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Anxiety and salivary cortisol in symptomatic and nonsymptomatic panic patients and healthy volunteers performing simulated public speaking

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Abstract

Anxiety and salivary cortisol were measured in subjects performing simulated public speaking (SPS), a procedure that has been neurobiologically related to panic disorder. The subjects were divided into three groups: 18 symptomatic panic patients, 16 nonsymptomatic, drug-treated panic patients, and 17 healthy controls. In the experimental session, subjective anxiety (Visual Analogue Mood Scale) and the total score of the Bodily Symptom Scale (BSS) were higher in symptomatic patients than in controls, with nonsymptomatic patients in between. Measures of cortisol taken at home showed that the level was higher at 9:00 h than at 23:00 h in every group, indicating a normal circadian regulation of the hypothalamic–pituitary–adrenal (HPA) axis in panic patients. Also in every group, the level of cortisol was high at the beginning of the experimental session and decreased after 70 min. This fall parallels the decrease in anxiety and BSS ratings, and appears to reflect habituation of initial, anticipatory anxiety. Preparation and performance of speech raised anxiety and BSS scores to the initial levels, but failed to increase cortisol measured over 60 min, starting at the end of the speech. Therefore, SPS does not seem to activate the HPA axis, as reported in panic attacks.

Keywords: Simulated public speaking; Anxiety; Salivary cortisol; Daily rhythm; Panic disorder

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1. Introduction

The simulated public speaking (SPS) test is an experimental method of inducing anxiety in human subjects that was originally developed by McNair et al. (1982). In this test, the subject is requested to prepare a speech and then speak in front of a videocamera. The performance is recorded on videotape. At different phases of the experimental session, the subject fills self-evaluation rating scales that measure anxiety and other subjective states.

Epidemiological studies revealed that the fear of speaking in public is highly prevalent among students (Geer, 1965), and its prevalence is independent of gender, ethnic group or age (Phillips et al., 1997). Moreover, public speaking is the most frequent social fear (Furmark et al., 1999; Stein et al., 1994), being a hallmark of social phobia (Stein et al., 1996; Brunello et al., 2000). A comparative study showed that SPS enhanced anxiety in healthy volunteers irrespective of trait anxiety level while the Stroop Color–Word Test, another experimental method for inducing anxiety, was anxiogenic only in persons with high trait anxiety (Palma et al., 1994).

Several pharmacological studies have been carried out to compare the SPS test with another method of inducing experimental anxiety in humans, the conditioning of skin conductance response (CSCR). The latter measures the amplitude of skin conductance responses to a tone before and after pairing with a loud white noise; all sounds being delivered to the subject's ears through a headphone (Guimarães et al., 1991). The obtained results clearly show that the pharmacological profiles of the SPS and of the CSCR are different (for critical reviews, see Deakin et al., 1994; Graeff et al., 1996, 2003). The main findings are that benzodiazepine anxiolytics reduce CSCR (Hellewell et al., 1999), but do not affect SPS-induced anxiety, although baseline anxiety measured before and after the SPS challenge is decreased (Graeff et al., 1985; Guimarães et al., 1987, 1989; Zuardi et al., 1993). Similarly, the non-benzodiazepine anxiolytic buspirone has been shown to impair CSCR (Hellewell et al., 1999) without affecting SPS-induced anxiety (Guimarães et al., 1989). In addition, it has been reported that the serotonin-2-receptor blocker ritanserin reduced CSCR (Hensman et al., 1991), whereas the same drug prolonged the anxiety generated by SPS (Guimarães et al., 1997).

A parallelism has been drawn between the pharmacological profile of the two tests and of two anxiety disorders, namely generalized anxiety disorder (GAD) and panic disorder (PD). In the same way as CSCR, GAD is improved by low doses of benzodiazepine anxiolytics or by buspirone (Gorman, 2002). Moreover, the results of a double-blind clinical assay revealed that ritanserin was as effective as lorazepam for reducing the symptoms of GAD (Ceulemans et al., 1985). Conversely, the response of the SPS test to drugs approaches that of PD. In this regard, the enhancement of SPS-induced anxiety by a single dose of either chlomipramine (Guimarães et al., 1987) or nefazodone (Silva et al., 2001) has been related to the worsening that occurs during the initial phase of the antidepressant drug treatment of PD. Also, a correspondence has been established between the lengthening of SPS-induced anxiety caused by ritanserin (Guimarães et al., 1997) and the reported absence of therapeutic effect of the same drug on PD (Deakin et al., 1990; Den Boer and Westenberg, 1990). Furthermore, the 5-HT releaser and inhibitor of 5-HT reuptake D-fenfluramine has been found to markedly decrease SPS-induced anxiety (Hetem et al., 1996) and, accordingly, the results of an open clinical study (Solyom, 1994) and a case report (Hetem, 1996) have shown that fenfluramine reduces panic attacks in PD patients.

A neurobiological interpretation of the above evidence led to the hypothesis that SPS mobilizes the same neural networks that control panic attacks. These neural systems have evolved to organize defensive responses, such as fight or flight, to cope with threats that are very close or in actual contact with the organism (Blanchard and Blanchard, 1988). In particular, the periaqueductal gray matter (PAG) of the midbrain seems to be a critical structure for integrating defensive reactions to proximal threat (Fanselow, 1991). As a consequence, the PAG has been implicated in PD. Furthermore, an inhibitory role has been attributed to the serotonergic fibers that originate in the dorsal raphe nucleus of the midbrain and innervate the PAG, in such a way that an impairment of inhibition might result in increased vulnerability to panic attacks, whereas the strengthening of serotonin inhibition in the PAG may be a