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Subjective anxiety measurements and cortisol responses in adults who stutter.
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Abstract:
Anxiety, as measured by self-report inventories and salivary cortisol levels, was examined in 11 adult males who stutter and 11 adult males who do not stutter during baseline, low stress, and high stress sessions. During the high stress session, salivary cortisol was significantly greater in Ss who stutter than in Ss who do not stutter. No significant differences were found between the 2 groups on the state or trait anxiety measures of the State-Trait Anxiety Inventory (STAI) or the Personal Report of Communication Apprehension. Significant differences in anxiety levels among the baseline, low stress, and high stress sessions for both groups of Ss were found for the STAI. No other significant differences or relationships were found between the 2 groups. (PsycINFO Database Record (c) 2009 APA, all rights reserved)
Subjects:
*Anxiety; *Hydrocortisone; *Stress; *Stuttering; Saliva

SUBJECTIVE ANXIETY MEASURES AND CORTISOL RESPONSES IN ADULTS WHO STUTTER

Anxiety, as measured by self-report inventories and salivary cortisol levels, was examined in 11 males who stutter and 11 males who do not stutter during baseline, low stress, and high stress sessions. During the high stress session salivary cortisol wes significantly greater in persons who stutter than in persons who do not stutter. No significant differences were found between the two groups on the State-Anxiety Inventory, Trait-Anxiety Inventory, or the Personal Report of Communication Apprehension. Significant differences in anxiety levels among the baseline, low stress, and high stress sessions for both groups of subjects were found for the State-Anxiety Inventory. No other significant differences or relationships were found between the two groups.

KEY WORDS: anxiety, cortisol responses, stuttering

Anxiety is a multidimensional construct that can be evaluated from a number of different perspectives. The anxiety response and the cortisol response are both components of arousal. A clear differentiation between "physiological arousal" (the psychophysiological response of the organism to real or anticipated threat), and "anxiety" (one of two possible subjective

interpretations of the arousal response), helps to clarify that subjective anxiety is not always associated with arousal response. The anxiety response can be defined as the subjective interpretation of increased arousal. The anxiety response and the cortisol response may form a common substrate for arousal. The arousal components of stress are a consequence of genetic and experiential processes that evolve with ontogeny (Dorn, Susman, & Petersen, 1993; Pancheri & Biondi, 1990). The occurrence of anxiety response may be precipitated, maintained, or exacerbated by psychological, social, and biological mechanisms. Anxiety can be measured in specific situations and related to individuals, their families, and society.

Researchers have examined transient "states" of anxiety, sometimes labeled mood states, as well as permanent "traits" of anxiety, sometimes labeled personality (Speilberger & Rickman, 1990). These types of psychobehavioral anxiety have been assessed through questionnaires and rating scales. Another form of anxiety, specific to speaking situations, has been labeled "communication apprehension" (McCroskey, 1978). This type of anxiety has been defined as the real or anticipated fear or anxiety associated with communication.

Persons who stutter report that anxiety occurs while speaking specific sounds or words, and in specific speaking situations. They also report anxiety as the result of chronic avoidances such as not answering the telephone or addressing a group of listeners (Bloodstein, 1987; Brutten & Shoemaker, 1967; Craig, 1990; Fitzgerald, Djurdjic, & Maguin, 1992; Greiner, Fitzgerald, Cooke, & Djurdjic, 1985; Miller & Watson, 1992; Sheehan, 1975; Van Riper, 1982). Anxiety was assessed in those who stutter through physiological measures examining changes in heart rate, skin response, autonomic nervous system activity arousal, or time pressure to complete a motor sequence (Baumgartner & Brutten, 1983; Kraaimaat, Janssen, & Brutten, 1988; Peters & Hulstijn, 1984; Weber & Smith, 1990). Conflicting and equivocal results were reported about the relationship between anxiety and stuttering.

Physiological Measurement of Anxiety

Researchers have attempted to study anxiety from a physiological perspective. Peters and Hulstijn (1984) examined heart rate, pulse volume, and tonic and phasic skin conductance in 24 persons who stuttered and 24 persons who did not stutter. Their results showed no differences between the two groups, but reported that during speech production tasks large increases in autonomic arousal were observed. Some studies have examined heart rate and galvanic skin response in an attempt to measure anxiety levels in persons who stutter (Baumgartner & Brutten, 1983; Kraaimaat, Janssen, & Brutten, 1988). These studies found positive correlations between subjects' inability to decrease stuttering repetitions and their increased skin conductance. Weber and Smith (1990) suggested that anxiety, as measured through autonomic nervous system activity, may interfere with production of fluent speech in persons who stutter. They viewed the stress response as a physiological breakdown in the motor or sensory activity of the nervous system. The authors examined the performance of 19 persons who stutter and 19 persons who do not stutter on a series of tasks assessing autonomic activity. Results revealed that increased sympathetic arousal (even though it was within normal ranges) was correlated with the occurrence and severity of stuttering. They concluded that stuttering was related to sympathetic activity but great variability was found in the levels of autonomic arousal for their subjects. Weber and Smith suggested that emotion or anxiety factors may have differential effects for

individuals who stutter. These studies suggest that anxiety may be related to physiological breakdowns in speech production.

Using hormonal indices for anxiety, Chmelova, Kujalova, Sedlackova, and Zelany (1975), and Leanderson and Levi (1967) examined catecholamine excretion in persons who stutter. They concluded that speech situations produced higher levels of hormone excretion in persons who stuttered. The studies examined a single sample of urine output as a post-test measure, but did not report multiple measurements over time.

Subjective Measurement of Anxiety

Researchers have studied anxiety from subjective and behavioral perspectives. These studies imply that maladjustment to the anxiety or inappropriately conditioned responses to anxiety reactions may be the cause of stuttering. Miller (1944) proposed that stuttering originated from a double approach-avoidance conflict. This theory was further developed by Sheehan (1975), who explained that stuttering resulted when the individual was caught in a role conflict between speaking and being silent. The conflict consisted of both speech and silence being perceived as having positive and negative features. Persons who stutter had an avoidance tendency for speaking (silence was an alternative), as well as an avoidance tendency for not speaking (fear of silence). The conflict resulted in a double approach-avoidance in that the avoidance did not come only from the fear and anxiety of speaking but also the competition between speaking and silence. Stuttering was the result of the person not being able to resolve the anxiety of the conflict.

Brutten and Shoemaker (1967) proposed a two-factor theory for stuttering, suggesting persons who stutter are classically conditioned to associate speech with fear, stress, and anxiety. Bloodstein (1987) suggested that anticipation of stuttering can sometimes be the greatest part of the disorder and the primary cause for tension and fragmentation.

Craig (1990), Fitzgerald et al. (1992), and Greiner et al. (1985), have reported support for a "generalized state of anxiety" for persons who stutter. Craig (1990) examined 102 people who stutter on state and trait anxiety measures before and after an intensive behavioral treatment for stuttering. His findings indicated that persons who stutter had higher levels of chronic fear in demanding speech situations before behavioral treatment, but not after the treatment. Persons who stutter also maintained higher levels of chronic anxiety than control subjects, both pre- and post-treatment.

Fitzgerald et al. (1992) suggested that the development of speech fluency is mediated and moderated by anxiety. Their model suggested that certain affective states or temperamental characteristics could influence fluency and stuttering. Speech-situation anxiety mediated the stuttering behavior, whereas generalized anxiety was the moderator of stuttering severity.

Miller and Watson (1992) reported that the anxiety/stuttering relationship was "speech specific anxiety" and related only to communication attitudes. They refuted the "generalized anxiety concept" with a study of 52 persons who stutter, using a number of self-report, psychological scales. The State-Trait Anxiety Scale (Spielberger, Gorsuch, & Lushene, 1970), Beck Depression

Scale (Beck, 1987), and the Erickson Modified 24 Scale (Andrews & Cutler, 1974) were administered to subjects from support groups for persons who stutter and from a matched group of control subjects. Results showed that persons who stutter did not differ significantly from persons who do not stutter on State- and Trait-Anxiety Inventory and depression measures. Persons who stutter demonstrated differences from persons who do not stutter only on communication attitudes. They interpreted the greater negative communication attitudes of persons who stutter as a reaction to continual negative responses from listeners.

Cortisol--An Anxiety Hormone

The physiological arousal associated with anxiety involves the activation of a number of hormones including catecholamine, adrenocorticotropin, cortisol, prolactin, and thyroid hormones (Meyerhoff, Oleshansky, & Mougey, 1988). According to Susman, Nottelmann, Dorn, Gold, and Chrousos (1989), the relationship between hormones and emotion may be bidirectional. Hormone secretion can cause certain emotional states and also be the response to emotional states such as anger, aggression, fear, or anxiety.

Cortisol is the main glucocorticoid hormone in humans and is released from the adrenal cortex in response to adrenocorticotrophic hormone (ACTH) and regulated by the hypo-thalamic-pituitary-adrenocortical (HPA) axis. Cortisol regulates many physiological systems and is one of the most prominent stress hormones. Cortisol is elevated under emotional states in normal subjects (Axelrod & Reisine, 1984). The primary actions of cortisol include stimulation of the hepatic gluconeogenesis, skeletal muscle glycogenesis and reduced glucose uptake to provide energy for the "fight or flight" response to stress (Norman & Litwack, 1987). Cortisol has been reported to be elevated in normal patients with caffeine-induced anxiety (Charney, Heninger, & Jatlow, 1985), in anxious subjects during mirror-drawing tasks (Miyabo, Hisada, Asto, Mizushima, & Uero, 1976), and in patients with simple phobias (Fredrikson, Sundin, & Franen-hauser, 1985). Elevated cortisol has been related to the severity of spontaneous panic attacks (Cameron, Lee, Curtis, & McCann, 1987) and to surgical stress (Tacker, Leach, Owen, & Rummel, 1978). In biobehavioral research, salivary cortisol measurements provide a relatively trauma-free and noninvasive technique to examine hormonal influences when compared to plasma cortisol collection (Laudat, Cerdras, Fournier, Guiban, Guilhaume, & Luton, 1988).

Although a number of investigations have studied the relationship between anxiety and stuttering, most of these studies have attempted to examine this multidimensional problem using either subjective measurements or physiological measurements. No studies have attempted to use an integrative approach to examine physiological arousal (through cortisol indices), general subjective anxiety (through state anxiety and trait anxiety), and communication anxiety (through communication apprehension measurements) simultaneously. We sought to investigate the relations among cortisol levels and self-report measures of trait and state anxiety, and perceived communication apprehension in groups of persons who stutter and persons who do not stutter.

Method Subjects

Eleven persons who stutter (mean age 21.5 years; range 19-36 years) and 11 persons who do not stutter (mean age 22.5 years; range 19-33 years) participated in the investigation. All subjects

were male, European Americans with normal hearing acuity, matched on age and educational level. Subjects ranged from 1 to 6 years of college education (mean = 2.3 years of college). Subjects were required to: (a) meet with the experimenters during a baseline session and provide a saliva sample, (b) meet with the experimenters during a low stress time and provide a saliva sample, (c) meet the experimenters during a high stress time (immediately preceding an important final examination) and provide a saliva sample, (d) be videotaped during 5-10 minutes of conversational speech at each of the baseline, low stress, and high stress sessions, and (e) complete a number of questionnaires for communication apprehension, stress levels, anxiety levels, optimism, and humor during each of the three sessions.

Volunteers were recruited from advertisements and flyers placed in the local community and university, requesting volunteers for a study on stress and stuttering. The advertisement flyer was also mailed to current and former clients at the Penn State Speech and Hearing Clinic. Subjects were paid $50.00 for their participation.

Severity ratings of persons who stutter were based on the Riley Stuttering Severity Instrument-SSI (Riley, 1980) using the speech samples from the baseline session. The SSI determines severity of stuttering based on reading and spontaneous speech samples using frequency of repetitions and hesitations, duration of the longest blocks, and concomitant behaviors. Three subjects were classified as mild, 4 subjects as moderate, and 4 subjects as severe. Persons who stutter had received from 2 to 13 years of speech treatment. At the time of the study, 5 of the subjects were enrolled in active treatment (Table 1).

Subjective Anxiety Assessment

Two standardized measures were employed to measure general anxiety, and one standardized measure was used to evaluate communication apprehension.

The State-Trait Anxiety Inventory-STAI (Spielberger et al., 1973) is one of the most commonly used measures of anxiety. The scale has good concurrent validity and reliability. The two 20 item scales (one measuring State anxiety, the other one measuring Trait anxiety) are self-administered. Subjects were asked to rate the 20 statements from each scale from: 1 (not at all), 2 (somewhat), 3 (moderately so), to 4 (very much so). Higher scores indicate greater anxiety. Individuals with higher scores are more prone to perceive situations as threatening and may respond to these situations with fear, nervousness, tension, worry, or some behavioral change.

The State Anxiety Inventory is reported to relate information about transitory emotional states, at any given time. It is expected that changes in the State Anxiety are related to internal and external stressors perceived by the individual. Statements include: I am happy; I am calm; I am tense; I tire quickly.

The Trait Anxiety Inventory is reported to reflect a certain overall disposition to anxiety and the individual's reaction to stress. This inventory is reported not to be influenced by situational stress. Subjects are asked to report how they generally feel. Higher scores indicated greater anxiety. Items include: I feel calm; I feel pleasant; I feel satisfied with my life.

The Personal Report of Communication Apprehension-PRCA (McCroskey, 1978) is a 25-item scale designed to examine communication apprehension, anxiety, and fear in speaking to another individual or individuals. Subjects were asked to rate their agreement with the statements on a 5-point scale where 1 was strongly agree and 5 was strongly disagree. This scale has been used in over 80% of the research in communication apprehension and has reliability estimates greater than 0.90. Examples of test items include: I have no fear of facing an audience; I always avoid speaking in public if possible; I would enjoy presenting a speech on a local television show; I feel relaxed and comfortable while speaking; I talk less because I'm shy, Sample means with over 12,000 college students and 4,000 adults consistently range between 73 and 75, with a standard deviation between 13 and 15. Scores above 88 are considered "high" in communication apprehension and scores below 58 are considered "low."

Cortisol Assessment

Collection of saliva samples. Subjects were seated in a clinic room where they were asked to produce approximately 5 ml of saliva in a plastic vial. Three appointments were scheduled between 3:30 p.m. and 6:00 p.m. to control for circadian variations in cortisol concentrations (Walker, Riad-Fahmy, & Read, 1978). Subjects were requested not to eat, smoke, or drink coffee after 12:00 p.m. Subjects rinsed their mouths with water and were given one quarter of a 2 x 2 inch piece of sterile gauze to place in their mouths to help stimulate saliva. The subjects were instructed to tip the head forward, spread the lips apart, and support the forehead by the hands. Saliva was allowed to run out of the parted lips into a plastic test tube with a screw cap. They were told not to cough up or attempt to fill the vial from throat clearing. Three saliva samples of approximately 5 ml in volume were collected at 20-minute intervals. The samples at 0, 20, and 40 minutes were collected to minimize the minute-to-minute fluctuations that normally occur in hormones. The mean values for the 3 samples were then used in the statistical analyses. Samples were immediately placed in a freezer and the specimens were sent to the laboratory and stored at -70 degrees Celsius until assayed.

Measurement of salivary cortisol. Salivary cortisol was determined by direct radioimmunoassay using 1-125 cortisol. The samples were assayed using the Amersham Amerlex Cortisol, RIA Kit. Salivary cortisol was measured according to the procedures of Gunnar, Marvinney, Isensee, and Fisch (1989). The assay method used has the cortisol compete with the cortisol 1-125 derivative for binding sites on the anticoated particles. Cortisol is then easily separated by centrifugation. After removal of the liquid, the radioactivity is measured in a gamma counter. The concentration of cortisol is estimated from the dose-response curves prepared using the standard sera.

Procedures

The study consisted of three sessions. The sessions were scheduled at the same time of the day to control for circadian rhythms that can affect cortisol elevations.

The baseline session was approximately 150 minutes in length and consisted of the completion of a number of questionnaires and scales evaluating anxiety, tension, humor, coping strategies, daily hassles, mood states, and speech. The subjects were provided instructions about completing the State-Trait Anxiety Scale and the Personal Report of Communication Apprehension.

Information regarding history of the stuttering problem, onset of the problem, treatment experiences, and attitudes toward stuttering were obtained using an interview format for persons who stutter. The frequency, duration, severity, and type of stuttering were evaluated from the videotaped conversation samples. All subjects were videotaped individually during 10 minutes of conversational speech for later fluency analyses. Upon completion of the scales, questionnaires, and videotaping, subjects were escorted to clinic rooms where they were provided with instructions and plastic vials for saliva collection, Subjects were left alone to fill the vials. The experimenters entered the room approximately 3 minutes after the subjects began producing saliva, asked the subjects to screw the caps on the vials, labeled the vials, and placed them in a freezer.

The second session was at least 8 days, but no more than 12 days, following the first session. This was the low stress session for 17 (77%) of the subjects. Subjects were encouraged to select a "stress-free, hassle-free" day at least 1 week after the initial interview, and call the experimenters to schedule an appointment for the afternoon. The same scales, questionnaires, videotaping, and saliva collection techniques were employed.

The third session tended to be the high stress session and occurred at least 1 week after the second session. This session was either scheduled a few hours before an important final examination, a speech in a public speaking class, or a day where "everything was going wrong" (e.g., failed an exam, forgot an important assignment, overslept for classes, and had a difficult time at work). Again, the same scales, questionnaires, videotaping and saliva collection techniques were employed.

Data Analyses

Univariate (ANOVA) and multivariate analyses of variance (MANOVA) with repeated measures were employed to determine statistical significance between the group means for the subjects. The independent variables were Group (persons who stutter and persons who did not stutter) and the Sessions variable was the repeated measures variable with three levels (baseline, low anxiety, and high anxiety). The dependent variables included: the State-Anxiety Inventory scores, Trait-Anxiety Inventory scores, Personal Report of Communication Apprehension scores, and the salivary cortisol responses. Post hoc comparisons were computed for comparisons between individual sessions. Chi-square analyses were performed on the number of subjects in groups for communication apprehension. Correlations were generated to determine relationships among subjective measurements, cortisol responses, and severity of stuttering (SAS, 1990).

Results Cortisol Responses

The cortisol assessment in this study was not limited to a single assessment but included three analyses from each of the saliva collections during baseline, low stress, and high stress sessions. This procedure was employed to increase the reliability of the sampling techniques. The mean data from the three analyses were submitted to analyses of variance and revealed no significant differences among the means for the subjects. Therefore, the cortisol response data were the mean of the three analyses for each subject's baseline, low stress, and high stress sessions.

Tests of normality were run on the cortisol data using the Shapiro-Wilks statistic. Results revealed that cortisol data were not normally distributed. The data were positively skewed and therefore the values were log-transformed before the statistical analysis were conducted. Means and standard deviations for the cortisol levels for the log-trans-formed data are reported in Table 2. The mean levels for cortisol data measured in micrograms per deciliter for persons who stutter were: baseline (.242); low stress (.245); andhigh stress (.267). The mean levels for cortisol measured in micrograms per deciliter for persons who do not stutter were: baseline (.236); low stress (.238); and high stress (.241). Analyses of variance (ANOVA) with repeated measures revealed a significant difference between the two groups with $F(1,20) = 10.6$; $p = .0004$, significant differences between the sessions with $F(2,20) = 5.8$; $p = .01$, and a significant interaction of group and sessions with $F(2,20) = 8.8$; $p = .002$. Post hoc tests revealed the interaction was a result of persons who stutter (mean = .264, standard deviation = .16) showing a significant difference ($p < .001$) in mean cortisol responses from persons who do not stutter during the high stress session. During this session, persons who stutter experienced higher cortisol levels than persons who do not stutter. Results also revealed that cortisol responses for persons who stutter were significantly higher during the high stress session than during the baseline and low stress session ($p < .001$). There were no significant differences among sessions for persons who do not stutter.

Subjective Anxiety Responses

The mean and standard deviations of the responses from the State-Anxiety Inventory (SAI) scores, Trait-Anxiety Inventory (TAI) scores, and Personal Report of Communication Apprehension (PRCA) scores are reported in Table 3. Inspection of the data shows similar scores on the SAI and TAI during the baseline and low stress sessions. Persons who stutter showed higher scores on the SAI during the high stress session than persons who do not stutter. PRCA scores were consistently higher for persons who stutter than for persons who do not stutter.

A multivariate analysis of variance (MANOVA) with repeated measures revealed no significant difference between the two groups on all three subjective anxiety measures using Wilks Lambda with Rao's $R(3,18) = 2.7$; $p = .08$, Significant differences were found among the sessions with $R(6,15) = 11.6$; $p = .0002$), and no significant interaction of group and sessions was found with $R(6,15) = 1,1$; $p = .43$). Closer inspection of the sessions data revealed that the SAI changed significantly from baseline to high stress for both groups of subjects ($p < .01$). This was expected because the inventory proposes to assess transient mood states. These data demonstrated that subjects did perceive increased anxiety on high stress days.

Subjects were classified as experiencing high and low communication apprehension based on the PRCA scores. Four (36%) persons who stutter were classified as experiencing high communication apprehension, while two (18%) persons who stutter were classified as experiencing low communication apprehension. Two (18%) of the 11 subjects who do not stutter were classified as experiencing high communication apprehension, while 5 (45%) of the subjects who do not stutter were classified as experiencing low communication apprehension. Five (45%) of the subjects who stuffer were classified as experiencing neither high nor low communication apprehension with mean scores very similar to those reported in the normative sample. The remaining four (36%) subjects who do not stutter were also classified in this manner. Chi-square

analyses revealed no significant differences between subjects who stutter and subjects who do not stutter in high and low communication apprehension groups.

Interrelationships Between Variables

The results of the correlation analyses revealed no significant correlations between cortisol means and state, trait, or communication apprehension scores.

Stuttering Severity, Anxiety Measurements, and Cortisol Responses

Persons who stutter were classified as mild (n = 3), moderate (n = 4), and severe (n = 4) from the baseline speech samples collected. The scores from the Stuttering Severity Instrument were used to determine correlations between cortisol and subjective anxiety measurements. It was hypothesized that higher cortisol responses, state anxiety scores, and communication apprehension scores would be related to higher ratings of severity. Spearman rank order correlations were computed and revealed that no significant associations existed between severity scores, cortisol responses, and subjective anxiety measures.

Discussion

The primary aims of this study were to investigate (a) cortisol responses, (b) subjective anxiety measurements, and (c) relations among cortisol levels and self-report measures of state and trait anxiety, and perceived communication apprehension in groups of persons who stutter and persons who do not stutter. Significant differences in cortisol responses were found during a high stress session between persons who stuffer and persons who do not stutter. No significant differences were found between persons who stuffer and persons who do not stutter for subjective anxiety measurements on a communication apprehension scale. Finally, no significant correlations were found between cortisol responses and subjective anxiety measures. In addition, no significant relationships were found among stuttering severity, cortisol responses, and subjective anxiety measurements.

The data suggest that when persons who stutter are under high stress, their cortisol levels are different than those of persons who do not stuffer. Salivary cortisol measurements can help to assess pituitary-adrenal function. During a self-selected high stress day, subjects who stuffer displayed higher cortisol levels than during either baseline or self selected low stress sessions. In addition, they also displayed significantly higher levels than persons who do not stutter. Persons who stutter showed cortisol responses similar during the baseline and low stress conditions to those of persons who do not stutter. This suggests that perceived high stress led to higher cortisol responses.

It may be that persons who stutter perceive stress or "daily hassles" in a different manner than do persons who do not stutter. Persons who stutter may have developed such a high threshold of accommodation to daily stress that when they scheduled their "high stress day" appointment, they were extremely stressed. It could also be speculated that persons who stuffer perceived speaking before a speech-language pathologist in a speech and hearing clinic on camera and completing a group of questionnaires as stressful. Because the data for baseline and the low

stress days are not significantly different from data from persons who do not stutter, it could be that the additional stress of this task during a high stress day explained the higher cortisol responses. Several studies have shown that merely the anticipation of a distressing event can be sufficient to induce cortisol elevation (Bassett, Marshall, & Spillane, 1987; Mason, Hartley, & Kotchen, 1973). Kirschbaum, Wust, and Hellhammer (1992) have suggested that cognitive and emotional processing of psychosocial stimuli can alter the activity of the hypothalam-icpituitary-adrenocortical (HPA) axis. Their results indicated that anticipating the stress of public speaking increased cortisol levels in male subjects. It may be that subjects who stutter perceived this situation as more stressful on days when they had reached their optimum in stresses, annoyances, and hassles. This may suggest that during perceived high stress times, persons who stutter set off a physiological "button" to their endocrine system, engaging a stress response. Cameron and Neese (1988) reporting on the psychobiology of stress indicated that once an anxiety reaction is triggered, the psychophysiological symptoms of individuals classified as having "an anxiety, panic or phobic" disorder and those of "stressed" normal individuals are very similar.

The cortisol assessments in this study were not limited to a single measurement but included three collections during each session. Therefore, the nature of the elevated cortisol is significant. In addition, all the samples were collected between 3:30 p.m. and 6:00 p.m. to control for circadian variations in cortisol concentrations. Cortisol is reported to be present at highest concentrations in the early morning and dissipates during the day. Although the design of this study was to have subjects self-select stressful days and necessitated afternoon collections, other studies might find differences between baseline or low stress with early morning salivary cortisol collections.

Weber and Smith (1990) have suggested that anxiety may interfere with production of fluent speech in persons who stutter. They concluded that stuttering was related to sympathetic activity and that emotion or anxiety factors may have differential effects for individuals who stutter. The present data support the concept of differential effects, and add the suggestion that perceived anxiety or stress may play a role in triggering a specific physiological response in persons who stutter. Future studies will need to determine the relationship between breakdowns in speech production and fluency and cortisol responses.

The finding that persons who stutter do not differ from persons who do not stutter on subjective anxiety instruments confirms the observations of Miller and Watson (1992). They showed no significant differences on state anxiety, trait anxiety, and a depression scale between persons who stutter and persons who do not. They indicated that their results on communication attitude supported their assumptions about speech-related and communication-related anxiety. The findings of this study suggest that as a group, persons who stutter have no unique anxiety profile. They are not more anxious than persons who do not stutter according to standardized general anxiety measurements. The results suggest that even in the area of communication apprehension, which would appear to present problems for persons who stutter, no significant differences were found between the groups. The results suggest that when persons who stutter are evaluated on a task not designed to separate persons who stutter from persons who do not stutter (Miller and Watson used the Erickson Modified 24 Scale, which includes specific items regarding stuttering attitudes), they perform similarly. Although a greater number of persons who stutter (36%) were classified as experiencing high communication apprehension, apparently the remaining subjects

(64%) have either overcome those fears or have developed appropriate coping skills so that they do not fear communication. This may also reflect one of the benefits of speech treatment.

The present findings are not consistent with those reported by Craig (1990). His data supported the idea that persons who stutter demonstrated higher State Anxiety and Trait Anxiety scores before treatment. It is interesting to note that his SAI mean data for persons who stutter are very similar to our data during the high stress time. Craig evaluated persons who stutter immediately prior to entering treatment. Perhaps individuals provided with questionnaires regarding their anxiety levels prior to a perceived radical transition in their lives, such as speech treatment, are experiencing greater stress. That would also account for a reduction in these anxiety levels at the termination of treatment. An explanation for the high trait levels cannot be accommodated with our data. These data are also not supportive of a generalized anxiety theory of stuttering.

The results of this study also offer a novel interpretation for Bloodstein's Anticipatory Struggle Hypothesis (ASH) of stuttering. The ASH states that stuttering is the reaction of struggle or avoidance in anticipation of imagined speech difficulty. Kirschbaum et al. (1992) found that anticipating the stress of public speaking increased cortisol levels in male subjects. If persons who stutter anticipate that the speaking situation will be difficult, they may "trigger" a physiological or hormonal anxiety response. In this study, subjects who stutter may have anticipated the high stress situation as more stressful on days when they had reached their optimum in stresses, annoyances, and hassles. In other words, the psychological theory behind ASH may be rooted more deeply in a programmed hormonal response.

Finally, it was surprising that there were no correlations between cortisol responses and subjects' behavioral responses, despite significant changes in both. The large range of responses on the SAI were not associated with changes in cortisol levels for either persons who stutter or persons who do not stutter. It is plausible that cortisol elevations occur when subjects anticipate stress or loss of control, but the SAI, TAI, and PRCA were not sensitive enough to these changes. It may also be that the homogeneous group of subjects used in this study routinely take subjective examinations and paper and pencil tests, and did not use the complete range of responses available on the measurements. It is also possible that greater changes in the cortisol taken at multiple times during the day (morning, noon, and late afternoon) would show some relationship between cortisol and subjective measurements. These findings suggest that these subjective anxiety and communication apprehension measures do not relate to the hormonal response reported by a select group of male subjects who either stutter or do not stutter. A systematic inquiry into what caused cortisol changes during a high stress session for persons who stutter will need to be conducted. Studies addressing the relationships among cortisol responses, reactivity, and situational and individual variation in persons who stutter should be conducted.

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TABLE 1 Summary table of the number of years in treatment, present treatment choices, and severity of stuttering of persons who stutter.

Legend for Chart:

A - Subjects
B - Age
C - Years in treatment
D - Present treatment choice
E - Stuttering severity

A	B	C	D	E
1	18	8	Receiving treatment	Moderate
2	19	4	Not receiving treatment	Mild
3	19	9	Not receiving treatment	Moderate
4	20	8	Not receiving treatment	Severe
5	20	5	Receiving treatment	Severe
6	20	2	Not receiving treatment	Mild
7	20	2	Not receiving treatment	Mild
8	20	8	Receiving treatment	Moderate
9	21	7	Receiving treatment	Severe
10	24	13	Receiving treatment	Severe
11	36	11	Not receiving treatment	Moderate

TABLE 2 Salivary cortisol means and standard deviations (in micrograms per decillter) for the two groups during baseline, low stress, and high stress sessions using log transformed values.

Legend for Chart:

A - Sessions
B - Group: Persons who stutter: Mean
C - Group: Persons who stutter: Standard deviation
D - Group: Persons who did not stutter: Mean
E - Group: Persons who not stutter: Standard deviation

A	B	C	D	E
Baseline	.242	.16	.236	.15
Low stress	.245	.09	.238	.11
High stress	.267[a]	.16	.241	.10

a High stress group mean score for persons who stutter was significantly different from baseline and low stress mean scores for persons who stutter. This score was also significantly different from baseline, low and high stress mean scores for persons who do not stutter at the $p < .001$ level of confidence.

TABLE 3 Means and standard deviations for the State Anxiety Inventory, Trait Anxiety Inventory, and the personal report of communication apprehension for the two groups during baseline, low stress, and high stress sessions.

Legend for Chart:

A - Sessions
B - Group: Persons who stutter: Mean
C - Group: Persons who stutter: Standard deviation
D - Group: Persons who did not stutter: Mean
E - Group: Persons who not stutter: Standard deviation

A	B	C	D	E
Baseline				
State Anxiety Inventory	34.4[a]	13.1	36.2[b]	8.1
Trait Anxiety Inventory	34.8	6.7	35.0	4.5
Personal report of communication apprehension	78.3	12.2	72.8	15.4
Low stress				
State Anxiety Inventory	32.4[a]	6.9	31.8[b]	7.0
Trait Anxiety Inventory	36.0	5.9	36.1	5.3
Personal report of communication apprehension	80.1	13.8	71.2	16.1
High stress				
State Anxiety Inventory	50.1[a]	9.4	44.1[b]	9.2
Trait Anxiety Inventory	35.9	6.1	36.0	5.4
Personal report of communication apprehension	78.7	14.3	68.5	16.4

a Denotes significant differences between the high stress, low stress, and baseline means for persons who stutter.

b Denotes significant differences between the high stress, low stress, and baseline means for persons who do not stutter.

References

Andrews, G., & Culter, J. (1974). Stuttering therapy: The relation between changes in symptom level and attitudes. Journal of Speech and Hearing Disorders, 39, 312-319.

Axelrod, J., & Reisine, T. D. (1984). Stress hormones: Their interaction and regulation. Science, 224, 452-459.

Bassett, J. R., Marshall, P. M., & Spillane, R. (1987). The physiological measurement of acute stress (public speaking) in bank employees. International Journal of Psychophysiology, 5, 265-273.

Baumgartner, J. M., & Brutten, E. J. (1983). Expectancy and heart rate as predictors of the speech performance of stutterers. Journal of Speech and Hearing Research, 26, 383-385.

Beck, A. T. (1987). The Beck Depression Inventory. San Antonio: The Psychological Corporation.

Bloodstain, O. (1987). A handbook on stuttering (4th ed.). Chicago: National Easter Seal Society.

Brutten, E. J., & Shoemaker, D. J. (1967). The modification of stuttering. Englewood Cliffs, N.J.: Prentice-Hall.

Cameron, O. G., & Nesse, R. M. (1988). Systemic hormonal and physiological abnormalities in anxiety disorders. Psychoneuroendocrinology, 13, 287-307.

Cameron, O. G., Lee, M. A., Curtis, G. C., & McCann, D. S. (1987). Endocrine and physiological changes during "spontaneous" panic attacks. Psychoneuroendocrinology, 12, 321-331.

Charney, D. S., Heninger, G. R., Jatlow, P. I. (1985). Increased anxiogenic effects of caffeine in panic disorders. Archives of General Psychiatry, 42, 233-243.

Chmelova, A., Kujalova, V., Sedlackova, E., & Zelany, A. (1975). Neurohumorale reaktionen bei stotterern und polterern. Folia Phoniatrica, 27, 283-286.

Craig, A. (1990). An investigation into the relationship between anxiety and stuttering. Journal of Speech and Hearing Disorders, 55, 290-294.

Dorn, L. D., Susman, E. J., & Petersen, A. C. (1993). Cortisol reactivity, anxiety and depression in pregnant adolescents: A longitudinal perspective. Journal of Psychoneuroendocrinology, 18, 219-239.

Fitzgerald, H. E., Djurdjic, S. D., & Maguin, E. (1992). Assessment of sensitivity to interpersonal stress in stutterers. Journal of Communication Disorders, 25, 31-42.

Fredrikson, M., Sundin, O., & Frankenhauser, M. (1985). Cortisol excretion during the defense reactions in humans. Psychosomatic Medicine, 47, 313-319.

Grelner, J. R., Fitzgerald, H. E., Cooke, P. A., & Djurdjic, S. D. (1985). Assessment of sensitivity to interpersonal stress in stutterers and nonstutterers. Journal of Communication Disorders, 18, 215-225.

Gunnar, M. R., Marvinney, D., Isensee, J., & Fisch, R. O. (1989). Coping with uncertainty: New models of the relations between hormonal, behavioral and cognitive processes. In D. S. Palermo (Ed.), Coping with uncertainty: Behavioral and developmental perspectives (pp. 101-129). Hillsdale, NJ: Laurence Erlbaum.

Kirchbaum, C., Wust, S., & Hellmammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. Psychosomatic Medicine, 54, 648-657.

Kraaimaat, F., Janssen, P., & Brutten, E. J. (1988). The relationship between stutterers' cognitive and autonomic anxiety and therapy outcome. Journal of Fluency Disorders, 13, 107-113, 67-84.

Laudat, M. H., Cerdras, S., Fournier, C., Guiban, D., Gullhaume, B., & Luton, J. P. (1988). Salivary cortisol measurement: A practical approach to assess pituitary-adrenal function. Journal of Clinical Endocrinology and Metabolism, 66, 343-348.

Leanderson, R., & Levi, L. (1967). A new approach to the experimental study of stress and stuttering. Acta Otoloaryngologica, 224 (Supplement), 311-316.

Mason, J. W., Hartley, L. H., & Kotchen, T. A. (1973). Plasma cortisol and norepinephrine responses in anticipation of muscular exercise. Psychosomatic Medicine, 35, 406-413.

McCroskey, J. C. (1978). Validity of the PRCA as an index of oral communication apprehension. Communication Monographs, 45, 192-203.

Meyerhoff, J. L., Oleshansky, M. A., & Mougey, E. H. (1988). Psychologic stress increases plasma levels of prolactin, cortisol and POMC-derived peptides in man. Psychosomatic Medicine, 50, 295-303.

Miller, N. E. (1944). Experimental studies in conflict: Personality and behavior disorders. New York: Ronald Press.

Miller, S., & Watson, B. C. (1992). The relationship between communication attitude, anxiety and depression in stutterers and nonstutterers. Journal of Speech and Hearing Research, 35, 789-798.

Miyabo, S., Hisada, T., Asto, T., Mizushima, N., & Uero, K. (1976). Growth hormone and cortisol responses to psychological stress: Comparisons of normal and neurotic subjects. Journal of Clinical Endocrinology and Metabolism, 42, 1158-1162.

Norman, A.W., & Litwack, G. (1987). Hormones. New York: Academic Press, Inc.

Pancheri, P., & Biondi, M. (1990). Biological and psychological correlates of anxiety; The role of psychoendocrine reactivity as a marker of the anxiety response. in N. Santorius, V. Andreoli, G. Cassano, L. Eisenberg, P. Kielholz, P. Pancheri, & G. Racagni (Eds.), Anxiety--Psychobiological and clinical perspectives (pp. 101-113). New York: Taylor & Francis Group.

Peters, H. F., & Hulstijn, W. (1984). Stuttering and anxiety. Journal of Fluency Disorders, 9, 67-84.

Riley, G. D. (1980). Stuttering Severity Instrument for Children and Adults. Tigard, Oregon: C. C. Publications.

SAS User's Guide; Statistics. Version 6 Edition, (1990). Cary, NC: SAS Institute.

Sheehan, J. G. (1975). Conflict theory and avoidance reduction therapy. In J. Eisenson (Ed.). Stuttering: A second symposium (pp. 97-198). New York: Harper and Row.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). Manual for the state-trait anxiety inventory. Palo Alto. Calif: Consulting Psychologists Press.

Spielberger, C. D., & Rickman, R. L. (1990). Assessment of state and trait anxiety. In N. Santorius, V. Andreoli, G. Cassano, L. Eisenberg, P. Kielholz, P. Pancheri, & G. Racagni (Eds.), Anxiety--Psychobiological and clinical perspectives (pp. 69-83). New York: Taylor & Francis Group.

Susman, E. J., Nottelmann, E. D., Dorn, L. D., Gold, P. W., & Chrousos, G. P. (1989). In D. S. Palermo (Ed.), Coping with uncertainly: Behavioral and developmental perspectives (pp. 101129). Hillsdale, NJ: Laurence Erlbaum.

Tacker, M. M., Leach, C. S., Owen, C. A., & Rummel, J. A. (1978). Levels of cortisol, corticosterone, and 11-Deoxycortisol in the plasma of stressed and unstressed subjects. Journal of Endocrinology, 76, 165-166.

Van Riper, C. (1982). The nature of stuttering. Englewood Cliffs, NJ: Prentice Hall.

Walker, R. F., Riad-Fahmy, D., & Read, G. F. (1978). Adrenal status assessed by direct radioimmunoassay of cortisol in whole saliva and parotid fluid. Clinical Chemistry, 24, 1460-1463.

Weber, C. M., & Smith, A. (1990). Autonomic correlates of stuttering and speech assessed in a range of experimental tasks. Journal of Speech and Hearing Research, 33, 690-706.

Wolpe, J. (1982). The practice of behavior therapy. New York: Pergamon Press.

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