Abnormal co-contraction in yips-affected but not unaffected golfers: Evidence for focal dystonia

C.H. Adler, MD, PhD; D. Crews, PhD; J.G. Hentz, MS; A.M. Smith, PhD; and J.N. Caviness, MD

Up to 30% of golfers develop the yips, an inability to complete a golf stroke, most often affecting short putts, which worsens with anxiety.[1,2] Yips may be organic (task-specific dystonia) or psychological (anxiety or "choking").[2-4] We previously found abnormal trains of 4 to 8 Hz, rhythmic, co-contracting bursts of EMG activity in arm muscles of three golfers suggesting a movement disorder. This led to the current investigation.

Methods. We studied 20 age- and handicap-matched male right-handed golfers, 10 with the yips and 10 without. Handicap is the golfer's average score over par over the past ~10 rounds of golf. Surface EMG electrodes were placed bilaterally on the pectoralis major, deltoid, biceps, triceps, wrist flexors, pronator teres, flexor pollicis longis, wrist extensors, abductor pollicis brevis, and abductor digiti minimi. EEG electrodes were in a standard montage. Recordings were made at a sampling rate of 1,000 Hz, bandpass 1 to 200 Hz using the Neuroscan system (Neuroscan Compumedics, El Paso, TX). Investigators were not blinded. Pre-putting conditions at rest and with arm activation maneuvers, including writing, were recorded. Somatosensory evoked potentials (SEP) were performed by median nerve stimulation at 2.2 Hz using an average ear reference.

Putting was performed on an indoor, artificial, flat putting green surface that was 12 feet long with a hole 2 feet from one end. Subjects were evaluated standing with arms relaxed with and without holding the putter. They then performed 75 putts: 30 putts of 6 feet, 10 putts of 3 feet, and 35 putts of 8 feet. The number of putts made and distance from the hole were recorded for each putt. An electronic photocell recorded the initiation of each stroke and the point of impact.

EEG-EMG polygraphy was assessed for abnormal activation of muscles including oscillating discharges, co-contractions, and high-amplitude short duration discharges. The N30 amplitude was measured from the averaged SEP at Fz and Cz. Rectification of EMG activity for all leads was performed 6 seconds before the first putter movement, through to at least 2 seconds after the ball was hit.

Results. There was no difference in age or current handicap, but yips-affected golfers had better "best" handicaps and had been golfing for longer than yips-unaffected golfers (table). Prior to putting no abnormal movements were found. During putting two yips-affected golfers reported feeling the yips, yet all yips-affected golfers appeared to have a visible jerk or twist of the wrist or forearms. There were no differences between groups in right or left forearm EMG activity being dominant, wrist flexor vs wrist extensor activity being dominant, or whether there was phasic flexor or extensor bursting. At 200 msec prior to impact of the putter with the ball there was co-contraction of wrist flexor and extensor muscles in 5/10 of the yips-affected golfers (in all putting conditions) and 0/10 of the yips-unaffected golfers ($p = 0.06$, exact McNemar test). The rest of the golfers had either no specific pattern or had reciprocal wrist flexor and extensor activity (6/10 yips-unaffected vs 3/10 yips-affected). SEP data revealed significant (one electrode) or trend (two electrodes) to higher amplitude N30 waves in the yips-affected group (see table).

There was a trend for yips-affected co-contracting golfers to be older, have higher handicaps, have yips for fewer years, make fewer putts, and have a greater degree of error in missing the putts than the non-co-contractors.

Discussion. Electrophysiologic analysis of age- and handicap-matched yips-affected vs yips-unaffected golfers found 5/10 yips-affected and 0/10 yips-unaffected golfers have wrist flexor/extensor co-contractions, even with just two of the yips-affected golfers having symptoms during testing. As co-contraction is a hallmark of dystonia,[5] the yips appears to be a movement disorder, such as a task-specific dystonia, in some golfers. The SEP results may also support the yips being a focal dystonia.[6]

Other explanations for the co-contraction might include another abnormal movement physiology or a phenomena called "double pull" in which agonists and antagonists apparently co-contract when under conditions of high arousal (although not studied with electrophysiology). As the co-contracting golfers golfed longer and had lower handicaps they were likely not anxious and 3/5 lacked a subjective feeling of the yips.

Limitations in this study include its small size, laboratory and not golf course location, and unblinded data collection and analysis. Despite these limitations, the results suggest some cases of

Table *Comparison of yips-affected and unaffected golfers (paired t test)*

Variable	No.	Unaffected	Affected	Difference	*p*	95% CI
Age	10	49.30 (17.85)	50.30 (14.91)	1.00 (3.77)	0.42	−1.70 to 3.70
Years golfing	10	25.90 (16.99)	37.60 (12.37)	11.70 (14.58)	0.03	1.27 to 22.13
Handicap	10	7.80 (6.44)	6.60 (6.28)	−1.20 (3.58)	0.32	−3.76 to 1.36
Best handicap	10	6.30 (5.54)	3.50 (4.86)	−2.80 (2.62)	0.01	−4.67 to −0.93
SEPs						
N20L	8	2.07 (0.38)	2.44 (1.02)	0.37 (1.08)	0.36	−0.53 to 1.27
N20R	8	2.11 (0.70)	2.47 (0.64)	0.36 (1.04)	0.36	−0.51 to 1.24
P25L	8	1.84 (1.30)	2.60 (1.11)	0.76 (1.05)	0.08	−0.12 to 1.64
P25R	8	2.19 (1.41)	2.80 (1.06)	0.61 (1.77)	0.36	−0.87 to 2.09
FzL	8	1.72 (0.50)	2.82 (1.34)	1.11 (1.39)	0.06	−0.06 to 2.27
FzR	8	1.70 (0.83)	2.29 (0.91)	0.58 (1.41)	0.28	−0.59 to 1.76
CzL	8	1.40 (0.41)	2.35 (1.00)	0.95 (0.82)	0.01	0.26 to 1.63
CzR	8	1.44 (0.90)	1.89 (0.85)	0.45 (1.21)	0.33	−0.56 to 1.47

Values are mean (SD).

SEPs = somatosensory evoked potentials. L and R refer to side of median nerve stimulation.

the yips are organic and further study on a golf course or under anxiety-provoking conditions is warranted. A better understanding of the yips may lead to advances for other task-specific disorders.

Acknowledgment

The authors thank Susan Bernstein, RN, for coordinating the study; Bonnie Mill, for technical assistance; Jose Hernandez, for statistical support; and Dave Pelz, for initial discussions about the study. They also thank the golfers for their participation.

From the Parkinson's Disease and Movement Disorders Center (Drs. Adler and Caviness) and Department of Biostatistics (J.G. Hentz), Mayo Clinic Scottsdale, AZ; Arizona State University (Dr. Crews); and Sports Medicine Center (Dr. Smith), Mayo Clinic Rochester, MN.

Supported by the Mayo Foundation for Medical Research and Education.

Received December 3, 2004.

Accepted in final form January 25, 2005.

Address correspondence and reprint requests to Dr. Charles H. Adler, Parkinson's Disease and Movement Disorders Center, Mayo Clinic Scottsdale, 13400 E. Shea Blvd., Scottsdale, AZ 85259; e-mail: cadler@mayo.edu

References

1. Smith AM, Laskowski ER, Cooney WP, et al. A multidisciplinary study of the 'yips' phenomenon in golf: an exploratory analysis. Sports Med 2000;30:423–437.
2. Sachdev P. Golfers' cramp: clinical characteristics and evidence against it being an anxiety disorder. Mov Disord 1992;7:326–332.
3. McDaniel KD, Cummings JL, Shain S. The "yips": a focal dystonia of golfers. Neurology 1989;39:192–195.
4. Smith AM, Adler CH, Crews D, et al. The 'yips' in golf: a continuum between a focal dystonia and choking. Sports Med 2003;33:13–31.
5. Ghez C, Gordon J, Hening W. Trajectory control in dystonia. Adv Neurol 1988;50:141–155.
6. Kanovsky P, Streitova H, Dufek J, Rektor I. Lateralization of the P22/N30 component of somatosensory evoked potentials of the median nerve in patients with cervical dystonia. Mov Disord 1997;12:553–560.

Presynaptic dopaminergic pathology in Chediak–Higashi syndrome with parkinsonian syndrome

C. Jacobi, MD; C. Koerner, MD; S. Fruehauf, MD; C. Rottenburger, MD; B. Storch-Hagenlocher, MD; and A. J. Grau, MD

Chediak–Higashi syndrome (CHS) is a rare autosomal-recessive lysosomal storage disorder caused by mutations of the lysosomal trafficking regulator gene (*LYST*) on chromosome 1q42. The disease is characterized by partial oculocutaneous albinism, immunodeficiency, peroxidase-positive granules in leukocytes and giant granules in other tissues (e.g., neurons, astrocytes), and neurologic disturbances. There are two distinct clinical patterns. In the childhood form, the hematologic system is affected with immunodeficiency, frequent bacterial infections, and an accelerated lymphoproliferative phase followed by early death. Neurologic signs and symptoms are uncommon.[1-3] In the adult form of CHS, which occurs between late childhood and early adulthood, hematologic dysfunction is milder and neurologic signs and symptoms are more prevalent: dementia, pyramidal and spinocerebellar signs and symptoms, peripheral polyneuropathy, and a parkinsonian syndrome.[2,3] The pathophysiology of the neurologic manifestations is still unknown.

Case report. A 22-year-old man was admitted to the Neurology Department because of progressive gait disturbance for 2 years. From childhood he had had partial albinism of the eyes and a convergent strabismus. During childhood and adolescence, infections often developed and a coagulation disturbance led to frequent bleeding.

The general examination revealed oculocutaneous albinism and pes cavus. He had a masklike face plus rigidity of the neck and all extremities; there was no tremor. Saccadic pursuit was present. The patient also presented with paraparesis, which was pronounced distally and on the left side. Deep tendon reflexes were diminished in the arms and absent in the legs. Babinski sign was positive on both sides. Except for reduced vibratory sensation at the ankles, findings from sensory examinations were normal. A stumbling gait caused frequent falls. Neuropsychological tests of memory (immediate and later recall), visuomotor speed, verbal fluency, and constructive abilities, and others were all clearly pathologic. His Mini-Mental State Examination score was 25/30 points.

The blood smear showed peroxidase-positive granules within lymphocytes and neutrophils. The hemogram depicted neutropenia, the bleeding time was delayed to 300 seconds (normal 94 to 193). Spontaneous migration and chemotactic activity of neutrophils and fluorescence-activating cell scanning (FACS) analysis of leukocytes were normal. Tibial nerve somatosensory evoked potentials to the left leg were delayed (P 40 after 51.2 msec). Sensory and motor nerve conduction studies were normal beside both sural nerves where no action potential could be elicited.

Brain MRI and EEG were normal. Disturbed presynaptic dopaminergic circuitry was demonstrated by markedly reduced binding in [^{123}I]N-omega-fluoropropyl-2beta-carbomethoxy-3beta-(4-iodophenyl)nortropane (FP-CIT) SPECT on both sides (figure 1a) on visual analysis.[4] On iodobenzamide (IBZM) SPECT, the quotient of the region of interest was calculated.[5] It was marginally lowered (left side: 1.47; right side: 1.45; normal reference: 1.54 $\pm$ 0.05). After an L-dopa test (250 mg), the patient improved from 24 to 8 points in selected areas (handwriting, speech, facial expression, rigidity, postural stability, body bradykinesia) of the Unified Parkinson's Disease Rating Scale. We initiated continuous L-dopa therapy, and the patient's condition stabilized. After 1 year of treatment, he stopped taking the medication and became rap-

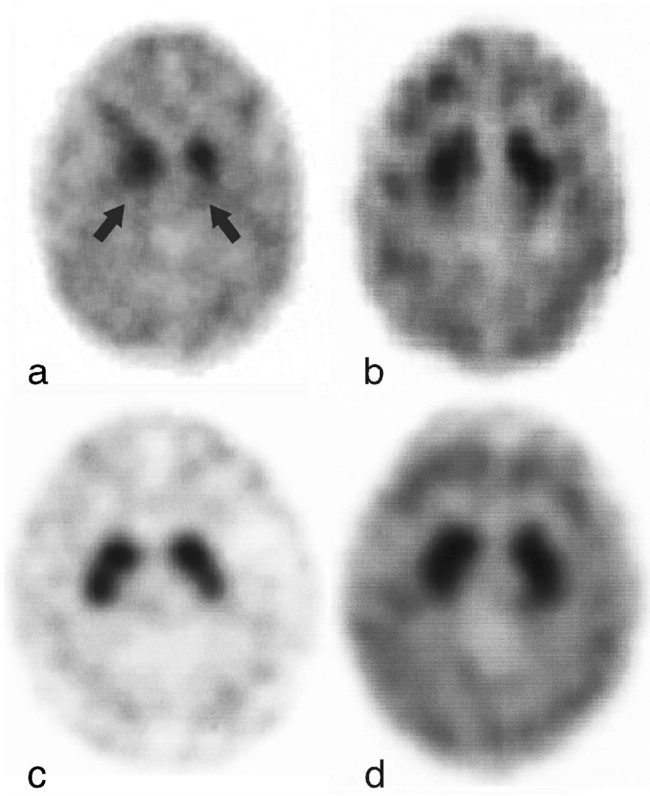

Figure. (a) [^{123}I] N-omega-fluoropropyl-2beta-carbomethoxy-3beta-(4-iodophenyl)nortropane (FP-CIT) SPECT of the CHS patient shows significantly lowered tracer uptake of the putamen on both sides (arrows). (b) Iodobenzamide (IBZM) SPECT of the patient with CHS. (c) Example of a normal [^{123}I] FP-CIT SPECT. (d) Example of a normal IBZM SPECT.