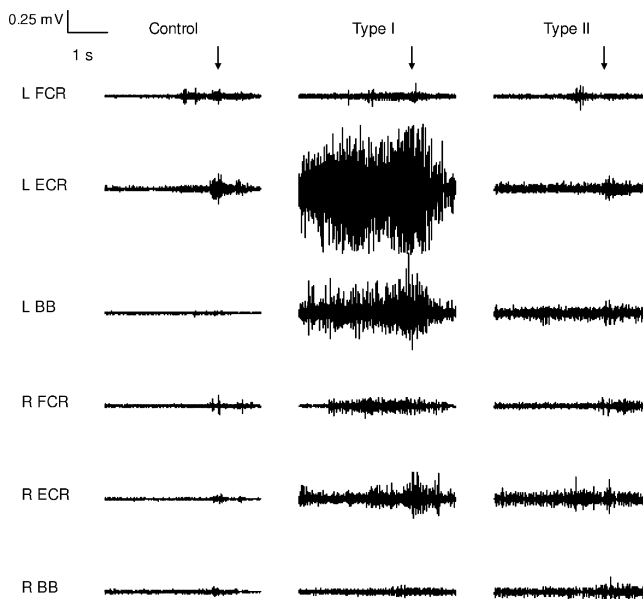


FIGURE 1—Typical EMG traces from flexor carpi radialis (FCR), extensor carpi radialis (ECR), and biceps brachii (BB) of the left (L) and right (R) arms, during single putts performed by control, Type I, and Type II yips golfers. The arrow indicates the time at which the ball was putted off the optical trigger embedded in the putting surface. Type I yips golfers typically exhibited greater levels of muscle activity both before and during each putt.

the four holes to putt toward each time, but that their accuracy would not be recorded. Only two experimenters were in the room during testing, one running the EMG collection system and the other indicating which hole to putt toward on each trial.

For the high-pressure condition, a video camera was positioned at the hole end of the putting surface to record all of the putts. Participants were told that this video would be reviewed by a biomechanics expert and used for teaching purposes. Participants were also introduced to a confederate and told that this person was an expert in sport psychology, with a particular interest in elite golf performance. The confederate told participants which of the four holes to putt toward each time and kept careful track of their accuracy. The confederate also placed 15 five-dollar bills on a table near the hole end of the putting surface and explained to the participant that each time they missed a putt, \$5 would be removed from the stack. The confederate also informed each participant that previous golfers had typically walked away with "about \$50" and that a similar performance was expected from them. The confederate provided negative feedback to each participant by specifically commenting on their missed putts and by refraining from commenting when their putts were successful. Previous researchers have successfully used financial inducements, audiences, and video cameras as pressure manipulations to induce competitive pressure (19,24). Although we have labeled the baseline condition "low pressure," we do not mean to imply low pressure in an absolute sense, but rather in a comparative sense.

In the first session, each participant completed the CSAI-2 questionnaire and then 40 putts under either the high- or the low-pressure condition. These 40 putts consisted of 10 putts to each of the four holes, in a pseudorandomized order. After 40 putts, the participant was asked to complete a golf history questionnaire based on the one used in previous yips research (33). The questionnaire took around 15 min to complete. Forty putts were then completed in a second session under the alternate condition (low or high pressure). When the first session was under the high-pressure condition, the low-pressure condition was established by removing the video camera and explaining to the participant that more EMG data were required but that their accuracy would no longer be recorded. Where the low-pressure condition was experienced first, the high-pressure condition was established by bringing the video camera into the room and introducing the confederate and financial reward (as described above). Participants completed the CSAI-2 questionnaire again once the conditions for the second session were established, as it related to the current putting task under the second condition. Participants who missed fewer than 15 of the 40 putts performed under the high-pressure condition were given the remaining cash at the end of that session. Putting accuracy for the low-pressure condition was recorded covertly to enable comparison of performance between the high- and low-pressure conditions.

Data Analysis

Putting task. EMG data were processed using custom software. Each EMG trace was rectified, the trials were averaged for each muscle under each condition, and a 5-Hz Butterworth filter was applied. The rms EMG values were calculated over 200-ms time bins for the duration of the trial. EMG values were expressed as a percentage of the rms EMG during the MVC. Baseline EMG was calculated as the average EMG activity during a 1200-ms period ending 1600 ms before contact with the ball. Peak EMG activity was calculated as the maximum EMG value achieved in the averaged EMG trace, for each muscle under each condition, for each individual.

Statistical Analysis

One-way analyses of variance (ANOVA) were used to test for differences between the three groups of golfers in age, present handicap, years spent playing golf, and current number of golf rounds played per year. Independent two-tailed *t*-tests were used to test for differences between the two yips groups in yips duration and handicap at onset of yips-like symptoms. Putting accuracy in the low- and high-pressure conditions was analyzed using a 2 (condition; high pressure, lowpressure) \times 3 (group; Type I, Type II, control) repeated-measures ANOVA. Putting accuracy while the monetary reward was available (pre) and after the money ran out (post) was analyzed using a 2 (time; pre, post) \times 3 (group; Type I, Type II, control) repeated-measures ANOVA. One-tailed paired sample *t*-tests were conducted comparing putting accuracy before and after the monetary reward ran out for each group, to test the hypothesis that the removal of the monetary reward would lower performance anxiety and improve performance.

For the CSAI-2 scale, a 2 (condition; high pressure, low pressure) \times 3 (group; Type I, Type II, control) repeated-measures ANOVA was used for each of the three state subscales (cognitive, somatic, and self-confidence). One-tailed paired sample *t*-tests were conducted for cognitive anxiety values comparing low- and high-pressure conditions for each group.

For baseline and peak EMG values, a 3 (group; Type I, Type II, control) \times 2 (condition; low pressure, high pressure) \times 3 (muscle; ECR, FCR, BB) \times 2 (side; left, right) repeated-measures ANOVA was conducted. For all statistical analysis, a significance level of $\alpha = 0.05$ was adopted.

Results

Participant characteristics. Participant characteristics are presented in Table 1. One-way ANOVAs revealed that the groups differed significantly in age ($F_2 = 4.26$, $P < 0.05$) and years spent playing golf ($F_2 = 3.54$, $P < 0.05$). Subsequent analysis with independent two-tailed *t*-tests revealed the Type I golfers were significantly older than the control golfers (t_{15} , $P < 0.01$). There was no significant difference in age between the two yips groups (t_{13} , $P > 0.3$). Type I golfers had also spent a greater number of years

TABLE 1. Participant characteristics: age, number of years spent playing golf, rounds of golf currently played per year, duration of yips symptoms, present handicap, and handicap at onset of yips symptoms.

	Control (N = 9)	Type I (N = 8)	Type II (N = 7)
Age			
Mean	39.6*	61.8*	54.0
SD	19.3	9.1	17.3
Range	18–64	51–75	25–69
Years playing			
Mean	21.6*	39.3*	23.4*
Range	5–48	25–56	10–45
Rounds per year			
Mean		5.6	10.6
Range		1–20	0.2–45
Handicap			
Mean	6.8	13.9	13.6
SD	7.0	9.0	9.3
Handicap at onset			
Mean		11.8	14.6
SD		4.5	7.3

One-way ANOVA and independent two-tailed *t*-tests revealed a significant difference in age between Type I and control groups (* $P < 0.05$), with no significant difference between Type I and Type II groups. This analysis also revealed significant differences in the number of years spent playing golf between Type I and Type II groups and between Type I and control groups (* $P < 0.05$). There were no significant differences between yips groups for any of the other variables (all P values > 0.3).

playing golf than the Type II golfers (t_{13} , $P < 0.05$) and control golfers (t_{15} , $P < 0.05$). There were no significant differences between the three groups of golfers in their present handicap or in the number of rounds of golf played per year. There were no significant differences between the two yips groups on duration of symptoms, present handicap, or handicap at onset of symptoms (all $P > 0.05$).

Putting accuracy. Putting accuracy as a percentage of total putts attempted is presented in Table 2 for high- and low-pressure conditions and for before and after the monetary reward ran out. There was no significant effect of condition (high pressure, low pressure) on putting accuracy. There was a significant main effect of the monetary reward on putting accuracy ($F_{1,13} = 12.26$, $P < 0.01$). One-tailed paired sample *t*-tests revealed that putting accuracy was significantly higher after the monetary reward ran out for the control group and Type II group but not for the Type I group (control t_8 , $P < 0.05$; Type II t_6 , $P < 0.05$; Type I t_7 , $P = 0.09$).

State anxiety. The mean scores on each anxiety subscale for each group are presented in Table 3. There was a significant main effect of condition (high pressure, low pressure) on the cognitive anxiety scores ($F_{1,23} = 8.870$, $P < 0.01$). No significant effects were found for somatic anxiety ($P = 0.10$) or self-confidence ($P = 0.07$). One-tailed paired sample *t*-tests revealed significant differences between cognitive anxiety values under low- and high-pressure conditions for control and Type I groups (control t_8 , $P < 0.05$; Type I t_7 , $P < 0.05$) but not for the Type II group (t_6 , $P > 0.05$).

EMG. Mean baseline rms EMG and peak rms EMG values (% MVC) for each muscle (FCR, ECR, BB) from each arm (right, left) under each condition (high pressure, low pressure) are presented in Figure 2. The ANOVA for baseline EMG values revealed significant effects of arm ($F_{1,20} = 7.727$, $P < 0.05$) and muscle ($F_{2,20} = 5.990$, $P < 0.05$). These effects arose because the muscles of the left arm were more active than those of the right and because the forearm extensors were more active than the flexors or biceps. A significant

interaction arose between muscle and group ($F_{4,20} = 2.615$, $P < 0.05$) because the two yips groups exhibited greater activity in the forearm extensors than the flexors or biceps, whereas the control group did not (control: FCR 0.28 ± 0.07 , ECR 0.58 ± 0.26 , BB 0.74 ± 0.18 ; Type I: FCR 0.46 ± 0.07 , ECR 1.05 ± 0.16 , BB 0.51 ± 0.18 ; Type II: FCR 0.57 ± 0.08 , ECR 1.02 ± 0.28 , BB $0.39 \pm 0.20\%$ MVC). For all groups, forearm-flexor activity was greater during the high-pressure condition than the low-pressure condition, producing a significant interaction ($F_{2,20} = 4.051$, $P < 0.05$).

The ANOVA for peak EMG values revealed a significant effect of muscle, which was attributable to the peak activity being highest for forearm extensors, followed by forearm flexors and biceps brachii ($F_{2,17} = 12.633$, $P < 0.01$). A significant interaction arose between condition, arm, and group ($F_{2,17} = 3.796$, $P < 0.05$). For the control group, muscle activity was equivalent in both arms under both conditions (low-pressure left 1.16 ± 0.21 , right $1.12 \pm 0.30\%$ MVC, t_7 , $P > 0.8$; high-pressure left 1.30 ± 0.23 , right $1.21 \pm 0.28\%$ MVC, t_7 , $P > 0.7$). Similarly, for the Type II group, muscle activity did not differ significantly between the arms under both conditions (low-pressure left 1.37 ± 0.34 , right $1.14 \pm 0.25\%$ MVC, t_6 , $P > 0.4$; high-pressure left 1.44 ± 0.33 , right $1.08 \pm 0.27\%$ MVC, t_6 , $P > 0.3$). The Type I group exhibited higher levels of peak muscle activity than the control group (t_7 , $P < 0.05$). In particular, under the low-pressure condition, left-arm peak muscle activity was greater in Type I golfers than in control golfers (Type I, 1.97 ± 0.34 ; control, $1.16 \pm 0.21\%$ MVC; t_7 , $P < 0.05$).

Discussion

The first hypothesis for this study was that Type I golfers would exhibit greater levels of upper-limb muscle activation during putting than Type II and control golfers. The EMG data paint a more complex picture in that both yips groups exhibited higher overall levels of baseline ECR muscle activity than the control golfers (Figs. 1 and 2). Peak muscle activity was equivalent in both arms under both low- and high-pressure conditions for the control and Type II golfers. However, under the low-pressure condition, peak muscle activity in the left arm was significantly higher in the Type I golfers than the control golfers. This suggests that even when putting under less stressful conditions, Type I golfers continue to exhibit abnormally higher levels of muscle activity, particularly in the left arm (top hand). Because all except one of the volunteers for this study were right-handed, the pattern of muscle activity in left-handed golfers with yips symptoms remains to be determined.

TABLE 2. Mean (SD) putting accuracy as a percentage of total putts attempted under each condition (high pressure, low pressure) and before and after the monetary reward ran out during the high-pressure condition (pre, post).

	Putting Accuracy (%)			
	High Pressure	Low Pressure	Pre	Post
Control	54 (17)	58 (14)	42 (10)*	51 (13)*
Type I	47 (17)	47 (10)	29 (16)	42 (11)
Type II	50 (22)	52 (14)	31 (24)*	54 (27)*

* Significant difference ($P < 0.05$) in accuracy before and after.

TABLE 3. Mean (SD) scores for each anxiety subtype under each condition (high pressure, low pressure).

	Cognitive/36		Somatic/36		Self-Confidence/36	
	High Pressure	Low Pressure	High Pressure	Low Pressure	High Pressure	Low Pressure
Control	16.4 (4.7)*	13.2 (3.4)*	14.2 (4.3)	12.2 (2.4)	25.6 (5.1)	28.0 (5.4)
Type I	18.3 (5.7)*	16.8 (4.4)*	13.5 (4.1)	12.0 (1.4)	24.0 (6.5)	23.0 (6.5)
Type II	17.7 (4.6)	16.0 (7.0)	14.3 (1.7)	13.7 (4.5)	22.4 (5.9)	25.1 (7.7)

Higher values for cognitive and somatic subtypes and lower values for self-confidence indicate increased state of anxiety. * Significant difference ($P < 0.05$) between high- and low-pressure conditions.

The second hypothesis was that Type II golfers' performance would be impaired to a greater extent under the high-pressure experimental condition than that of the Type I and control golfers. Although there was no significant effect of experimental condition, Type II golfers' performance was significantly better in the absence of a monetary reward. This finding supports our third hypothesis (Table 2).

The fourth hypothesis was that Type II golfers would have higher cognitive-state anxiety scores than Type I and control golfers. Although Type II golfers' cognitive anxiety scores were not significantly higher than those of the other two groups, they were not affected by experimental condition (low pressure, high pressure). This is in contrast to the cognitive anxiety scores of the other two groups, which were significantly higher under the high-pressure condition (Table 3). The outcomes of experiment 1 are discussed in context with those of experiment 2 in the General Discussion below.

EXPERIMENT 2

Method

Participants. The participants were the same group who had participated in experiment 1, detailed above.

Behavioral measure (anticipated response task)

and procedure. Participants were seated in front of a personal computer with the distal aspect of their dominant index finger positioned over the "0" button of a raised keyboard (pointing posture). The forearm rested on the table and was positioned midway between pronation and supination and cushioned with a foam support. Surface EMG was recorded from the first dorsal interosseus (FDI) muscle of the participant's dominant hand using 10-mm-diameter surface Ag/AgCl Hydrosport electrodes (Physiometrix, North Billerica, MA) and following standard skin-preparation techniques. A ground electrode was positioned over the lateral epicondyle. EMG was acquired as in the putting task and stored for offline analysis.

The participants' ability to inhibit a prepared action was tested using a custom-built LabVIEW program (National Instruments, Austin, TX) based on a paradigm first used by Slater-Hammel (32). Participants viewed a circular analog sweep dial. Participants were instructed to press a key with their index finger, using only as much force as was needed to maintain the key in its depressed state. One second later, the sweep indicator began its clockwise revolution, which took 1 s to complete a full revolution. Participants were

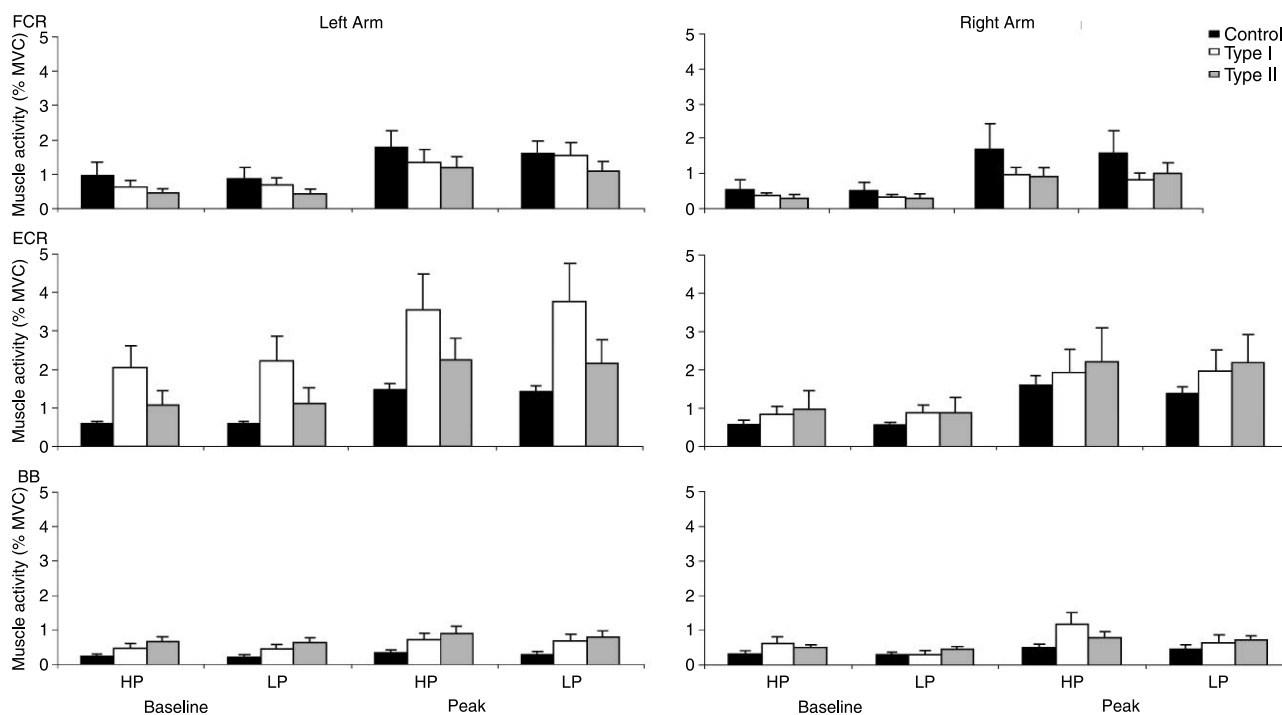


FIGURE 2—Baseline and peak EMG activity expressed as a percentage of maximum voluntary contraction (MVC) for each arm (left, right) under high-pressure (HP) and low-pressure (LP) conditions. FCR, flexor carpi radialis; ECR, extensor carpi radialis; BB, biceps brachii. Filled bars, control group; white bars, Type I; gray bars, Type II; error bars, standard error.

instructed that lifting their finger off the key would stop the sweep indicator. Their task was to lift their finger from the key to stop the indicator 800 ms after the beginning of the sweep, at a position identified with an arrow. These trials are referred to as “go” trials. The software provided participants with instant feedback of their performance, indicating the time, in milliseconds, that they released the key relative to the “8.” Participants were instructed to perform this task as accurately as possible.

Each participant observed a demonstration and then completed a series of practice trials to become familiar with the protocol. After several practice trials, they were informed that on a proportion of trials, the sweep indicator might stop on its own before reaching the 8 and that for these trials, they were to not to lift their finger from the key. These trials are referred to as “stop” trials. If the sweep indicator were stopped late in the movement preparation period, the participant would sometimes inadvertently lift their finger. Participants were instructed that this might occur and were informed to do their best to keep the key depressed but not to be too concerned if they inadvertently lifted their finger. This was reinforced by the feedback given on completion of the trial. If they did not lift their finger from the key, the feedback stated “response inhibited.” If they did lift their finger from the key, the feedback stated “the clock hand stopped too late for the response to be inhibited.”

The experiment consisted of nine blocks, each consisting of 30 trials. Within each block, both go and stop trials were presented in a randomized order. Feedback, as above, was presented at the end of the block for performance on go trials only. There were 270 trials in total, 180 of which were go trials and 90 of which were stop trials. For the stop trials, the sweep indicator stopped 500, 525, 550, 575, 600, 625, 650, 675, and 700 ms after the start of its sweep, with a total of 10 trials for each stop time randomly presented during the course of the experiment. The EMG collection system was triggered by the start of each trial and recorded data for 1.2 s.

Data Analysis

Data were analyzed using custom LabView software (National Instruments, Austin, TX). The EMG burst onsets in the FDI muscle were defined as the time when the rms EMG first deviated by more than five standard deviations above baseline rms EMG (300–500 ms from start of trial). The experimenter was blinded to whether each trial was a go or stop trial and to the time at which the sweep dial stopped.

TABLE 4. “Go” task performance.

	Control		Type I		Type II	
	Mean (ms)	SE	Mean (ms)	SE	Mean (ms)	SE
Lift time	812.8	2.5	824.2	2.2	816.4	3.8
Burst onset	703.4	8.3	696.7	17.2	707.4	9.3
Mean error	12.8	2.5	24.2*	2.2	16.4	3.8
Absolute error	28.1	2.2	38.7*	1.7	35.2	2.5
Variable error	32.4	2.6	42.7*	2.0	40.6	2.3

* Significant difference relative to control group.

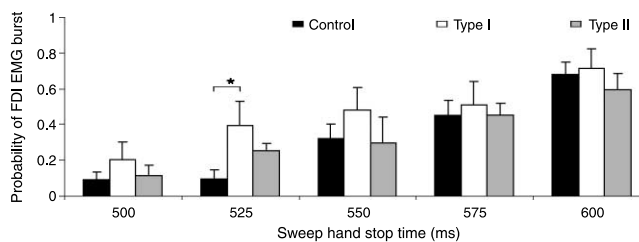


FIGURE 3—Probability of EMG burst in prime mover (FDI) during a successfully inhibited response as a function of sweep-dial stop time (ms). * Significant difference ($P < 0.05$) between the control and the Type I yips group. Error bars, standard error.

Trials were then separated into go and stop trials. For go trials, the mean EMG burst onset and lift time were calculated for each participant along with the mean error, absolute error, and variable error. The mean error is the mean of the deviations from the specified lift time, the absolute error is the mean of the absolute deviations from the specified lift time, and the variable error is one standard deviation of the distribution of lift times. Stop trials were sorted by stop time and then according to whether the response had been successfully inhibited. For stop trials, the probability of a partial response was determined as a function of stop time. A partial response is defined as a burst of EMG in the prime mover (FDI) in the absence of an overt behavioral response (i.e., a burst of EMG on trials when the response was successfully inhibited). The probability of a partial response was only analyzed for stop times if the probability of inhibiting a response was greater than 50%. Thus, the probability of a partial response was only analyzed for the first five stop times.

Statistical Analysis

For go trial performance, the dependent variables of mean error, absolute error, and variable error were analyzed using one-way ANOVA with the factor group (control, Type I, Type II). Where main effects were found, *post hoc* comparisons were made, with a Bonferroni correction for multiple comparisons.

For stop trial performance, the probability of a partial response was analyzed with a mixed ANOVA with a between-subjects factor of group (control, Type I, Type II) and a within-subject factor of time (five levels). A planned contrast of the partial-response probability for the control and Type I group was conducted.

Results

A summary of go trial performance is provided in Table 4. The one-way ANOVA revealed a significant main effect of group on mean error ($F_{2,19} = 4.6, P = 0.02$), absolute error ($F_{2,19} = 6.8, P < 0.01$), and variable error ($F_{2,19} = 5.4, P = 0.01$). *Post hoc* comparisons revealed that the Type I yips group had significantly larger error values relative to controls (mean error $t_{12}, P < 0.05$; absolute error $t_{12}, P < 0.01$; variable error $t_{12}, P < 0.05$). No other comparisons were significant.

The probability of a partial response is shown in Figure 3. The ANOVA revealed a main effect of time ($F_{4,76}$, $P < 0.001$). Planned contrasts revealed a significantly greater probability of a partial response in the Type I yips group relative to controls at the 525-ms stop time (t_{12} , $P < 0.05$). No other planned contrasts were significant.

Discussion

The final hypothesis for this study was that Type I golfers would exhibit more partial EMG bursts when successfully inhibiting a prepared action than Type II and control golfers. This hypothesis was partially supported in that the number of partial responses exhibited by Type I golfers was significantly higher than that observed in control golfers (Fig. 3).

GENERAL DISCUSSION

The purpose of this study was to determine whether the classification of yips into Type I and Type II as described by Smith et al. (33) is supported by a range of physiological, behavioral, psychological, and performance measures. In general, the results of experiments 1 and 2 lend support to this model.

The overall findings for Type I golfers can be summarized as follows. This group exhibited higher levels of muscle activity in the extensors of the left forearm (top hand) in the baseline period immediately before the putt compared with the control group. Peak activity in the left forearm extensors was also higher than control golfers and was less sensitive to the experimental conditions (low pressure, high pressure) than for the Type II golfers. These golfers also exhibited more variable performance and a higher likelihood of initiating an undesired response on an anticipated response task. These findings are similar to the deficits observed in people with focal hand dystonia performing this task (36). Type I golfers' putting performance was unaffected by the presence or absence of a monetary reward, and their cognitive anxiety increased in response to the high-pressure condition, as did the control golfers'. Together, these findings suggest that the putting difficulties experienced by Type I golfers are related to impaired initiation and execution of movement rather than factors related to performance anxiety.

In contrast, participants classified as Type II golfers were indistinguishable from control golfers in their performance of the anticipated response task and their ability to inhibit an undesired response. Rather, they exhibited impaired putting accuracy in the presence of a monetary reward. Unlike the control and Type I groups, the Type II golfers' cognitive anxiety scores were insensitive to environmental manipulations designed to increase stress. This may reflect a greater contribution of self-awareness and internally generated performance pressure to their anxiety levels. Together, these findings suggest that the putting difficulties experienced by Type II golfers are related to performance anxiety rather than to factors related to the neural control of movement.

In the present study, the Type I group had more years of playing experience than members of the Type II group and the control group. The Type I group tended to be older and to have experienced yips for fewer years than the Type II group. They also had generally lower overall putting accuracy, under all experimental conditions, than the Type II group. This is consistent with a recent study by Adler et al. (1), who found that yips-affected golfers who exhibited cocontraction of the forearm musculature during putting (Type I yips) tended to be older, to make more putting errors, and to have a more recent onset of symptoms than those who did not exhibit cocontraction. Together, these two studies suggest that Type I yips is associated with abnormal and excessive muscle activity and is more likely to affect older golfers with a more rapid and detrimental effect on performance. This raises the hypothesis that aging-related dopamine depletion may play a role in the development of Type I yips. Basal ganglia dysfunction is thought to be the pathophysiological basis of dystonia, including the focal dystonias (4). Furthermore, an early-onset hereditary form of dystonia (dopa-responsive dystonia, DRD) is caused by a genetic disturbance that results in deficient dopamine production (18,31). Although levodopa is effective for alleviating the symptoms of DRD (31), evidence of dopamine depletion in Type I golfers would need to be established before the potential for treatment with levodopa could be considered.

Differences in symptom presentation and course of the yips may help to distinguish between the two subtypes, enabling more appropriate management. The symptoms of Type II yips are more likely to respond to treatment strategies that focus on the underlying causes of performance anxiety than those that attempt to manage the outward motor symptoms. In addition to anxiety, emotions have an impact on the performance of motor tasks. Aversive stimuli are associated with greater extensor activity and increased muscle force (16), and appetitive stimuli are associated with greater flexion (9). Indeed, it has been hypothesized that focal dystonia is insensitive to treatment, including retraining, because of the coupling between emotional memory involving the basal ganglia and the frontostriatal system and the motor memories that interact with the central and peripheral nervous system (25). Although emotion is an important aspect of movement, a study of emotion was beyond the scope of this study. The Smith model (33) does not specifically include the interrelatedness of emotion and movement. Future research addressing this issue through theoretical frameworks such as Lang's biphasic theory of emotion (21,22) is warranted.

The management of Type I yips could draw from the treatments used for occupational dystonias, such as writer's cramp and musician's cramp. A range of therapeutic strategies have been trialed for these types of dystonia, including immobilization of the affected hand or upper limb (10,28), often combined with ergonomic changes (20), motor training (11,41), and sensory training (7,40). These types of interventions have produced mixed results in people with writer's and musician's cramp and would require a considerable time commitment from the golfer, under the

guidance of a trained physical therapist. If the overactive forearm muscles can be clearly identified in an individual, injection of botulinum toxin can markedly reduce the level of involuntary activity (5,6,15). However, this treatment also carries the risk of the toxin spreading to adjacent musculature that is not intended for treatment (29), and the effects of treatment are temporary, lasting approximately 3–4 months. Other symptomatic treatments include oral medications (e.g., trihexyphenidyl) and exercise (8,20).

Although these findings lend support to the model of the yips proposed by Smith et al. (33), the present study does have some limitations. First, the putting accuracy of all three groups of golfers was around 50%. This suggests that the putting task may have been quite difficult and, therefore, less sensitive to between-group differences in performance attributable to a floor effect. Second, the control group of golfers tended to be younger and to have a lower handicap than the two yips groups. This is to be expected, given that the yips takes a number of years to develop and to compromise performance, and is consistent with previous reports (27). The Type I golfers had significantly more years of golf experience than both the Type II golfers and the control golfers. Given that the Type I golfers also exhibited (nonsignificantly) lower levels of putting accuracy, it seems that their additional

experience has conferred no performance advantage. Furthermore, there were no significant differences between groups on current handicap. Therefore, it seems that despite the control group being significantly younger, the groups were adequately matched for skill level. Differences in skill level probably do not explain the between-group differences in the physiological, psychological, and behavioral variables. Third, the model proposed by Smith et al. (33) identified an intermediate group who had experienced a mixture of Type I and Type II symptoms. The characterization of this group was beyond the scope of the present study, but it should form part of future research. Finally, further work is required to determine how the stressors encountered in a genuine game context interact with the factors underlying Type I and Type II yips.

The present study provides evidence in support of the model proposed by Smith et al. (33). Classification of golfers with the yips, using their description of their symptoms, could provide a useful framework for the appropriate management of yips symptoms.

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