

CASE STUDIES AND CLINICAL  
REPLICATION SERIES

Panic Attack Associated with Perceived Heart Rate  
Acceleration: A Case Report

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A patient with uncomplicated Panic Disorder (without any phobic avoidance behavior) responded to false feedback of a sudden increase in heart rate with an unequivocal panic attack. Together with findings of a high prevalence of palpitations in naturally occurring panic attacks, this occurrence has implications for the possible etiology of "spontaneous" panic attacks.

Panic attacks are puzzling for the behavioral researcher because many patients insist that they experience at least some attacks as coming "out of the blue." If indeed panic attacks occur without any triggering events, these attacks would be difficult to explain by stimulus-response models and hard to treat by exposure therapy. Recent psychological approaches have, in general, assumed the existence of *internal* triggers for so-called spontaneous panic attacks (Clark, Salkovskis, & Chalkley, 1985; Margraf, Ehlers, & Roth, 1986; Barlow, in press). A central hypothesis of these models is a positive feedback loop between perceived physiological arousal and subjective anxiety that leads to an ascending "spiral" ending in the

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full blown panic attack. So far, this hypothesis has not been tested experimentally. We report the case of a patient in which an unequivocal panic attack was associated with false feedback of a sudden heart rate (HR) increase.

### CASE REPORT

Ms. A, a 25-year-old Caucasian woman, presented with a 9-year history of panic attacks but without complaints of significant phobic avoidance (Mobility Inventory, mobility alone: 2.3, Chambless, Caputo, Jasin, Gracely, & Williams, 1985). Using the Structured Clinical Interview for DSM-III—Upjohn Version (SCID-UP, Spitzer & Williams, 1983), Ms. A was diagnosed as having uncomplicated Panic Disorder. For the 8 days prior to her visit to our laboratory, she reported a total of 7 panic attacks on a diary form (with a maximum of two per day). There were no signs of depression or any other psychiatric problem (Beck Depression Inventory score of 0, Beck & Beck, 1972). Ms. A was in good physical health and worked full-time. Prior to participating in a treatment study at Stanford University and the Palo Alto Veterans Administration Medical Center, Ms. A gave informed consent to undergo a psychophysiological test battery that included a HR feedback paradigm.

The testing took place in an electrically-shielded, sound-attenuated chamber. Ms. A was told that she would receive feedback of her HR as registered by EKG electrodes in order to study the accuracy of her HR perception. The auditory feedback consisted of 1,000 Hz tone pips that were triggered by every R-wave of the EKG. To enhance the credibility of the feedback, the experimenter had Ms. A stand up while true, accurate feedback was being given and demonstrated how her HR changed with posture. Then Ms. A was left alone in the recording chamber. Between minute 0 and 5, the subject received accurate feedback of her HR. Between minutes 5:00 (minute:second) and 6:19, she received false HR feedback produced by a function generator set at her 5-minute HR level. Then, at 6:20 the frequency of the function generator was gradually increased by 50 beats per minute (bpm) over a period of 30 seconds. Similar HR increases have been found in naturally occurring panic attacks by Lader and Mathews (1970) and Cohen, Barlow, and Blanchard (1985). At 7:10 the experimenter gradually decreased the frequency of the function generator back to the original level (decrease of 50 bpm over 30 seconds). The paradigm lasted a total of 8 minutes during which HR and skin conductance level were continuously monitored. Automatic measurements of blood pressure and self-ratings of anxiety on a scale from 0 to 10 were taken at minutes 1, 3, 5, and 7.

While there were no significant changes in anxiety or physiological measures during accurate feedback, Ms. A showed a strong response to the false feedback. Within 20 seconds after beginning the false feedback of increasing HR, her actual HR began a surge of over 50 bpm. Immediately after the run, she told the experimenter that she had experienced

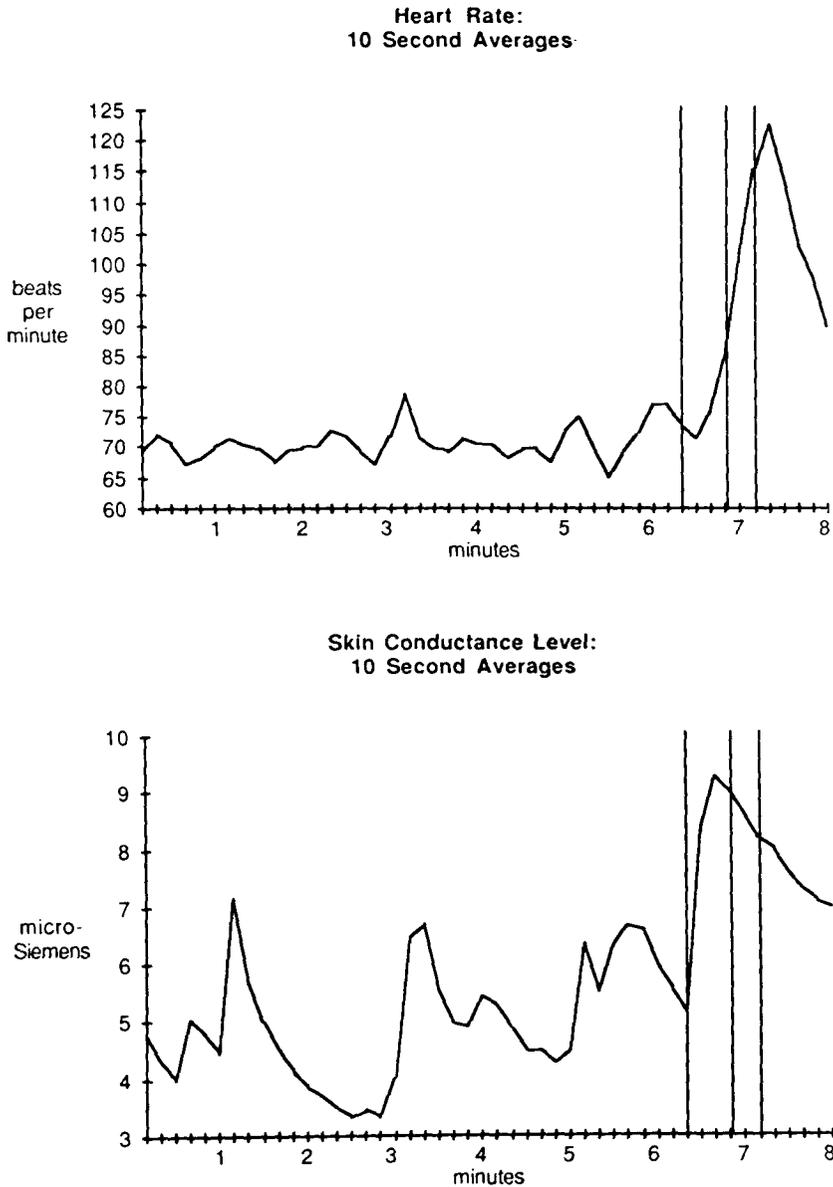


FIG. 1. Ten-second averages for heart rate and skin conductance level during accurate and false heart rate feedback. False feedback started at minute 5. The first vertical line indicates the beginning of false feedback of a 50 bpm increase, the second line, the end of the increase, and the third line, the beginning of the decrease back to baseline.

a panic attack at the time of the last blood pressure measurement. Her symptoms included sudden fearfulness, palpitations, dizziness, light-headedness, trembling, shaking, hot flashes, and chills. On her panic attack diary form, Ms. A recorded this experience as a major *spontaneous* panic attack with an intensity of 8 on a scale from 0 to 10. The state form of the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970) showed an increase from 30 (before any feedback) to 44 (after the end of the false feedback paradigm). During the first three assessments (accurate feedback), anxiety ratings (1, 1, 1) and blood pressure (99/60, 97/56, 100/61) remained stable. At the peak of the false feedback while Ms. A was experiencing panic, her blood pressure rose to 113/70 and her anxiety level from 1 to 6. Figure 1 shows 10-second averages of HR and skin conductance level. A debriefing session revealed that Ms. A had not doubted the accuracy of the HR feedback. The sudden increase, therefore, made her feel that she had lost control over her heart beat.

## DISCUSSION

Anxious subjects may experience panic attacks while being studied in a psychophysiological laboratory in the absence of any particular stressor (Lader & Mathews, 1970). What is the probability that Ms. A had a spontaneous panic attack that just happened to coincide with delivery of the false feedback? By her report, she had an average of a little under one attack, and a maximum of two attacks, per day, so in any given one-minute period an attack would be highly unlikely. Furthermore, our laboratory setting in itself does not seem to induce panic attacks. No attack occurred during the 15-minute baseline of this patient, or during more than 200 such baselines of other untreated panic attack patients. Thus, it seems probable that the perception of physiological arousal was indeed the trigger for a panic attack in this subject prone to panic. The fact that this perception did not have to be accurate emphasizes the role of cognitive factors.

The case reported here is consistent with the hypothesis of a positive feedback loop in the experience of panic, a hypothesis also supported by Hibbert's (1984) interview study. He found that the most common sequence of events reported for natural panic was the perception of an unpleasant bodily event followed by anxious catastrophizing cognitions and the full-blown picture of a panic attack. Beck (1985) reports similar findings in his patients. In the same vein, Ley (1985) observed that somatic symptoms preceded fear in the majority of patients interviewed. A heart rate increase is a plausible initiating event, since palpitations are the most common bodily symptom of panic attacks (Barlow, Vermilyea, Blanchard, Vermilyea, DiNardo, & Cerny, 1985; Margraf, Taylor, Ehlers, Roth, & Agras, in press). The regular presence of such initiating events would blur the distinction between "spontaneous" panic and other kinds of anxiety.

Although false feedback is one way of studying the positive feedback

loop proposed by most psychological theories of panic attacks, we do not mean to imply that panic attacks can regularly be induced by false feedback of heart rate or of any other signal. Our preliminary results indicate that unequivocal panic attacks are the exception rather than the rule for patients in our heart rate feedback paradigm, having occurred in only one of 11 patients so far. Such feedback is doubtlessly a rather imperfect experimental analogue of the complex interoceptive cues associated with a natural panic attack in an individual patient (Margraf et al., in press). The same imperfections exist in other so-called panic induction methods like lactate infusion (Ehlers, Margraf, & Roth, 1986) and carbon dioxide inhalation (Ehlers, Margraf, Roth, Taylor, Maddock, et al., 1986), which also rarely produce as clear-cut panic attacks as the one observed in Ms. A (for a review, see Margraf et al., 1986). In our laboratory only one out of 10 patients during lactate and one out of 24 during carbon dioxide have shown concurrent changes in heart rate and subjective anxiety of comparable abruptness and magnitude.

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