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Introduction

Panic Disorder in Cross-Cultural and Historical Perspective

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PANIC DISORDER (PD), as currently conceived, is a medical condition that may be diagnosed when a person experiences recurrent, unexpected attacks of panic or anxiety, followed by persistent concern about having additional attacks or about losing control, going crazy, or having a heart attack. Panic attacks are intense periods of fear or discomfort, feelings that sometimes seem quite irrational. They are described as "attacks" because they often develop rapidly and include such symptoms as palpitations, sweating, trembling, shortness of breath, a feeling of choking, chest pain, nausea, dizziness, derealization or depersonalization, and numbness or chills or hot flushes, as well as fear of losing control or fear of dying.

PD, according to contemporary psychiatric classification, belongs to a group of neuropsychiatric conditions for which anxiety is the hallmark symptom. Although anxiety disorders are often thought of as relatively mild conditions, researchers estimate that in the United States these disorders account for 32 percent of the total economic costs of psychiatric illness, exceeding the costs associated with schizophrenia (21 percent) and mood disorders, including depression (22 percent) (Taylor 2000:4). Within the costs of panic attacks are emergency room visits and extensive medical tests to determine whether those experiencing the panic are suffering a heart attack or some other life-threatening condition as they fear. PD most commonly begins when the sufferer is between fifteen and thirty years of age. Studies suggest that between 1.5 and 3.5 percent of members of a population will suffer PD sometime during their lifetime.

PD was first recognized—or invented—as a distinctive form of mental illness in the 1970s; codified in 1980 as part of the third edition of the American Psychiatric Association's *Diagnostic and Statistical Manual*, or *DSM–III* (APA 1980); and popularized as a treatable clinical entity in the 1980s. Also in the 1980s, many researchers argued that PD had a largely physiological and genetic basis rather than a primarily psychological one. Since Freud, acute anxiety had been interpreted as resulting from unconscious feelings surfacing into partial awareness.[1] PD thus figures prominently in the history of the biological revolution in psychiatry. During the 1990s, PD was, to an important extent, reconceived by cognitive psychologists as spiraling episodes of anxiety arising from *catastrophic cognitions* that trigger physiological experiences of terror or panic. PD continues to be an important domain of research, clinical care, and pharmaceutical investment within psychiatry, both in the United States and globally. The story of PD is thus an intriguing chapter in the contemporary sociology of psychiatric knowledge and practice.

Acute anxiety and panic, however, quickly escape the confines of current diagnoses of anxiety disorders. They belong, on the one hand, to a long history of changing conceptualizations of neuropsychological distress in North American and European medical writing and practices, sharing complex relationships with categories such as cerebrocardiac neuropathy, irritable heart syndrome, neurasthenia, agoraphobia, anxiety reaction, anxiety neurosis, and neurasthenic neurosis. On the other hand, as Jackie Orr (2006) has recently shown in her book *Panic Diaries: A Genealogy of Panic Disorder*, acute anxiety and panic belong to a much broader cultural domain of changing experience and understanding in the twentieth-century United States—from the social panic produced by the 1938 radio broadcast of H. G. Wells' *War of the Worlds*, to concerns about measuring and managing fears and anxieties in American society, to David Sheehan's popular book *The Anxiety Disease* (1983), to multiple pharmaceutical interventions, clinical trials, and research studies sponsored by the National Institute of Mental Health (NIMH) and the pharmaceutical industry. The story of PD thus belongs to a very broad range of social, political, cultural, and medical concerns in Western societies.

What is little recognized, however, in most accounts of PD, whether medical writing, historical analyses, or cultural studies, is that acute anxiety and panic-like conditions appear in local social and cultural worlds in many parts of the globe. Psychiatric research has investigated cross-cultural differences almost exclusively in epidemiological terms—asking whether PD, defined uniformly across cultures, is more or less prevalent in one society or social class or subculture or risk group than in another, and concluding simplistically that "prevalence, course, gender

distribution, and age of onset of PD appear to be generally consistent throughout the world" (Taylor 2000:5). It is only recently that rich ethnographic research on acute anxiety and panic conditions has begun to provide real understanding of what *panic* and *panic disorder* may mean in local cultural worlds—for example, in the lives of Tibetan Buddhist refugees or contemporary Chinese psychiatric patients or Puerto Ricans suffering from *ataques de nervios*. Such research begins to provide a much deeper understanding of the relation of PD to culture, allowing questions to be asked, research to be conducted, and generalizations to be argued in ways that were impossible as recently as a decade ago.

This book is a collection of essays examining the complex relationships among culture, social conditions, and PD from three broad perspectives. The first chapters of the book are theoretical, developing a general framework for investigating the relationship of acute anxiety experiences to culture through an anthropology of the sensations, cultural phenomenology, theories of catastrophic cognitions, and ethnotheories of the body, illness, and healing. These essays make a strong argument that current psychological theories of panic attacks—which understand these attacks as resulting from an escalating cycle of catastrophic interpretations of bodily experience—provide a particularly rich basis for cross-cultural studies of panic and PD. The second set of chapters is historical, providing a partial cultural history of acute anxiety and panic in the West. The essays in this section make clear just how historically specific and contingent are current conceptualizations of PD, complicating enormously any effort to compare PD as an entity across time and space, as one might compare tuberculosis through social, cultural, and historical lenses. The third set of essays is positioned within cross-cultural psychiatry and medical anthropology, providing ethnographic and clinical accounts of PD and panic-like conditions in a number of specific settings. These chapters begin to answer questions about how panic attacks and PD may vary across cultures, and how PD as conceived by contemporary psychiatry relates to local idioms of distress, local categories of illness, and local moral worlds.

In this introduction we outline some of the critical issues and themes that emerge in the volume. We begin by reviewing the history of the professional biologization of PD, its conceptualization as a seizure-like entity minimally related to cultural or social context. We do so by tracing the history of the *DSM*, starting with the Sterling conference held in 1983. The next section describes the emergence of theories of the cause of PD that challenged this simple biologization, and outlines the theoretical ramifications of the most prominent of these theories: the catastrophic cognitions theory of panic. The final section gives an overview of the contributions of the current volume to advancing the study of PD in historical and cross-cultural perspective.

The Biologization of Panic Disorder in the 'DSM'

We begin by tracing the development of the *DSM–III* and *DSM–IV* conceptualization of PD, placing it in the context of theories of anxiety and panic in the 1980s and 1990s.

Reimagining Anxiety: From a Psychoanalytic to a Physiopsychological Perspective

In September 1983, nearly sixty researchers and administrators associated with the NIMH gathered in the Sterling Forest Conference Center in Tuxedo, New York, to discuss the state of the field of clinical and biological research about anxiety and anxiety disorders. Organized by the Clinical Research Branch of NIMH, the conference was designed to identify research issues, outline critical directions for new research, and stimulate broad scientific interest in the study of anxiety and anxiety disorders. The conveners of the conference argued that the major preoccupation of the psychiatric research community in the 1950s and 1960s was with schizophrenia, and in the 1970s, with affective disorders. They predicted confidently, however, that in the 1980s anxiety disorders would replace schizophrenia and affective disorders as the most critical site of progress in research in the neurosciences and psychiatry. The conference was designed both to stimulate and to give direction to that research, as a similar conference sponsored by NIMH had done for depression ten years earlier.

The edited book that resulted from the conference, *Anxiety and the Anxiety Disorders* (Tuma and Maser 1985), provides a sense of the field at the beginning of the 1980s. The initial chapters focus on basic biological and psychological research on anxiety, with papers on the neurobiology of anxiety and fear, on cognition and psychophysiology,[2] and on the use of animal models for research on fear and anxiety based in classic learning theory. The chapters that follow provide a broad picture of the state of clinical research on anxiety and anxiety disorders at that time. What the book fails to convey, however, is the excitement among participants at the meeting—the talk around the tables and in the corridors, the feeling of exhilaration about what emerging research was revealing, the powerful sense that the time was ripe for rapid progress in studying anxiety—and the place of PD in generating that excitement.[3]

It should be remembered that 1983 was only three years after the publication of the third edition of the *DSM* (APA 1980). In the context of psychiatric nosology, the *DSM–III* was a revolutionary document, purposely based in a *neo-Kraepelinian* or *descriptive* model of psychiatric classification and diagnosis, a model that eschewed efforts to classify disorders by their psychological causes in favor of establishing clear, symptom-based criteria that could be validated through empirical

investigation. (For a critical description of the neo-Kraepelinian movement, see Good 1992:182–187; for apologists' accounts, see Blashfield 1984; Klerman 1978; Weissman and Klerman 1978). The *DSM–III* represented a rejection of the previous theoretical framing of psychiatric classification of disorders in psychoanalytic terms, as represented by the *DSM–II*. Although advocates for the so-called neo-Kraepelinian approach claimed that the *DSM–III* was "atheoretical," it was solidly grounded in a medical- or biological-psychiatry paradigm. The *DSM–III* represented psychopathology as a set of discrete, heterogeneous disorders or diseases, based on the hypothesis that such disorders would ultimately be shown to result from pathologies of structure and function at the level of human neurobiology. This view stands in stark contrast with the psychoanalytic assumptions about subjectivity, personality, and psychopathology that framed much of the previous diagnostic manual, the *DSM–II*, particularly in its classification of the neuroses. The *DSM–III* represented symbolically a major paradigm shift within psychiatry, with diagnosis and classification, neurobiology, and pharmaceutical treatments all assuming new importance.

The emergent paradigm reflected in the *DSM–III* provided the organizing frame and the context for the Sterling Forest Conference. The participants exuded a sense of confidence that research on anxiety and anxiety disorders had finally begun to catch up with other areas of the neurosciences, that enormous progress was under way, and that PD was a critical site for demonstrating the value of the neo-Kraepelinian approach. Studies of neurotransmitters and the neuroendocrine system supplemented the classic fight-or-flight-response understanding of anxiety as a distinctive physiological system based in human evolution. Studies had begun to demonstrate the role of specific neurotransmitters to explain how benzodiazepines and other anxiolytic medications function and to provide a deeper understanding of the hypothalamic-pituitary-adrenocortical system. Basic research on learning, studied experimentally in animal models, was helping to explain, at the level of molecular biology, classic observations about relations among stressful stimuli, fear responses, arousal, habituation, and inhibition or disinhibition. These studies were being linked through cognitive psychology to clinical phenomena and newly emerging cognitive therapies for the anxiety disorders.[4] There was strong support for the categorization of anxiety disorders into five basic types—phobias, panic, generalized anxiety disorder, obsessive-compulsive disorder, and posttraumatic stress disorder (PTSD). The organizers and most participants in the meeting took it for granted that these categories are based in natural reality and that it would prove most fruitful to pursue research and clinical approaches that focus primarily on one or another of these discrete disorders.

But why was PD seen as so important to this field? Why was there such excite-

ment about PD, a sense that it would be *the* major "growth industry" in anxiety research in the 1980s, as Robert Spitzer, a driving force behind the *DSM–III*, predicted?[5] The discussion around the conference tables and in the dining room seemed to make clear what was only occasionally referred to in the scientific panels: not only was PD a newly recognized psychiatric disorder, especially promising for yielding scientific knowledge, but it also provided neo-Kraepelinian psychiatrists and neuroscientists with a unique opportunity to attack the stronghold of psychoanalysis. Whereas psychoanalysis had made little progress in decades of writing about anxiety and anxiety attacks, many at the conference felt that in a very short time biological psychiatry had made enormous scientific progress and promised far more to come. Only three of the forty-three scientific papers in the meeting addressed psychodynamic perspectives on anxiety, and the talk in the corridors was of psychoanalysts as dinosaurs, about to disappear.

PD provided a particularly powerful basis for arguing that the neo-Kraepelinian paradigm could advance understanding of acute anxiety, long considered the domain of the neuroses and psychoanalysis. There were three broad reasons for this claim. First, new evidence suggested that PD is a discrete, heterogeneous disorder, distinct from other anxiety disorders. Particularly interesting was the discovery that panic attacks do not respond to the benzodiazepines, typically used for anxiety, but respond specifically to the drug imipramine, usually considered an antidepressant (Klein 1980).[6] Klein had shown that imipramine was effective against spontaneous panic attacks but not against chronic anxiety, suggesting that panic attacks are a distinctive disorder (see Barlow 2002:125–126; McNally 1994:1–4).[7] Defining clear criteria for PD allowed researchers to investigate the specific biological, genetic, pharmacological, and epidemiological characteristics of this distinctive disorder.

In addition, Klein (1980) and Sheehan (1983) argued strongly that PD consisted of panic attacks that were largely *unprovoked* and experienced by sufferers as coming *out of the blue*. This formulation suggested that panic attacks are generated physiologically rather than psychologically, that they result from neurobiological processes rather than from the surfacing of unconscious psychological conflicts associated with seemingly unrelated stimuli. This argument was supported by findings of apparent physiological differences associated with PD, including intriguing research suggesting that infusion of sodium lactate would trigger panic attacks in persons suffering unmedicated PD, but not in normal populations.

Finally, a series of papers had recently argued that agoraphobia, a severe and little-understood condition, could be explained through conditioning-type learning theory as rooted in anxiety associated with those locations in which the person had suffered a terrifying panic attack, followed by the gradual development of an

irrational fear that leaving his or her house, particularly without the support of another person, might provoke another anxiety attack (Klein 1980). PD was thus conceived as *discrete*, as *unprovoked*, as having symptoms that map directly onto a *physiological substrate*, and as producing *secondary elaborations* that make sense of other distinctive anxiety disorders (see Good 1992:188–189). This might be called a *physiopsychology*, in which a physiological event is primary (it causes panic) and psychology—namely, the psychological state of fear—is secondary; and in this model, psychological process is reduced to conditioning: the pairing of fear to a certain place.

This brief account of a moment in the history of American psychiatry provides insight into current dominant view of PD, with its strong attachment both to criteria that define *panic disorder* as a discrete, heterogeneous condition and to the idea that panic attacks are unprovoked. In the next part of this section, we briefly describe the emergence of PD within the APA's diagnostic and statistical manuals, and in the following sections we suggest how recent psychological research, cross-cultural studies, and historical research challenge this dominant model.

Panic Disorder in the Subsequent 'DSM' Tradition

In recent theory, anxiety and fear are considered distinct emotions (for a review, see Barlow 2002). Fear is a primitive alarm in response to a perceived immediate danger. It leads to arousal; to activation of both the sympathetic and parasympathetic nervous systems, experienced in bodily sensations such as palpitations and sweating; and to certain action tendencies (freezing or fleeing). In contrast, "anxiety is considered to be a future-oriented emotion, characterized by perceptions of uncontrollability and unpredictability over potentially dangerous events" (Barlow 2002:104).

According to current thinking, in the psychology and psychiatry enshrined in the *DSM–IV* (published in 1994), there are five general domains of distinct anxiety disorders:

- *Generalized anxiety disorder*, characterized by excessive worry about current life problems and future events, leading to muscular tension and other symptoms
- *Posttraumatic stress disorder*, marked by constant arousal and reactivity to any reminder of past traumas, as well as a tendency to reexperience past traumas as though they were current
- *Obsessive-compulsive disorder*, identified by contamination fears and a compulsion to repeat certain behaviors, especially checking behaviors
- *Phobias*, characterized by unreasonable levels of fear concerning objects, places, or situations, and anxiety about contact with these[8]

- *Panic disorder*, defined by episodes of acute fear or anxiety (with enough symptoms to constitute a "panic attack"), the fear often focusing on concerns of dying from internally arising bodily dysfunction.

Although PD first appeared as a diagnosis in *DSM–III* (1980), experiences of acute anxiety, with physical symptoms similar to those that serve as criteria for panic attacks, have a longer history in the APA's diagnostic manuals. In the *DSM–I*, published in 1952, what is now called PD was diagnosed most commonly as either an *anxiety reaction*, defined as a general state of fear with frequently associated somatic symptomatology, or as one of the *psychophysiologic autonomic and visceral disorders*. In *DSM–II*, published in 1968, the term *anxiety reaction* was replaced with *anxiety neurosis*, and *psychophysiologic autonomic and visceral disorders* was replaced by *psychophysiologic disorders*. Only in the *DSM–III*, published in 1980, did *panic disorder* become identified as a separate psychiatric entity. The diagnosis of *agoraphobia* first appeared in *DSM–III*, along with PD.

Table 1.1 outlines criteria for a PD diagnosis in the *DSM–III*. Criterion C specified that a PD diagnosis could not be made in the presence of other diagnostic entities. Thus, according to the *DSM–III*'s hierarchical model of diagnosis, when PD co-occurred with another disorder, it was to be understood as an epiphenomenon,

Table 1.1 Diagnostic criteria for panic disorder in the *DSM–III*

A. At least three Panic Attacks within a three-week period in circumstances other than during marked physical exertion or in a life-threatening situation. The attacks are not precipitated only by exposure to a circumscribed phobic stimulus.

B. Panic Attacks are manifested by discrete periods of apprehension or fear, and at least four of the following symptoms appear during each attack:
   (1) dyspnea
   (2) palpitations
   (3) chest pain or discomfort
   (4) choking or smothering sensations
   (5) dizziness, vertigo, or unsteady feelings
   (6) feelings of unreality
   (7) paresthesia (tingling in the hands or feet)
   (8) hot or cold flashes
   (9) sweating
   (10) faintness
   (11) trembling or shaking
   (12) fear of dying, going crazy, or doing something uncontrolled during an attack

C. Not due to a physical disorder or another mental disorder, such as Major Depression, Somatization Disorder, or Schizophrenia.

SOURCE: *Diagnostic and Statistical Manual of Mental Disorders*. 3rd. ed. [*DSM–III*.] Pp. 231–232. Copyright 1980, American Psychiatric Association. Reprinted with permission.

a symptom of another condition and an indicator of illness severity rather than a unique entity (Baker 1989; Carey 1985). Major depressive disorder, for example, was higher in the hierarchy than PD, so PD could not be diagnosed in a person who had major depressive disorder.[9]

In the *DSM–III–R* (the revised edition of *DSM–III*, published in 1987), five major changes were made in the PD category (see Table 1.2):

1. No longer did three panic attacks have to be experienced in a period of three weeks to meet criteria; instead, a person was required to have suffered only one panic attack in the preceding month.
2. Nausea was added to the symptom list. The delay in including nausea in the list of panic attack symptoms reflects the dominance of cardiac symptoms and

Table 1.2 Diagnostic criteria for panic disorder in the *DSM–III–R*

A. At some time during the disturbance, one or more Panic Attacks (discrete periods of intense fear or discomfort) have occurred that were (1) unexpected, i.e., did not occur immediately before or on exposure to a situation that almost always causes anxiety, and (2) not triggered by situations in which the person was the focus of others' attention.

B. Either four attacks, as defined in criterion A, have occurred within a four-week period, or one or more attacks have been followed by a period of at least a month of persistent fear of having another attack.

C. At least four of the following symptoms developed during at least one of the attacks:

(1) shortness of breath (dyspnea) or smothering sensations

(2) dizziness, unsteady feelings, or faintness

(3) palpitations or accelerated heart rate (tachycardia)

(4) trembling or shaking

(5) sweating

(6) choking

(7) nausea or abdominal distress

(8) depersonalization or derealization

(9) numbness or tingling sensations (paresthesias)

(10) flushes (hot flashes) or chills

(11) chest pain or discomfort

(12) fear of dying

(13) fear of going crazy or doing something uncontrolled

D. During at least some of the attacks, at least four of the C symptoms developed suddenly and increased in intensity within ten minutes of the beginning of the first C symptoms noticed in the attack.

E. It cannot be established that an organic factor initiated and maintained the disturbance, e.g., amphetamine or caffeine intoxication, hyperthyroidism.

SOURCE: *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed., rev. [*DSM–III–R*.] Pp. 237–238. Copyright 1987, American Psychiatric Association. Reprinted with permission.

shortness of breath in twentieth-century Western scientific theories of panic etiology, and the neglect of dizziness-type complaints and nausea. The latter two complaints are particularly prominent in Asian populations, as essays in this volume demonstrate (see, for example, Chapters 7, 8, and 10).

3. It was specified that to be considered a panic attack, at least four symptoms must have developed suddenly, and increased in intensity to a state of panic within ten minutes.
4. A diagnosis of PD could be made even if another disorder was present; for example, a person could be diagnosed as suffering both PD and major depressive disorder. This represented a shift from a hierarchical to a comorbidity view of illness, a shift from a model in which one primary disorder generated all the other so-called secondary symptoms to a model of multiple, simultaneously occurring disorders. (However, as the next paragraph shows, the "cue" criteria led to PD not being diagnosed in the presence of PTSD.)
5. In *DSM–III–R*, the panic attacks in PD were defined as "uncued," that is, as unprovoked eruptions of anxiety. Only one type of panic attack in PD was thought to be provoked by a cue: agoraphobia-type panic attacks, in which being in certain external spaces, such as a mall, might trigger a panic attack, in essence constituting "place-caused" panic. This insistence on the uncued nature of panic attacks in PD implied that panic attacks in the context of PTSD could not be considered PD-type panic attacks. That is, if a patient feared death or insanity from the symptoms (for example, palpitations or racing thoughts) caused by encountering some reminder of a trauma event—that is, a trauma cue—such as someone resembling a perpetrator or a place similar to the location of the trauma, and if that fear escalated to panic, the panic attack could not be classified as PD in type.

In the *DSM–IV*, published in 1994, the criteria defining PD were minimally changed (see Tables 1.3 and 1.4). As in the previous edition, a person must experience "recurrent, unexpected panic attacks" that develop suddenly in less than ten minutes. Here we have the widely debated out-of-the-blue and rapid-crescendo criteria that configure panic as a sort of periodic seizure, unrelated to stimuli that awaken hidden conflicts or to cognitions about concurrent actions or bodily states. In *DSM–IV* (as in *DSM–III* and *DSM–III–R*) one type of triggering cue is allowed in defining panic attacks: the cue associated with agoraphobia. Being in certain places or situations, including being outside the home alone, being in a crowd, standing in a line, being on a bridge, or traveling in a bus, train, or automobile are considered *situationally predisposed panic attacks*. When these situations trigger panic attacks, the disorder is classified as PD with agoraphobia (see Table 1.5).

Table 1.3 Diagnostic criteria for panic disorder in the *DSM–IV*

A. Both (1) and (2):

(1) recurrent unexpected Panic Attacks

(2) at least one of the attacks has been followed by one month (or more) of one (or more) of the following:

(a) persistent concern about having additional attacks

(b) worry about the implications of the attack or its consequences (e.g., losing control, having a heart attack, "going crazy")

(c) a significant change in behavior related to the attacks

B. The Panic Attacks are not due to the direct physiological effects of a substance (such as a drug of abuse, a medication) or a general medical condition (such as hyperthyroidism).

C. The Panic Attacks are not better accounted for by another mental disorder, such as Social Phobia (e.g., occurring on exposure to feared social situations), Specific Phobia (e.g., on exposure to a specific phobic situation), Obsessive-Compulsive Disorder (e.g., on exposure to dirt in someone with an obsession about contamination), Posttraumatic Stress Disorder (e.g., in response to stimuli associated with a severe stressor), or Separation Anxiety Disorder (e.g., in response to being away from home or close relatives).

SOURCE: *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. [*DSM–IV*.] Pp. 401–402. Copyright 1994, American Psychiatric Association. Reprinted with permission.

Table 1.4 Diagnostic criteria for panic attack in the *DSM–IV*

A discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes:

(1) palpitations, pounding heart, or accelerated heart rate

(2) sweating

(3) trembling or shaking

(4) sensations of shortness of breath or smothering

(5) feeling of choking

(6) chest pain or discomfort

(7) nausea or abdominal distress

(8) feeling dizzy, unsteady, lightheaded, or faint

(9) derealization (feeling of unreality) or depersonalization (being detached from oneself)

(10) fear of losing control or going crazy

(11) fear of dying

(12) paresthesia (numbing or tingling sensations)

(13) chills or hot flashes

SOURCE: *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. [*DSM–IV*.] P. 395. Copyright 1994, American Psychiatric Association. Reprinted with permission.

Table 1.5 Diagnostic criteria for agoraphobia in the *DSM–IV*

A. Anxiety about being in places or situations from which escape might be difficult (or embarrassing) or in which help may not be available in the event of having an expected or situationally predisposed Panic Attack or panic-like symptoms. Agoraphobic fears typically involve characteristic clusters of situations that include being outside the home alone; being in a crowd or standing in a line; being on a bridge; and traveling in a bus, train, or automobile.

B. The situations are avoided (e.g., travel is limited) or else are endured with marked distress or with anxiety about having a Panic Attack or panic-like symptoms, or require the presence of a companion.

C. The anxiety or phobic avoidance is not better accounted for by another mental disorder, such as Social Phobia (e.g., avoidance limited to social situations because of fear of embarrassment), Specific Phobia (e.g., avoidance limited to a single situation such as elevators), Obsessive-Compulsive Disorder (e.g., avoidance of dirt in someone with an obsession about contamination), Posttraumatic Stress Disorder (e.g., avoidance of stimuli associated with a severe stressor), or Separation Anxiety Disorder (e.g., avoidance of leaving home or relatives).

SOURCE: *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. [*DSM–IV*.] P. 396. Copyright 1994, American Psychiatric Association. Reprinted with permission.

As did the previous editions, the *DSM–IV* continues a sharp distinction between PD and PTSD, owing to the insistence on the untriggered nature of panic attacks in PD. Thus, if a panic attack is triggered by a trauma cue—for example, an experience that provokes a memory of a traumatic event, followed by physiological symptoms of anxiety and concerns about a heart attack—the episode is not considered a PD-type panic attack. According to the *DSM–IV*'s definition of PD, such hybrid entities do not meet PD criteria, but rather should be classified simply as PTSD.

One important change in the *DSM–IV* should be noted. The diagnosis of panic attacks was separated out from the diagnosis for PD. This reflected a growing realization that panic attacks were found in many disorders (for a discussion, see Chapter 6 in the present book), a realization that has continued to grow.

In the current edition of the psychiatric diagnostic manual, *DSM–IV–TR* (2000), the criteria for PD are unchanged from *DSM–IV*. The *DSM–V* Work Group on Panic Disorders has been meeting, but with no indication to date of any substantial changes from current criteria.

Recent Findings and Theories Challenging the 'DSM' Conceptualization of Panic Disorder

In this section we outline recent critiques of the *DSM–IV*'s biological conceptualization of PD, and the emergence of a psychological view of panic's origin. We discuss the important implications of a catastrophic cognitions theory in respect to historical and cross-cultural variations in panic.

Controversies Surrounding the Definition of Panic Disorder

Several elements in the *DSM* definition of PD have been particularly controversial. These elements relate in large measure to efforts to constitute PD as an autonomous, largely physiological disease entity. First, there remain important questions about the discrete set of anxiety symptoms listed as diagnostic criteria. In some cultural contexts, acute anxiety symptoms other than the thirteen listed in the *DSM–IV* take center stage. For example, among Cambodian refugee populations, tinnitus is called "*khyâl* shooting from the ears" (*khyâl choenh pii troechia*), and this somatic event is thought to indicate bodily weakness and a disorder in the "*khyâl* physiology." (According to the Khmer ethnophysiology, *khyâl* is a windlike substance that courses through blood vessels.) Tinnitus is a common—and personally and clinically important—complaint during anxiety and panic states in this group (Hinton et al. 2006a, in press). In addition, *DSM–IV* places certain symptoms together under a single criterion (such as feeling "dizzy, unsteady, lightheaded, or faint," or having "chills or hot flashes"), suggesting that they are somehow equivalent. Data suggest that these symptoms may be symbolically equivalent in one society but not in another, and that the experience and categorization of symptoms may differ across cultures in important ways.

Second, specific questions about the defining characteristics of panic attacks remain unresolved. For example, the research reported in this book suggests that it is arbitrary to require that for an experience of anxiety to qualify as a panic attack the anxiety must start abruptly and reach a peak in ten minutes. If a person feels increasingly anxious over a longer period—for example, over the course of an hour, finally reaching a state of panic—the *DSM–IV* would define this experience as not meeting the criteria for a panic attack. Findings presented in this book provide evidence that across cultures there are important variations in the pattern of onset of anxiety, raising serious questions about this criterion.

Third, in *DSM–IV*, the presence of PTSD excludes a diagnosis of PD. Criterion C (see Table 1.3) states that panic attacks should not be better accounted for by another disorder, specifying that the panic should not be a response to a "stimulus" associated with a traumatic event. If such a stimulus—for example, dizziness—both recalls a past traumatic event (such as being beaten on the head by police or soldiers in settings of conflict or torture) and causes thoughts about imminent bodily disaster, such as a stroke, the clinician should make the diagnosis not of PD but of PTSD. To this extent *DSM–IV* remains hierarchical, indicating that if one diagnosis (PTSD) is present, another diagnosis (PD) cannot be made. In respect to PD, the *DSM–IV* proscribes hybrid or comorbid entities. The relations among memories of trauma, panic attacks, PD, and PTSD raise important questions for empirical, cross-cultural research, and the exclusion of comorbid diagnoses of PTSD and PD remains controversial.

Fourth, in order to meet *DSM–IV* PD criteria, the panic attack should be

"unexpected," that is, come out of the blue (see criterion A, number 1 in Table 1.3). Such a view, as we have suggested, aims to characterize a panic attack as an autonomous physiological process, a sort of seizure of the nervous system. In fact, most psychological researchers today think that panic is frequently triggered rather than unexpected; the anthropological data would also suggest this to be the case, as we note later and as the research reported in this book shows.

These rather straightforward controversies point to much more essential questions for the field. Are panic attacks indeed physiological eruptions, akin to a seizure, or are they (sometimes? always?) far more psychologically motivated and organized? Are there fundamental differences across cultures in the experience of panic attacks that raise important doubts about the universality of current diagnostic criteria? Can panic attacks be initiated by events or experiences that trigger fears or anxieties through hidden semantic networks or psychological associations that are out of awareness or unconscious? Indeed, are the panic attacks that occur in trauma disorders and PD (always? sometimes?) heterogeneous, or are they overlapping conditions? One of the goals of this book is to provide the best examples of cross-cultural, ethnographic, and clinical research that speak to questions such as these.

The Emergence of a Psychological Theory: Catastrophic Cognitions and Panic Disorder

There has long been a tension between theorizing panic attacks in neo-Kraepelinian terms—as a distinct pathological entity, largely understood as spontaneous biological events—and a more psychological interpretation of panic attacks. The Sterling Forest Conference, which occurred right after the publication of the *DSM–III*, represented a moment in which powerful claims were made about the adequacy of a biological understanding of panic attacks—claims that panic attacks are spontaneous physiological events in persons genetically predisposed to such a disorder. This noncultural, nonpsychological view of the core processes generating PD would continue in *DSM–IV*, as we have described.

Any modeling of panic attacks in PD also had to respond to observations that panic attacks occur in many *DSM*-defined disorders. Some panic attacks are "stimulus bound" (phobias) or "situationally predisposed" (social phobias), and memories (particularly traumatic memories) may trigger panic attacks or experiences of acute anxiety (PTSD). Modeling of panic attacks in PD required understanding the process of the rapid crescendo of panic symptoms, the focus on the body and fear of dying, or the worry that one was out of control and might be going crazy. Even the strongest proponents of a biological approach to understanding PD were required to introduce psychology—usually via behavioral learning theory and cognitive psychology, namely, *conditioning* theories—to understand the clinical phe-

nomena, such as anticipatory anxiety and agoraphobia, even as they were claiming that biological psychiatry provided a clear, scientific alternative to psychoanalytic understandings of acute anxiety.

Over time, the hypothesis that panic attacks in PD were primarily unprovoked, seizure-like events was challenged empirically, leading to the development of more robust psychological theories of PD. The development of theories of catastrophic cognitions, in particular, has been extremely influential and has led to very different models for understanding and treating PD than were present in 1983.

Critiques of the Biological Explanations of the Laboratory Induction of Panic Attacks in Panic Disorder Efforts to isolate physiological processes that cause panic have a long history, such as theories that focused on a heart etiology in the Civil War and World War I (see Chapters 4 and 5). One important hypothesis, which in 1983 was still referred to as evidence for a biological theory of PD, suggested that lactate might play a critical physiological role. In an influential research report, Pitts and McClure (1967) demonstrated that lactate caused panic among persons with what was called *effort syndrome:* when they injected those patients with lactate, acute episodes of headache, dizziness, faintness, weakness, and chest tightness, resulting in panic, followed. In the 1970s, researchers working within a cognitive paradigm challenged this purely biological formulation. In an article published in 1974, Ackerman and Sachar questioned the validity of the lactate theory of panic and hypothesized that panic was not produced by the effects of lactate (or of lactate-induced low calcium) in the brain, but rather by catastrophic cognitions about lactate-induced symptoms. Cold extremities associated with lactate injection could result in fear of stroke, they argued. Pitts and McClure and other cognitive theorists suggested that PD patients are hyperreactive to a wide spectrum of agents other than lactate, including carbon dioxide, yohimbine, and norepinephrine. According to cognitive theorists, the most parsimonious explanation for this broad reactivity was hypersensitivity to somatic sensations, irrespective of the method of induction (Barlow 2002:171, 178–179).

Panic Attacks as Triggered by Catastrophic Cognitions About Somatic Sensations In the 1980s, cognitive theorists increasingly argued that the *DSM*'s out-of-the-blue criterion—that true panic attacks are unprovoked—should be eliminated (for reviews, see Beck 1988; Craske 1991; McNally 1994; Rapee et al. 1995; Street et al. 1989). Instead, they argued, catastrophic cognitions about bodily sensations constitute a core process in provoking panic attacks, and the feared bodily sensations that provoke panic may be induced by a wide range of "triggers." Clark (1986:462) summarized this catastrophic cognitions theory of panic as follows (see Figure 1.1):

> The trigger for an attack often seems to be the perception of a bodily sensation which itself is caused by a different emotional state (excitement, anger) or by some

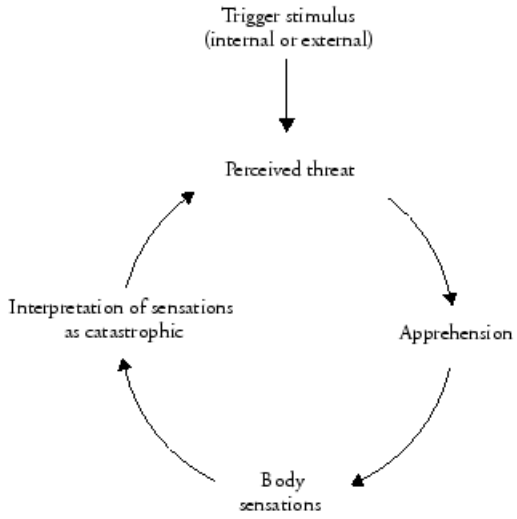

Figure 1.1 A cognitive model of the generation of a panic attack, as depicted by Clark (1986)

quite innocuous event such as suddenly getting up from the sitting position (dizziness), exercise (breathlessness, palpitations) or drinking coffee (palpitations). Once perceived, the bodily sensation is interpreted in a catastrophic fashion and then a panic attack results.

Other common ways in which sensations might be induced, and which then trigger panic attacks, were also explored, for example, hyperventilation, which causes a host of bodily sensations, including blurry vision and hand numbness (Beck 1988); and temperature and humidity changes (such as those resulting from entering a sauna or moving from a warm to a cold space), which cause somatic symptoms such as sweating or cold extremities (Rapee et al. 1995).

Theoretical Implications of the Catastrophic Cognitions Theory of Panic Disorder If the catastrophic cognitions theory of panic (which is often called the *cognitive theory of panic disorder*) is valid, it has important implications in respect to the nature of PD and its historical and cross-cultural variability. Following are several hypotheses that emerge from a catastrophic cognitions theory of the generation of PD that have particular significance for investigating cross-cultural differences in the phenomenology and distribution of panic attacks and PD.

HYPOTHESIS 1: *The severity of catastrophic cognitions should predict panic severity and frequency.* According to the catastrophic cognitions theory of panic, the more severe a person's catastrophic cognitions about particular sensations are, the greater

should be the frequency and severity of panic attacks. In support of this hypothesis, multiple studies demonstrate that the severity of catastrophic cognitions about panic sensations is strongly related to the severity and frequency of PD panic attacks (for a review, see Hinton et al. 2006b). This would suggest a close relationship between cultural interpretations of the danger of particular sensations and rates of PD associated with those sensations.

HYPOTHESIS 2: *Decreasing catastrophic cognitions should improve PD.* According to the catastrophic cognitions theory of panic, treatments that reduce a person's catastrophic cognitions about panic sensations should decrease the frequency and severity of PD panic attacks. In support of this hypothesis, multiple studies illustrate that reducing catastrophic cognitions of PD patients is at least as effective as medication in allaying the severity and frequency of PD panic attacks (for reviews, see Barlow 2002; McNally 1994; Taylor 2000). This finding provides further evidence that catastrophic cognitions occurring during PD panic attacks, which would be expected to vary by culture (see Hypothesis 3), are some of the very "cogs" of the disorder.

HYPOTHESIS 3: *The emphasized symptoms of PD panic attacks will vary across cultural groups and historical periods.* According to the catastrophic cognitions theory of panic, the sensations most prominent in PD panic attacks will vary depending on which sensations are viewed as potentially catastrophic by members of a society or social group. Given local illness concepts and syndromes, certain bodily sensations will be viewed with more fear, and those symptoms will form the critical symptoms associated with panic in those contexts.[10] The main symptoms focused on by persons suffering PD panic attacks should vary by cultural groups and historical periods, as well as across individuals within particular cultural groups and historical periods.

HYPOTHESIS 4: *The events or actions that trigger the sensations that cause PD panic attacks will vary across cultural groups and historical periods.* According to the catastrophic cognitions theory of panic, in different individuals, cultural groups, and historical periods what induces the feared sensations and triggers PD-type panic attacks may vary. Given local illness concepts and syndromes, specific bodily sensations will be viewed with more fear in certain situations, such as upon going into a crowded mall, upon engaging in worry, upon standing up, or upon going outside and being hit by a strong wind when feeling weak.[11] Put another way, the events and actions that trigger panic will vary radically by cultural group and historical period.

HYPOTHESIS 5: *PD catastrophic cognitions will vary across cultural groups and historical periods.* According to the catastrophic cognitions theory of panic, the catastrophic cognitions or modes of rationality associated with panic attacks may

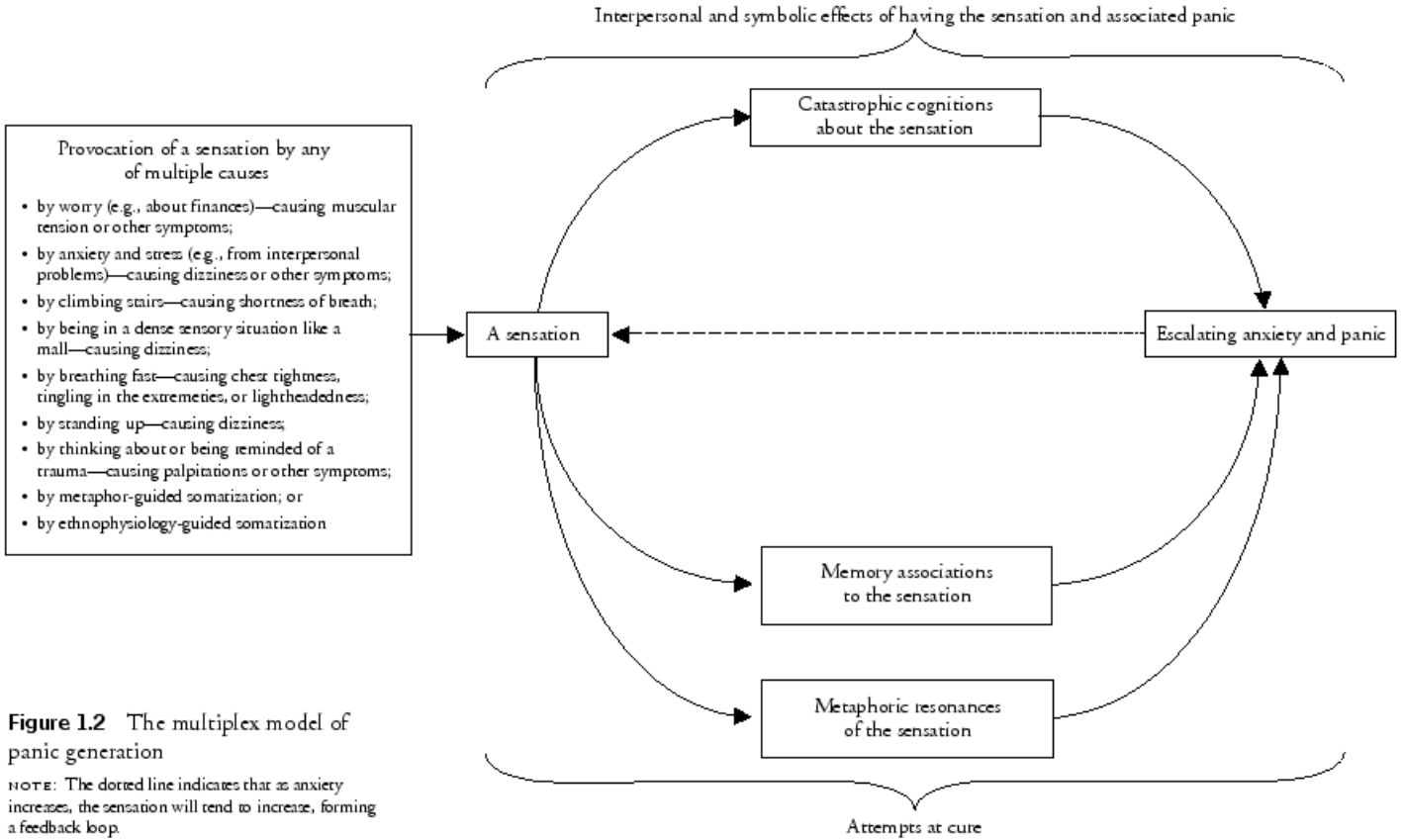

Figure 1.2 The multiplex model of panic generation

NOTE: The dotted line indicates that as anxiety increases, the sensation will tend to increase, forming a feedback loop.

vary depending on local ideas about human physiology and on local cultural syndromes. As conceptualizations of the body, its physiology, and illness syndromes have shifted, so too have the nature of catastrophic cognitions and the structure of the panic ontology. Catastrophic cognitions will thus vary significantly across cultures, cultural subgroups, and historical periods, depending on the regnant syndromes and ethnophysiologies.

HYPOTHESIS 6: *The onset of PD panic attacks may be relatively prolonged.* According to the catastrophic cognitions theory of panic, episodes of PD panic attacks are not seizure-like events. They may crescendo rapidly (in less than ten minutes, as specified in the *DSM–IV* criteria) or they may crescendo more slowly, even over several hours, worsening as the patient's catastrophic cognitions worsen. In fact, a recent study indicates that the ten-minute criterion should not even be applied to English-speaking North Americans (see Scupi et al. 1997).

HYPOTHESIS 7: *Hybrid panic attacks should occur that combine PTSD and PD characteristics.* According to the catastrophic cognitions theory of panic, PD panic attacks should frequently co-occur with trauma-related disorder. Encountering a trauma-related stimulus (such as conditions resembling the location of the original trauma) or thinking about a trauma may trigger various sensations. These sensations may in turn be cognized or interpreted as threatening and potentially catastrophic. For example, palpitations may recall a traumatic event in which the person experienced strong palpitations, and palpitations may concurrently evoke catastrophic cognitions (for instance, those of a heart attack). There is evidence that such hybrid panic attacks are common (see, for example, Hinton et al. 2006b). How these hybrid panic attacks occur can be depicted by a multiplex model such as that represented by Figure 1.2 (for further discussion of the components of this model, see Chapter 3).

Contributions of This Volume

Many of the chapters in the current volume draw on a catastrophic cognitions theory of panic and explore one or more of the hypotheses just outlined. The chapters provide support for these proposed hypotheses, illustrating the heuristic value of a catastrophic cognitions approach to the study of panic across cultural contexts and historical periods. Several chapters illustrate the great variability in the symptoms that form the core symptom of PD-type panic attacks; they demonstrate how historically and culturally varying syndromes and ethnophysiologies give rise to catastrophic cognitions that produce panic, and they demonstrate the hybrid nature of panic, linking panic and trauma symptoms. This approach thus provides one model for making psychological and cultural *processes* central to investigations of culture and PD, rather than focusing more narrowly on cultural variability in symptom criteria and diagnostic entities.

As we have already suggested briefly, the catastrophic cognitions approach may be integrated with an understanding of interpretive practices through which distinctive modes of reality are constituted (Good 1994), as well as with a cultural phenomenology and anthropology of sensations, as outlined by Hinton and Good in Chapter 3 of this book. An anthropological approach assumes that catastrophic cognitions are grounded in diverse symbolic forms, linked outwardly to social and political domains, and inwardly to bodily and psychological experience. Local bodily logics, networks of semantic or cultural associations, body metaphors, complex memory processes, and other symbolic forms are critical to the processes of appraisal and interpretation that produce responses to sensation that may trigger panic attacks (see Figure 1.2).

Theoretical Perspectives on Panic Disorder

The two chapters in Part I argue for the importance of studying PD from both a historical and a cross-cultural perspective, and they present some analytic tools for doing so: an anthropology of sensations (namely, sensations embedded in complex networks of meanings and within specific technological, political, and bodily practices); a catastrophic cognitions theory of panic; meaning-forming processes; and the link between power and knowledge (that is, an examination of who is empowered by a certain conceptualization of the cause of panic). They contend that the panic experience can be understood only when situated in the context of a particular life that exists in a specific cultural-historical moment, in a distinct environmental location (with a certain architecture, a certain range of sensoryscapes, a certain configuration of dangers), in a set sociological situation, and in a determined economic position.

Historical Perspectives on PD–Like Disorders

The second section of this collection provides contributions to a cultural history of PD. Any claim to studying the history of a disorder formally codified as recently as 1980 obviously falls prey to a presentist bias, raising questions about what phenomena should belong to such a history. Nonetheless, it is clear that broad categories of psychopathology have quite different cultural and civilizational histories. In European and American history, schizophrenia is embedded in ideas of genetic decline and degeneracy, dangerousness, incurability, and asylums, and these are in turn linked to ideas about human difference and evolution rooted jointly in colonial encounters with others and subsequent interpretations of these encounters. Depression has a quite different cultural history, being linked, on the one hand, to classical Hippocratic writings about melancholia and, on the other, to the Christian category *acedia*, one of the cardinal sins that affected early ascetics with a "weariness

or distress of heart" "akin to dejection" (Jackson 1985). Anxiety has yet a different cultural trajectory—embedded in ideas of nerves and the depletion of nervous energy associated with industrialization, modernity, and the "age of anxiety," but also associated with danger and fear, linked to the battlefield and trauma as well as to acute experiences of cardiac distress; to debates about the role of physiological and functional sources of such distress; and to a broad range of irrational fears and phobias. Despite our ability to point to such broad cultural trajectories, isolating a cultural history of panic among these trajectories is particularly challenging.

The book's historical section aims to provide a genealogy[12] of current conceptualizations of PD—to identify phenomena that share a family resemblance to panic attacks; to trace epistemic shifts in understandings of these conditions; to note links between epistemic changes and other shifts in society; to look closely at diverse representations of acute fear and anxiety, particularly in medicine and psychiatry; and to observe debates and emerging ideologies about their causation. These chapters suggest that the acute onset of multiple symptoms, accompanied by fear of death or insanity, gave rise to multiple causal theories and to associated therapeutic practices and "technologies of the self" (Martin et al. 1988), providing PD with a distinctive cultural history.

Cross-Cultural Studies of Panic Disorder

For the Sterling Forest Conference, Byron Good and Arthur Kleinman were asked to provide a cross-cultural review of the literature on anxiety disorders, a review situated among the triumphalist claims of a universal biological psychiatry and neo-Kraepelinian understandings of psychiatric disorders. Their review (Good and Kleinman 1985) staked out a position in conversation with the neo-Kraepelinian views that were dominant at that conference. "Is there evidence that anxiety and disorders of anxiety exist in other cultures?" this review asks, and then poses the following questions. Are the five basic subcategories of anxiety disorders the same across cultures, or do subtypes of anxiety disorders differ by culture or by ethnomedical tradition? Are *DSM* symptom criteria universal, as the biological paradigm suggests, or does culture shape the symptoms associated with particular disorders? What is the relationship between particular local illness categories—for example, *ataques de nervios* among Puerto Ricans, or subtypes of neurasthenia in the Japanese Morita psychotherapy system—and *DSM* categories? What does the cross-cultural research tell us about these issues? Although it focused primarily on the cross-cultural literature, the review was intended to raise similar questions for diverse American ethnic groups, subcultures, and immigrant populations.

The Good and Kleinman review purposely challenged the assumptions of most researchers attending the Sterling Forest Conference as well as the architects of the

DSM system: that symptoms directly reflect physiological abnormalities and that categories of anxiety disorders are natural reflections of disordered human biology. It was also intended to raise questions about the relationship between culture and psychopathology that could be addressed only through research. Although in 1983 there was adequate evidence to raise these questions and to suggest substantial cross-cultural variation, for none of the anxiety disorders or particular cultures was the research strong enough to answer any of these questions in a convincing, empirical manner.

Over the past two decades, a wide-ranging literature based on both empirical research and clinical experience has begun to emerge, providing a basis for responding to many of these questions. The research reported in the chapters in the third section of this book goes considerably beyond the questions formulated by Kleinman and Good in 1983,[13] speaking to the controversies that have emerged in the past two decades. As we shall see, the studies also provide data relevant to new ways of thinking about PD.

To point forward, the ethnographic chapters in this book suggest the following:

- The data provide strong support for the relevance of using PD as a comparative category for research. Panic attacks are recognizable and widely reported in the cultures represented in this book.
- The studies presented here suggest that interdisciplinary research combining clinical, ethnographic, and epidemiological methods that draw on concepts from current psychiatry and critical social sciences is the most fruitful way to proceed.
- The ethnographic chapters in this book follow recent clinical investigations in raising serious doubts about a narrow focus of attention on those panic attacks that appear to be unprovoked or out of the blue. A clear distinction between panic attacks that are provoked and those that are not is often impossible to make. Basing a diagnostic approach on this distinction seems quite dubious. The cross-cultural research supports a far more social and psychological view of panic attacks than that represented by the narrow biological models.
- In many parts of the world and for many persons, a neat separation of panic attacks as found in PD and trauma experiences is not tenable. Several of the chapters suggest a rich and complex relationship between PD-type panic attacks and trauma experiences, which has clear implications for the *DSM* formulation of these as noncomorbid conditions.
- Some of the essays in this volume speak specifically to the question of the phenomenology of panic attacks, including both those symptoms that are most

prominent and the pattern of emergence and crescendo of symptoms within the ten-minute period indicated by the *DSM* system. Clearly the *DSM* definitions do not adequately represent the empirical data obtained from cross-cultural studies.

- The chapters in this book provide extremely rich empirical data on the relationship between local illness categories and PD in settings as diverse as Puerto Rican mainland and island communities, Rwanda, Thailand, Tibetan refugee communities, and China. These chapters indicate that although there is a relationship between local illness categories and *DSM* categories, there is no one-to-one relationship between a particular local illness category and *DSM* categories. *Ataques de nervios* is not a Puerto Rican term for PD; *ataques* are culturally meaningful and phenomenologically consistent, but they are heterogeneous in terms of *DSM* disorders. There is now empirical evidence to indicate precisely the nature of the heterogeneity.

Overall, the ethnographic chapters in this book do not support the conceptualization of PD as a spontaneous, biological eruption with universal phenomenology. Instead, they open onto a more complex modeling of panic experiences. As such, they also suggest a more complex relationship between culture and PD than that represented by the 1983 formulation of Good and Kleinman (1985), which aimed to provide data relevant to the neo-Kraepelinian paradigm. The chapters here suggest that a wide range of cognitive and psychological processes may trigger experiences of panic as well as anxiety about the likelihood and danger of future episodes. They suggest that catastrophic cognitions play a significant role in panic attacks across diverse societies, and that culture is enormously significant in shaping which bodily sensations or internal experiences may be considered threatening or potentially catastrophic by individuals in these societies. They suggest that catastrophic cognitions are one type of "interpretive practice" (Good 1994), and therefore that interpretive cultural studies may be profitably linked to psychological studies in research on PD. The ethnographic studies further suggest that an anthropology of sensations may contribute significantly to understanding essential characteristics of panic attacks and PD.

Conclusion

This book provides a fundamental challenge to naturalizing accounts of PD that suggest that PD is a universal disease entity with relatively little variation across cultures. The essays in this collection show with great specificity that although panic experiences certainly exist widely across time and cultures, they belong to very different frames of social and cultural experience. Symptoms critical to panic

attacks, the phenomenology of the panic experience, and the link to memories of trauma all vary by culture and individual history. These findings have great relevance for the development of culturally appropriate forms of clinical care, as well as for many of the most salient debates within anxiety disorder research. The essays suggest the enormous importance of an anthropology of sensations and detailed understandings of social and psychological experience for the basic science of anxiety and anxiety disorders, as well as for the development of therapeutics and mental health services. They suggest the importance for cultural and psychological anthropology of systematic, cross-cultural studies of panic attacks, PD, and anxiety disorders more broadly.

The chapters of this book represent a significant step forward in understanding panic and PD from a cross-cultural perspective. Simple epidemiological studies based on criteria derived from studies of middle-class American experiences of panic attacks can no longer be accepted as sufficient for making claims about PD being essentially invariant or generally consistent throughout the world (see, for example, Taylor 2000). At the same time, the research represented here is only the beginning of a rich understanding of anxiety, panic, and fear in social and cross-cultural contexts. Orr's *Panic Diaries* (2006) suggests the broad range of social and historical materials that may be relevant to a deeper understanding of PD. Linking such wide-ranging studies to investigations of individual lives in diverse local cultures remains a challenge to be taken up by new generations of scholarship.

Notes

1. In 1895, Freud gave one of the clearest descriptions of what we now call *panic attacks*, labeling them *anxiety attacks* (see McNally 1994). In Freud's view, accumulation of sexual tension, arising from abstinence, coitus interruptus, and the like, erupts into panic.

2. **Here the term *psychophysiology* means the study of the physiological correlates of** emotional states such as fear and joy: *autonomic arousal pattern*, as in palpitations, sweating, and muscle tension; and *endocrinal states*, as in elevation of cortisol or epinephrine levels. It might better be called an *emotion-physiology*.

3. The first author of this introduction, Byron Good, was one of the participants in the conference, where he presented the first draft of the paper "Culture and Anxiety: Cross-Cultural Evidence for the Patterning of Anxiety Disorders," which was published as Chapter 13 of the Tuma and Maser volume (Good and Kleinman 1985). The observations about the 1983 conference are his.

4. In the conference and in the resulting volume (Tuma and Maser 1985), the notion of ***cognitive* was rather limited. In that conference and volume, the so-called cognitive theory of** anxiety examined mainly how fear was represented in the mind, the most prominent model being Lang's (1985) associative network model, in which an emotional state was operationalized as a network consisting of a certain label of the emotion, an associated event, a certain

pattern of physiological arousal, and a certain posture. Cognitive-type treatment was conceptualized as "exposure" to a "fear network." These models focused on memory encoding, retrieval, and "extinction," which were linked to animal models of learning.

5. See Good 1992:187–193 for a more detailed description of the Sterling Forest Conference and its discussions of PD.

6. In fact, this is not the case. Benzodiazapines are effective for PD (Rosenbaum et al. 2005).

7. Imipramine does not have these specific effects, as later research would reveal. It is also effective for depression and chronic anxiety, as in its proven efficacy for generalized anxiety disorder (Rosenbaum et al. 2005).

8. Phobic disorders include *social phobia*, characterized by fear in social situations and anxiety about entering such situations; and *specific phobia*, characterized by fear of certain places or things, such as snakes.

9. The *DSM–III*'s hierarchical conceptualization of diagnosis had an unfortunate consequence: the neglect of PD as an object of inquiry by cross-cultural researchers who had a primary interest in other disorders, such as depression. As an example, in his study of neurasthenia patients, Kleinman (1986:203) found remarkably high rates of PD (in the one hundred patients diagnosed with neurasthenia, 35 percent suffered PD and 87 percent suffered major depressive disorder). Interestingly, in the follow-up study of these same patients who were treated for depression, 33 percent continued to meet criteria for major depressive disorder, 29 percent for PD (Kleinman 1986:219). In line with contemporary diagnostic views, Kleinman (1986: 75) suggested that "anxiety problems seemed secondary to depression," and panic never became a primary focus of his research. The fact that many of the patients in his study had experienced severe trauma associated with the Cultural Revolution in China, a major finding of Kleinman's research, suggests that panic symptoms linked to that trauma may have been quite significant complicating factors in the pathology of the sample he studied.

10. American panic attack sufferers have been shown to focus frequently on one symptom, presenting to the emergency room or to a physician's office with one somatic complaint, such as dizziness or chest pain, not mentioning the other symptoms of autonomic arousal (Katon 1984, 1989). If the physician does not ask about the presence of other autonomic arousal symptoms (such as palpitations, sweating, and shortness of breath), the complaint may be misdiagnosed either as a physical ailment (for example, chest pain as a heart attack, or dizziness as vestibular disorder), thereby resulting in inappropriate and expensive testing, or as a chronic symptom of simple somatization, thereby resulting in ineffective treatment.

11. As an example, Cambodian refugees fear dizziness upon standing; it indicates excessive *khyâl* surging in the body, which may cause "*khyâl* overload" (*khyâl koa*), an extreme and dangerous form of "*khyâl* attack" that may bring about syncope and various physical disasters (see Chapter 3). This "*khyâl* overload" must be treated by culturally indicated methods such as "coining" the body and "biting the ankles," or death may well result. Owing to syndrome-generated catastrophic cognitions, dizziness upon standing causes much greater fear for a Cambodian than for an American. The frequency with which certain sensation-inducers bring about panic varies by culture. Common triggers of panic among Cambodian refugees include standing and feeling dizzy, seeing a spinning object, and smelling car-exhaust fumes

(see Chapter 3); among Vietnamese refugees, triggers include standing and feeling dizzy, a cold wind hitting the body, and urination (see Chapter 3). Both Cambodian and Vietnamese refugees fear that "worry" can damage the body and mind, so if any sensations—particularly dizziness—occur during worry episodes, a spiral of panic may ensue.

12. For Foucault's notion of *genealogy*, see Gutting (1989) and Visker (1995).
13. See Good and Kleinman (1985).

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