

Neuropsychological effects of stress on social phobia with and without comorbid depression

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Abstract

Background: The influence of stress on neuropsychological functioning was assessed in socially phobic (SP), comorbid socially phobic/major depression (CM), and asymptomatic control subjects (AC) under baseline and stressor conditions.

Methods: Subjects were 33 clinically diagnosed undergraduates aged 18 to 41 years. Neuropsychological measures included the Trail Making Test, Wisconsin Card Sorting Test (WCST), Spatial Span, and Digit Span administered during a baseline condition and a psychosocial stress condition (videotaping).

Results: Spatial Span scores were reduced for SP during stress, improved for AC, and showed no change for CM. TMT B times showed an interaction effect, with completion time improving significantly less for SP than for AC and CM during stress. Analyses of the normative data for WCST total errors indicated that AC and CM improved significantly during stress, while SP performance declined during stress.

Conclusions: The present findings suggest that comorbid depressed versus non-depressed SP subjects respond uniquely to stress in terms of their neuropsychological functioning and self-reported mood and experiences; generalized social phobia may be associated with spatial working memory disturbance during social stress. Therefore, situations involving potential social and personal evaluation (e.g., examinations or presentations) may have a significant impact on the neuropsychological functioning of SP individuals.

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Introduction

The study of physiology and neurocognition in anxiety disorders has received relatively little research attention, although anxiety disorders are highly prevalent and impairing. Social phobia, for example, is the most common of all anxiety disorders with approximately 13.3% of the US population meeting criteria for social phobia over their lifetime according to the National Comorbidity Survey (Kessler et al., 1994).

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In addition to the high percentage of those with a diagnosis, social phobia also is often chronic and unremitting (Dingemans, van Vliet, Couvee, & Westenberg, 2001; Reich, Goldenberg, Vasile, Goisman, & Keller, 1994). The consequences of unremitting and untreated social phobia include interference with romantic relationships, poorer educational attainment, and career difficulties (Wittchen, Fuetsch, Sonntag, Muller, & Liebowitz, 2000). Ameringen, Mancini, and Farvolden (2003) found that 49% of social phobics left school prematurely. These academic difficulties may be due to impaired cognition during stressful situations in socially phobic (SP) individuals, but the neuropsychology of anxiety disorders is not well studied or understood.

In an effort to further understand the effects of anxiety symptoms on cognitive functioning, Paterniti, Dufouil, Bisserbe, and Alépovitch (1999) found that high anxiety was associated with poor performance on measures of verbal attention, working memory, and set shifting. Preliminary studies of other anxiety disorders suggest that subjects with panic and obsessive-compulsive disorder also perform significantly worse on measures of set shifting, spatial attention, and working memory tasks compared to depressed subjects (Purcell, Maruff, Kyrios, & Pantelis, 1998), but very little research has examined neuropsychological functioning in social phobia.

Among the limited studies that have assessed potential neuropsychological impairment in social phobia, one study found that SP individuals were more impaired than controls on measures of verbal attention, but not on measures of spatial attention (Asmundson, Stein, Larsen, & Walker, 1994). In contrast, a study by Cohen et al. (1996) found that SP individuals had lower Trail Making Test (TMT) performances (spatial working memory and set shifting) than controls. Based on these studies, SP individuals may be impaired on tasks of verbal attention, working memory, and set shifting, with spatial attention a domain requiring additional testing; conclusions based on two studies which are not in complete agreement, however, are tentative at best.

One major limitation in this area of work is the absence of studies reporting on neuropsychological performance in comorbid social phobia and major depression. Social phobia is frequently comorbid with major depression with estimates ranging from 44% to 70% (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Lecrubier & Weiller, 1997; Perugi, Frare, Toni, Mata, & Akiskal, 2001). According to several studies, social phobia presents a significant risk for the development of depression in non-depressed individuals (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992; Stein et al., 2001). Thus, clearer understanding of the relationship between these disorders and characteristics of their comorbid presentation are highly relevant. Given the prognosis for poorer educational outcomes with a comorbid presentation, neuropsychological functioning is an important focus of investigation.

Characterization of stress-related neuropsychological functioning in comorbid versus non-comorbid social phobia also may contribute to theories of cognitive and physiological response in social phobia. With regard to physiology, Gotthardt et al. (1995) report that depressed subjects exhibit increased heart rate in response to stress, but had a higher baseline heart rate than control subjects. In contrast, several studies have found that baseline heart rate does not differ for SP individuals versus controls, despite reports of increased anxiety and subjective experience of increased heart rate among those with social phobia (Edelmann & Baker, 2002; Grossman, Wilhelm, & Kawachi, 2001; Papageorgiou & Wells, 2002). While SP subjects appear to have relatively normal baseline heart rate function, several studies have documented elevated heart rate or hyperarousal in public speaking phobia during social stress (Clark and Agras, 1991; Heimberg, Hope, Dodge, & Becker, 1990; Levin et al., 1993), although mixed results have been found for stress-related hyperarousal in generalized social phobia (e.g., Gerlach, Wilhelm, Gruber, & Roth, 2001; Grossman et al., 2001; Hofmann, Newman, Ehlers, & Roth, 1995). Taken together, previous research suggests that comorbid subjects may exhibit elevated baseline *and* stressor heart response while non-depressed SP subjects may experience normal baseline heart rate with cardiac hyperarousal during social stress. Thus, comorbid subjects may experience less change in heart rate associated with stress and therefore show less effect from stress-related arousal. This scenario might yield the counterintuitive finding that comorbid depression may represent a protective factor for some autonomic response systems, like heart rate, during social stress.

Differential arousal during stress may also influence the cognitive appraisal of the stressor differentially for comorbid versus non-depressed SP participants. For example, a body of literature suggests that anxious cognition is characterized by vulnerability and attention to threat, while depressive cognitions focus on negative views of the self, world, and future (Beck, Emery, & Greenberg, 1985; Beck, Rush, Shaw, & Emery,

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