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Autonomic instability during relaxation in panic disorder

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Abstract

The ability to relax was assessed in 14 patients with panic disorder (PD) and 15 non-anxious control subjects for 10 min. Before and after relaxation, subjects performed a standardized activating task of talking continuously for 4 min. The fractional decline in reported anxiety, tension, and alertness between the first talking period and the relaxation minimum did not differ between groups, although absolute levels of anxiety and tension were higher for PD patients. The fractional decline in skin conductance between the first talking period and the last minute of relaxation was less for PD patients than control subjects, while their increase in skin temperature was greater. Skin conductance showed a linear decline over the logarithm of relaxation time, the slope of which was less steep for PD patients. Goodness of fit of skin conductance over log time was also significantly poorer for PD patients. Heart rate levels or slopes did not differ between groups. Autonomic differences between PD and control subjects were largely due to six patients who reported having panic attacks during the test and higher pretest anxiety levels. In conclusion, indicators of relaxation were inconsistent. Skin conductance suggested autonomic instability during quiet sitting in patients who panic or who are prone to panic.

Keywords: Panic attacks; Skin conductance; Heart rate; Skin temperature

1. Introduction

The inability to relax is a common complaint of patients suffering from anxiety and worry, and is often treated by medications such as benzodiazepines or by various kinds of relaxation training such as Jacobson's 'progressive muscular relaxation' (Bernstein and Borkovec, 1973) or Öst's 'applied relaxation' (Öst, 1988). Congruent with their self-reports, anxious patients sitting quietly in the laboratory often exhibit a slower than normal decline in skin conductance (e.g. Lader and Wing, 1966; Roth et al., 1986, 1990). However, in many of these experiments, auditory sti-

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muli were delivered periodically in order to elicit orienting or defense responses, leaving an uncertainty as to whether anxious subjects would also have shown greater reduction in autonomic activation in the absence of such stimuli.

One reason for poor relaxation in patients with panic disorder (PD) could be the occurrence of panic attacks, which can appear at any time. Increases in anxiety or panic attacks have even been observed when anxious subjects were explicitly trying to relax (Heide and Borkovec, 1983, 1984; Adler et al., 1987). In fact, the absence of external distraction from feared somatic sensations or the direction of attention to these sensations as part of relaxation training may precipitate attacks. The physiological features of panic attacks were first described for three anxiety patients by Lader and Mathews (1970), all of whom exhibited impressively large surges in heart rate (30–50 b.p.m.) and smaller but distinct increases in skin conductance (3–12 μS). Patients prone to panic might also show less prominent signs of autonomic instability than those observed by Lader and Mathews, reflecting subclinical attacks or ones below the threshold of awareness.

We report here a comparison of PD patients and non-anxious control subjects on physiological measures of relaxation. Immediately prior to the relaxation period, subjects were given the task of describing their activities during the previous week for 4 min. The purpose of this was to bring subjects to comparable initial activation levels before relaxation began and to establish a benchmark against which to 'calibrate' responses of subjects (cf. Lykken et al., 1966). For the ensuing 10-min eyes-closed relaxation period, we estimated the speed, depth, and continuity of relaxation from physiological measures derived from skin conductance, heart rate, and skin temperature. We expected that PD patients compared to control subjects would show less decrease in anxiety and tension between talking and the end of relaxation, and that the same would be true for skin conductance, heart rate, and vasodilation as indexed by a rise in finger temperature (cf. Ackner, 1956). In addition, if panic attacks occurred, these physiological channels would register large increases in activation. Smaller increases in activation might be detected in non-specific skin conductance fluctuations or lack of fit of regression of autonomic data with time. Thus, our study differed from previous ones in several particulars: use of a standardized activating task before relaxation, lack of stimulation during the relaxation period, inclusion of a measure of cutaneous vasodilation as an index of relaxation, and detailed analysis of fluctuations in activation.

2. Methods

2.1. Subjects

Fourteen patients with PD were recruited by newspaper advertisements for medication studies or studies in which venipunctures for hormonal levels were done. Fifteen control patients were also recruited by an advertisement offering them US$50 to participate. They too underwent venipuncture. Diagnosis of patients and exclusion of anxiety disorders and other axis I disorders in control subjects were done according to a Structured Clinical Interview and DSM-IV criteria (American Psychiatric Association, 1994). Eligible subjects signed a consent form explaining the experiment described in this report prior to participation. All patients and control subjects denied having taken psychoactive or cardiovascularly active medication in the 2 weeks prior to testing. The two groups were well age- and sex-matched. The mean (S.D.) age of the patients was 41.6 (10.2) years, and that of the control subjects was 40.3 (12.6). Fifty-seven percent of patients and 60% of control subjects were women.

Before testing, subjects filled out a number of questionnaires including the Agoraphobic Cognitions Questionnaire (Chambless et al., 1984), Mobility Inventory (Chambless et al., 1985), Beck Depression Inventory (Beck et al., 1961), and State–Trait Anxiety Inventory-Trait (Spielberger et al., 1970). Physiological testing took place on a different day from venipuncture.

2.2. Apparatus

Physiological recording was done with the Kölner Vitaport System (Becker Meditech, Karls-