CLINICAL REPORT

Why Do Episodes of Panic Stop?

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Abstract—Episodes of panic are self-limiting—they all come to an end. However, it has not been explained how and why they end. Neither the cognitive nor the biological theory of panic deals with this phenomenon. As a first step toward an understanding of what stops a panic, we collected evidence about the self-limiting nature of these episodes. A semistructured interview was developed and conducted with 25 participants who had received a diagnosis of panic disorder. Participants reported a variety of triggers of panic, signs that a panic was ending, and strategies used to terminate panic episodes. A substantial proportion of participants indicated that there was a refractory period following panic episodes. The theoretical and therapeutic significance of this self-limiting feature of panic episodes is considered, as is the probable occurrence of a postpanic refractory period.

A striking but neglected fact about panic is that the episodes run a limited course; they do come to an end. A satisfactory explanation of why these episodes come to an end would be of considerable theoretical interest. The average duration of an episode of panic has been estimated to be in the range of 10 to 20 minutes (e.g., Barlow, 1988). However, this and other estimates are based on patients’ retrospective reports. Based on prospective diary reports that were supported by ambulatory monitor data, Taylor et al. (1986), found that these episodes had a much longer duration, with a mean of between 15.9 (SD = 7.4) and 49.2 (SD = 51.0) minutes.

A great deal of research has been devoted to the causes of these episodes, and two competing theories have been proposed to explain them. One is an
essentially biological theory, which proposes that episodes of panic arise from the firing, or misfiring, of a physiological suffocation alarm (Klein, 1993). The other theory, psychological in nature, proposes that episodes of panic are caused by a catastrophic misinterpretation of one’s bodily sensations (Clark, 1986). Neither theory deals directly with the question of why episodes of panic come to an end. It is not known why an alarm signaling the threat of suffocation should turn off. Nor is it known why a catastrophic misinterpretation comes to an end.

If panic is caused by a falsely triggered biological alarm then, presumably some form of automatic biological correction, comes into action; perhaps a homeostatic mechanism is involved. However, questions about what switches the alarm off, and when and why, have not been asked. One possibility is that the suffocation alarm triggers compensatory rapid breathing, which then turns the alarm off. (Indeed Papp, Klein, and Gorman (1993) have suggested that hyperventilation is more likely to be the result rather than the cause of panics.) An allied possibility is that the alarm triggers compensatory behavior, such as the frantic search for access to fresh air (e.g., rushing out of a theater). Conceivably, access to fresh air then turns off the alarm mechanism.

Following the cognitive theory of panic, presumably the panic should end immediately after the person’s catastrophic misinterpretation ends. This should happen either because the bodily sensations are reduced (e.g., heart rate resumes normal levels) or because the person changes the interpretation placed on these sensations. Cognitive theory would, therefore, predict that episodes are terminated when the provoking bodily sensations that are the basis for the particular catastrophic misinterpretation decline/disappear, and/or the catastrophic misinterpretation is removed/replaced (e.g., “Now that my heart rate has resumed a normal level, I’m not in danger of having a heart attack.”).

Tactics that might be adopted during an episode of panic include: Imposing a control that reduces the threat (e.g., slow breathing), escaping from the situation, and seeking authoritative help/reassurance that one is not in imminent danger (e.g., emergency medical attention). It follows from the psychological theory that information/events that fail to reduce the perceived threat, or even increase the perceived threat, should prolong the episode of panic. (The biological theory would presumably predict that information certainly, and most events, should have no effect on the duration of the panic).

A potential problem for the cognitive theory is to explain why a change in misinterpretation that successfully terminates an episode of panic should fail to be more effective in preventing recurrences.

Both theories need to explain why the average duration of a panic is 10 to 20 minutes (or 15–50 minutes, according to Taylor et al., 1986). More particularly, the two theories lead to differing expectations about the time course of repeated panics. The time required for a biological “switch” to “turn off” a panic should not vary much from panic to panic; it should remain relatively
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constant. The time required for a psychological ‘‘switch’’ to ‘‘turn off’’ a panic may well vary, and as the person becomes more adept at making safe interpretations, the duration of the episodes of panic should show a progressive decline.

In all cases of panic disorder, the mechanism that is involved in producing an episode of panic is retriggered on later occasions; the panics return. It follows that if a particular episode comes to an end because the catastrophic misinterpretation stops, the potential for a return of the catastrophic misinterpretation at a later date nevertheless persists. Similarly, if an episode ends because a suffocation alarm is turned off, that same alarm can be turned on (and trigger another episode of panic) at some later point.

Since the two competing theories are bound to make different predictions about what ends an episode of panic, an exploration of these factors would help to elucidate the more appropriate explanation of the disorder.

In addition to information about the termination of an episode of panic, an unexplored facet of this phenomenon is what happens immediately after a panic. While the two theories make predictions about the causes of panics and can be used to draw deductions about the cues or factors that might end an episode, neither makes any predictions or leads to any speculation about the period of time that immediately follows an episode. We know that refractory periods can follow physical exertion or intense emotional states (e.g., the refractory period that follows sexual activity in males; Kinsey, Pomeroy, & Martin, 1948) and also, at a more basic level, refractory periods follow neural firing (Hodgkin & Huxley, 1952). It seems reasonable to assume that a refractory period occurs following the emotional and physiological intensity of an episode of panic, and we, therefore, decided to search for evidence of such a refractory period. A postpanic refractory period could have implications for panic theory and also for therapy. Both the suffocation alarm theory and the cognitive theory may need to be expanded to explain such a phenomenon, since neither theory currently mentions it.

The purpose of the present study was to gather preliminary information about onset and offset cues and refractory periods following episodes of panic, as a first step to answering the main question of ‘‘what stops a panic?’’ To this end we interviewed 25 people about their experiences of panic, with particular attention to the onset and offset triggers, panic duration, and the postpanic period.

METHOD

Participants

Twenty-five people who had received a diagnosis of panic disorder were recruited from the University of British Columbia Fear and Anxiety Laboratory.
register of research volunteers for participation in the current study. Seventy-two percent of the subjects were female and the total sample had a mean age of 41.7 years ($SD = 12.7$). Subjects received no compensation for their participation.

**Interview Development**

A semistructured interview was developed, which included questions about panic onset and offset triggers, behavior intended to bring an episode to an end, panic duration, and feelings about safety following an episode. The results were then coded into several variables for analysis. Some of these categories (e.g., the offset cue of a “reduction in physiological symptoms”) were developed from our original hypotheses of panic cessation; others (e.g., the offset cue of “feeling a positive emotion”) were derived from common themes in participants’ responses.

**Procedure**

Subjects were contacted and participated in the interview by telephone. Each interview took approximately 30 minutes to complete.

**RESULTS**

Participants reported a variety of panic triggers and these were divided into four main categories (see Figure 1). More than half of all participants said that

![Fig. 1. What triggers episodes of panic? Number of endorsements made by participants of each type of panic trigger.](image-url)
their panics were triggered by their emotional state (e.g., “I will panic if I am feeling stressed or upset”). Almost half said that their panics were triggered by specific situations (e.g., going to the supermarket). A minority of subjects said that their episodes of panic were caused by physiological sensations or by their thoughts.

Participants described several offset triggers that signaled the end of an episode of panic (see Figure 2). Most participants said that a reduction in physiological sensations was the cue that signaled the end of a panic (e.g., “When my heart stops racing, I know the panic has ended”). Other frequently endorsed offset cues included feeling calm, experiencing a reduction in catastrophic thoughts, and feeling exhausted.

Participants also described several types of behavior that they used to speed the end of panic episodes (see Figure 3). Behaviors most commonly used to hasten the end of panic episodes were breathing exercises and refocusing (e.g., “I need to tell myself that everything is okay”). Other panic behavior included trying to calm down and escaping the situation.

Comparisons of reports of panic duration between participants’ “early panics” and “current panics” revealed that panic duration generally decreased over time from 83.0 (SD = 88.45) minutes to 56.5 (SD = 63.83) minutes (total episode duration), \( t(22) = 1.71, p < .10 \), and from 35.3 (SD = 44.48) minutes to 13.0 (SD = 10.09) minutes (peak episode duration) \( t(22) = 2.35, p < .05 \). Participants reported receiving an original diagnosis of panic disorder a mean of 7.3 (SD = 8.16) years ago.
Refractory Period

Over 64% of participants said that immediately after an episode has ended they feel safe from another panic for some period of time. Over half of these respondents said that they feel panic-safe for minutes or hours, and one third said that they feel panic-safe for days or weeks after an episode of panic has ended. Of all participants surveyed, 57% believed that they would experience a second panic if re-exposed to the trigger immediately after the first episode had subsided, and 52% reported actually experiencing consecutive panic episodes. These participants reported a mean duration of 31.4 minutes ($SD = 39.9$) between consecutive panic episodes.

DISCUSSION

The responses obtained from research interviews indicated that in explaining the onset and offset of panic episodes, people offer a range of explanations, including both biological and psychosocial triggers. Participants most often reported that their episodes of panic were triggered by emotional and situational stimuli, although psychological and cognitive stimuli were also reported to trigger the onset of panic episodes for a minority of the participants. In contrast, participants most often reported that triggers signaling the offset of a panic episode were reductions in physical symptomatology, with emotional states, reductions in panic cognitions, and exhaustion less often reported as offset cues.

Within this sample of people with panic disorder, the majority of whom have received pharmacological and/or psychological treatment for panic, a variety of strategies to cope with or make episodes of panic end were reported. Breathing exercises were most often reported as a method to end panic episodes, followed...
by refocusing, calming oneself, and removing oneself from the situation in which the onset of the panic episode occurred. Less commonly reported strategies were medication, visualization, and staying in the situation until the panic episode subsides. Finally, the majority of participants reported a refractory period during which they felt safe from another panic episode. Nonetheless, just over half of the participants in this sample also reported the belief that they would experience a second episode of panic if re-exposed to the onset trigger immediately following the offset of a panic episode, and half of the participants reported having experienced consecutive episodes of panic.

These results do not enable one to discriminate between the two theories of panic disorder. Both physiological and psychological onset cues were reported to trigger episodes of panic. However, emotional triggers were cited most frequently as leading to panic episodes.

The offset cues reported can be explained by either theory. For example, the suffocation alarm model of panic could explain that the perceived reduction in physiological symptoms (especially dyspnea) simply represents the termination of the suffocation alarm. Following the cognitive model of panic, a reduction in physiological symptoms would alter or remove the basis for cognition that the symptom represents some kind of imminent catastrophic event (e.g., a decrease in heart rate would dampen the cognition that the person is experiencing a heart attack). There is no longer a bodily sensation to misinterpret.

Similarly, the behavior reported by participants to end an episode of panic does not allow for discrimination between the two theories. It is interesting to note, however, that the two most frequently endorsed panic-reducing types of behavior are breathing exercises (a behavior that presumably attempts to reduce the discomforting physiological sensations) and refocusing (a behavior that presumably attempts to reduce or remove catastrophic cognitions).

There is enough evidence here to support the possibility of a postpanic refractory period in a substantial proportion of people with panic disorder. The existence of this period does not embarrass either theory (the refractory period can be viewed as a biological “recharging state” or a psychologically perceived period of safety) but this new fact will need to be accommodated. It seems possible that the two theories can be used to generate differing expectations about the refractory period. There is no obvious purely psychological reason why people should be nonresponsive after a panic, but the biological theory can accommodate the finding by pointing out that refractory periods are common in many biological systems. However, if the refractory period is indeed a “spontaneous” biological homeostatic correction mechanism, then we have reason to expect the duration of each refractory period to remain relatively constant across episodes. If refractory periods turn out to be of variable lengths, then this might encourage a psychological explanation (e.g., the durability of the particular corrective interpretations may well vary). The fact that panic-episode duration appears to decrease over time may also pose problems for the biological theory.

At this early stage, there is no obvious explanation for the evidence that
some people do not experience a refractory period after an episode of panic. This finding may pose an additional problem for the biological theory.

Since neither theory of panic makes any statement about events following an episode, it is difficult to speculate about what other results might discriminate between them. If the psychological theory explains a refractory period in terms of perceived safety, compared with a homeostatic correction mechanism in a more biological approach, exposure during a refractory period should be therapeutically beneficial according to a psychological model and irrelevant under a biological explanation of this phenomenon.

The presence of a postpanic refractory period would allow clinicians to do both cognitive restructuring and behavioral exposure to threatening stimuli (both internal bodily sensations and external threatening situations) during a period that is very likely to be panic-free. It is less clear whether a refractory period has any therapeutic implications for the suffocation alarm theory (this would depend on how such a period is explained by the theory).

A direct examination of postpanic refractory periods, including the collection of prospective data, is underway. We hope to address questions such as who is likely to experience such periods, how long they last, whether they vary in duration, what mechanism facilitates them and how can treatment procedures benefit from the utilization of post-panic refractory periods?

REFERENCES