CHAPTER 6: Panic, Anxiety, Obsessions, and Their Disorders

Chapter Overview/Summary

Although anxiety disorders were initially considered neuroses, this term has been largely abandoned ever since DSM-III (1980). The anxiety disorders have panic or anxiety or both at their core. Today anxiety impacts 25–29 percent of Americans. Anxiety is defined as an anticipation for possible future danger and fear is in response to immediate danger. Panic is a basic emotion that involves activation of the fight-or-flight response of the autonomic nervous system. Anxiety is more diffuse, including blends of high levels of negative affect, worry about possible threat or danger, and a sense that threats are unpredictable or uncontrollable. Although everyone has identifiable, rational, realistic sources of anxiety, people with anxiety disorders, by definition, have irrational sources of, and unrealistic levels of, anxiety. Mood-congruent information processing, such as attentional and interpretive biases, seem to maintain all anxiety disorders.

Specific phobias are intense and irrational fears of specific objects or situations accompanied by avoidance of the feared object. Stimuli may acquire phobic properties through conditioning or other learning mechanisms or through activation of constitutional predispositions. Because stimuli such as heights and menacing animals that posed a threat to our early ancestors are better able to become the target of phobias, it is thought that we are biologically “prepared” to associate them with trauma. Phobia subtypes include: (1) animals—the fear of snakes, spiders, dogs, insects, and birds; (2) natural environment—fear of storms, heights, and water; (3) blood-injection-injury—fear of seeing blood or an injury, receiving an injection, or seeing a person in a wheelchair; (4) situational—fear of public transportation, tunnels, bridges, elevators, flying, driving, and enclosed spaces; and (5) other—phobias associated with choking, vomiting, or “space phobias.”

Social phobia, also known as social anxiety disorder, involves disabling fears, or even panic attacks, in one or more social situations, usually out of fear of negative evaluation by others or fear of acting in an embarrassing or humiliating manner. Social stimuli signaling dominance and aggression from other humans, including facial expressions of anger or contempt, appear “prepared” in the evolutionary sense to elicit phobic responses. The preoccupation with negative self-evaluative thoughts characteristic of social phobia tends to interfere with the ability to interact in socially skillful ways. Panic disorder involves unexpected panic attacks that often create a sense of stark terror, which usually subsides in a matter of minutes. The fear of future panic attacks is known as “anxious apprehension.” Many people with panic disorder also develop agoraphobic avoidance of situations in which they fear they might have an attack and would find it difficult to escape or would be especially embarrassing. According to the conditioning theory of panic disorder, interoceptive bodily symptoms associated with early stages of prior attacks come themselves to be able to elicit panic attacks. According to the cognitive theory of panic disorder, it is the catastrophic misinterpretation of these bodily cues that produces panic attacks, especially among those with high levels of preexisting anxiety sensitivity. Biological theories of panic disorder emphasize biochemical abnormalities in the brain as well as abnormal activity of the neurotransmitter norepinephrine and probably also serotonin. The area of the brain known as the amygdala is thought to be an especially important source of panic attacks.

Another anxiety disorder is panic disorder, which involves recurrent and unexpected attacks wherein the individual worries about having more attacks. The average panic attack lasts around ten minutes. A panic attack usually includes feelings of heart racing, sweating, shaking, shortness of breath, and so on as just some examples. About 85 percent of people who experience a panic attack think it is a heart attack and may show up at the emergency room. Agoraphobia is the fear of public places such as crowded spaces, shopping malls, and movie theaters. Agoraphobia is a frequent complication of panic disorder, where someone has recurrent panic attacks and presence of agoraphobia. However, because not all patients with agoraphobia experience panic, the DSM-5 now lists agoraphobia as a distinct disorder.

Generalized anxiety disorder (GAD) involves chronic and excessive worry about a number of events or activities and high levels of psychic and muscle tension. People with GAD may have extensive experience with unpredictable and/or uncontrollable life events as well as having schemas through which strange and dangerous situations promote automatic thoughts focused on possible threats. The neurobiological bases of GAD differ from those related to panic disorder, involving the neurotransmitter GABA and the limbic system of the brain.
Obsessive-compulsive disorder (OCD) involves unwanted and intrusive distressing thoughts or images usually accompanied by compulsive behaviors designed to neutralize those thoughts or images. Checking and cleaning rituals are most common. Genetic, brain function imaging, and psychopharmacological studies all suggest significant biological contributions to OCD. The anxiety-reducing qualities of the compulsive rituals may help maintain OCD.

Medical treatments of people with anxiety disorders often include anti-anxiety and anti-depressant medications. These medications suppress anxiety symptoms, have high addiction potential, and tend to be associated with high relapse rates once the medications are discontinued. Behavioral and cognitive therapies are effective for anxiety disorders. Behavior therapies involve prolonged exposure to feared situations to allow fear or anxiety to habituate. With OCD, the rituals also must be prevented following exposure to the feared situations. Cognitive therapies focus on getting clients to understand their underlying automatic thoughts, which often involve cognitive distortions such as unrealistic predictions of catastrophes that in reality are very unlikely to occur, and to change these thoughts and beliefs through cognitive restructuring.

Detailed Outline

I. The Fear and Anxiety Response Patterns

A. Fear
   2. Physiological components in the absence of any external danger.
   4. Anxiety—involves feeling of apprehension about possible future danger.
   5. Fear—a response to immediate danger.
   6. Panic Attack—a response that occurs. When the fear response occurs in the absence of any obvious external danger

B. Anxiety
   1. Adaptive value.
   2. Has cognitive/subjective, physiological, and behavioral components.
   3. Unconditional versus Learned Sources of Fear and Anxiety
      a. Conditionability of fear.
      b. External versus internal (interoceptive) cues.

I. Overview of the Anxiety Disorders and Their Commonalities

A. Unrealistic and Irrational Fears of Disabling Intensity
B. DSM-5 Recognizes Five Anxiety Disorders
   1. Specific phobia
   2. Social phobia or social anxiety disorder.
   3. Panic disorder
   4. Agoraphobia
   5. Generalized anxiety disorder.

C. Anxiety Disorders are Relatively Common
   1. Most common group of disorders among women.
   2. Comorbidity is typical.
   3. Phobias are the most common of the anxiety disorders.
   4. Commonalities in causes across these disorders:
      a. Common genetic vulnerability is the personality trait of neuroticism.
      b. Brain structures most commonly involved are generally in the limbic system.
      c. Most common neurotransmitters involved are GABA, norepinephrine, and serotonin.
      d. Classical conditioning is common.
      e. People with perceptions of lack of control over their environment and their emotions are more vulnerable.
   5. Commonalities across effective treatments:
a. Graduated exposure is the single most effective treatment.
b. Cognitive restructuring.
c. Benzodiazepines and anti-depressants.

II. Specific Phobias (See Table 6.1 for a brief overview)—a person is diagnosed with a specific phobia when she or he shows a persistent fear that is excessive and unreasonable

A. Blood-Injection-Injury Phobia
1. Occurs in about 3%–4% of the population.
2. Disgust is as typical a response as fear.
3. Initial heart acceleration followed by a drop in rate and pressure.

B. Prevalence, Age of Onset, and Gender Differences
2. Animal phobias—about 90%–95% are women.
3. Lifetime prevalence rate is about 12%.
5. Agoraphobia and claustrophobia begin in adolescence and early adulthood.

C. Psychological Causal Factors
1. Psychoanalytic viewpoint:
   a. View of phobia as defense against anxiety via repression of id impulses; anxiety is then displaced onto some external object or the situation is symbolically linked to the real object of the anxiety.
2. Phobias as learned behavior:
   a. Classical conditioning and generalization.
   b. Direct traumatic conditioning.
   c. Vicarious conditioning of phobic fears.
   d. Prepared learning—when primates and humans acquire fears of certain objects or situations that posed real threats.
   e. Sources of individual differences in the learning of phobias:
      (1) History of previous positive experiences reduces the likelihood of a phobia developing.
      (2) Events during conditioning such as inescapable and uncontrollable events.
      (3) Experiences after a conditioning event such as the inflation effect.
      (4) Cognitive factors maintaining phobias.
   f. Evolutionary preparedness for the development of fears and phobias.

D. Biological Causal Factors
1. Affect the speed and strength of conditioning of fear.
2. Behavioral inhibition and fear—high levels in early development correlate with developing multiple specific phobias by 7–8 years of age.
3. Twin studies indicate modest heritability—but nonshared factors play a larger role.

E. Treatments
1. Exposure therapy—involves controlled exposure to the stimuli or situations that elicit phobic fear.
2. Participant modeling.
3. Virtual reality environments.
4. Cognitive and pharmacological treatments are ineffective.
5. Some evidence that anti-anxiety medications may interfere with the positive effects of exposure therapy.

III. Social Phobias
A. Prevalence, Age of Onset, and Gender Differences
1. Approximately 12% of the population qualifies for a social phobia. More than half of these suffer from one or more additional anxiety disorder during their lives.
2. 60% of individuals are female.
3. Starts in early or middle adolescence—early adulthood.
4. The disorder results in lower employment rates and lower SES.
B. Psychological Causal Factors
1. Social phobias as learned behavior
   a. Direct or vicarious conditioning, such as experiencing or witnessing a perceived social defeat or humiliation, or being or witnessing someone else being the target of anger or criticism.
   b. 92% of an adult sample of those with social phobia recalled severe teasing as a child.
   c. Those with social phobia are also more likely to have grown up with parents who were socially isolated and avoidant.
2. Social fears and phobias in an evolutionary context
   a. Proposes that social phobias are a by-product of dominance hierarchies.
   b. Evolutionarily based predisposition.
3. Perceptions of uncontrollability and unpredictability
   a. Lead to submissive and unassertive behavior.
   b. Likelihood increases if person has experienced an actual social defeat.
   c. Diminished sense of personal control that may, in part, have developed from overprotective parents.
4. Cognitive biases
   a. Danger schemas concerning others.
   b. Expect they will behave in an awkward and unacceptable way resulting in rejection.
   c. Preoccupied with bodily responses and negative self-images in social situations.
   d. A negative attribution bias may also come into play here.

C. Biological Causal Factors
1. Genetic and temperamental factors
   a. Modest genetic contribution—about 30% due to genes.
   b. Behavioral inhibition—those high on behavioral inhibition between 2–6 years of age are three times more likely (22%) to be diagnosed with a social phobia even in middle childhood.

D. Treatments
1. Cognitive and behavioral therapies
   a. Exposure.
   b. Challenge of negative, automatic thoughts.
   c. Cognitive restructuring—review the faulty beliefs.
2. Medications
   a. Antidepressants may also be effective.
   b. Monoamine oxidase inhibitors (MAOIs).
   c. Selective Serotonin Reuptake Inhibitors (SSRIs).
   b. One study found that cognitive behavioral therapy was more effective than medication and had better long-term results.

IV. Panic Disorder
A. Panic Disorder—defined as the occurrence of panic attacks
   1. As many as 85% seek help from an emergency room or doctor’s office.
   2. Case of Mindy Markowitz.
B. Agoraphobia
   1. Conceptualized as a marked fear or anxiety in varied situations.
   2. Case of John D.
   3. Most commonly avoided situation is crowded places and streets (see Table 6.2)
   4. Agoraphobia without Panic
      a. Usually a gradually spreading fearfulness.
      b. Extremely rare in clinical settings.
C. Prevalence, Age of Onset, and Gender Differences
   1. Prevalence increasing with younger generations.
   2. Onset most common between 15–24 years.
   3. Twice as common in females, probably for sociocultural reasons (see Table 6.3 for chart of gender differences in anxiety disorders).
D. **Comorbidity with Other Disorders**
1. High comorbidity with other anxiety disorders.
2. 30%–50% will experience serious depression.
3. 83% of people with panic disorder also have at least one comorbid disorder.

E. **The Timing of a First Panic Attack**
1. Frequently follows feelings of distress or a highly stressful life situation.
2. Panic attacks more common (23% of population) than panic disorder.

F. **Biological Causal Factors**
1. Genetic factors
   a. Only moderate heritability.
   b. Liability is probably for panic disorder and phobias.
2. Panic and the brain
   a. **Amygdala**—a collection of nuclei in front of the hippocampus in the limbic system of the brain, which is key in the interpretation of fear.
   b. Abnormally sensitive fear network.
   c. Hippocampus implicated in conditioned anxiety.
   d. Higher cortical centers mediate cognitive symptoms.
3. Biochemical abnormalities
   a. Biological challenge procedures suggest that no single neurobiological mechanism is implicated.
   b. Noradrenergic and serotonergic systems are implicated.
   c. GABA recently shown to be implicated in anticipatory anxiety.
   d. **Panic provocation procedures**—something that produces panic attacks in panic disorder.

G. **Psychological Causal Factors**
1. Comprehensive learning theory of panic disorder
   a. “Fear of fear” hypothesis and process of *interoceptive* and *exteroceptive* conditioning.
   b. Anxiety conditioned to internal and external cues.
   c. Panic attacks themselves are likely conditioned to certain internal cues.
   d. Constitutional and experiential vulnerabilities.
2. The cognitive theory of panic (see Figure 6.2)
   a. Catastrophic interpretations of bodily sensations.
   b. Automatic thoughts become the triggers of panic.
   c. Evidence that cognitive therapy for panic works supports the prediction that changing cognitions about bodily symptoms may reduce or prevent panic.
3. Learning and cognitive explanations of results from panic provocation studies
   a. Catastrophic cognitions are not needed in conditioning theory.
   b. Cues can be unconscious.
   c. Learning theory is better than cognitive model at explaining nocturnal panic attacks and panic attacks that occur without any preceding negative (catastrophic) automatic thoughts.
4. Anxiety sensitivity and perceived control
   a. **Anxiety sensitivity** is a trait—like belief that certain bodily sensations may have harmful consequences.
   b. Anxiety sensitivity predicted the development of spontaneous panic attacks during a highly stressful period.
   c. Psychological manipulations, such as having a sense of perceived control or having a “safe” person, may block panic.
5. Safety behaviors and the persistence of panic
   a. Disconfirmation does not occur because people with panic disorder engage in “safety behaviors” such as breathing slowly.
   b. Safety behaviors believed to prevent catastrophe.
   c. Safety behaviors need to stop for effective treatment.
6. Cognitive biases and the maintenance of panic
   a. People with panic disorder interpret ambiguous bodily sensations and situations
as threatening.

b. Attentional bias toward threat cues.
c. Memory bias favoring threatening information.

H. Treatments
1. Medications
   a. Benzodiazepines/anxiolytics, e.g., xanax or klonopin
      (1) Rapid effects.
      (2) Addictive.
      (3) Withdrawal must be gradual.
      (4) Rebound panic and relapse.
      (5) Interfere with cognitive therapy.
   b. Antidepressants (primarily the tricyclics and the SSRIs)
      (1) Non-addictive.
      (2) Slow effects—may take up to 4 weeks.
      (3) Side-effect problems—SSRIs better tolerated.
      (4) High relapse rates.
2. Behavioral and cognitive-behavioral treatments
   a. Prolonged exposure is effective in 60%–75% of patients.
   b. Interoceptive exposure.
   c. Integrative cognitive-behavioral techniques.
   d. Combined medication and cognitive-behavior therapy seems to always lead to greater relapse.

V. Generalized Anxiety Disorder
A. General Characteristics
   1. Future-oriented mood state of chronic worry and “anxious apprehension.”
   2. Restless, easily fatigued, poor concentration, irritable, tense, indecisive.
   3. Worry experienced as uncontrollable.
   4. The “basic” anxiety disorder.
   5. Subtle avoidance such as procrastination and checking.
   6. High vigilance, muscle tension, and sleep disturbance.
   7. A graduate student with GAD.
B. Prevalence, Age of Onset, and Gender Differences
   1. Relatively common.
   2. Twice as common in women.
   3. Most continue to function despite symptoms.
   4. Age of onset difficult to determine with as many as 60%–80% report being anxious all their lives.
C. Comorbidity with Other Disorders
   1. Seen with other Axis I disorders, especially other anxiety and mood disorders.
   2. Excessive use of tranquilizing drugs, sleeping pills, and alcohol complicates the clinical picture.
D. Psychological Causal Factors
   1. The psychoanalytic viewpoint
      a. Unconscious conflict between id and ego.
      b. Defenses broken down or never developed.
      c. No object to displace upon.
      d. Theory is not testable and has basically been abandoned.
   2. Perceptions of uncontrollability and unpredictability
      a. Cognitive processes associated with prior aversive events.
      b. Unpredictability of important past events generalizes to future ones.
      c. Lack of safety signals.
   3. A sense of mastery: The possibility of immunizing against anxiety
      a. “Master” and “yoked” infant monkeys, rhesus monkeys.
      b. “Masters” coped better with stress when older.
      c. Suggests that early experiences with control and mastery can immunize the
individual against the harmful effects of stressful situations.

4. The central role of worry and its positive functions
a. Five benefits of worry identified by people with GAD: superstitious avoidance of catastrophe, actual avoidance of catastrophe, avoidance of deeper emotional topics, coping and preparation, motivating device.
b. Suppression of emotional and aversive physiological responding may serve to reinforce the process of worry.
c. Worry impairs the processing of the event, thereby preventing fear from being extinguished.

5. The negative consequences of worry
a. Worrying is itself not pleasant.
b. Attempts to control thoughts and images actually increase them.

6. Cognitive biases for threatening information
a. Attention is drawn toward threat cues.
b. Interpret ambiguous stimuli as threats.

E. Biological Causal Factors
1. Genetic factors
a. Small to modest heritability.
b. Inherited predisposition is to neuroticism (proneness to experience negative mood states); shared with major depression.

2. Neurotransmitters and neurohormonal abnormalities
a. A functional deficiency of GABA.
b. The corticotrophin-releasing hormone system and anxiety.

3. Neurobiological differences between anxiety and panic
a. Biology of panic and GAD are not the same.
b. Amygdala and fight-or-flight for fear and panic, limbic system for GAD.

F. Treatments
1. Medications
a. Benzodiazepines not as effective as believed by public.
b. Buspirone is a new, non-addictive, non-sedating, but slow drug.
c. Antidepressants are useful.

2. Cognitive behavioral treatment
a. Therapy involves applied muscle relaxation and cognitive restructuring, is quite effective.

VI. Obsessive-Compulsive and Related Disorders
A. Obsessive-Compulsive Disorder
1. Types of obsessive thoughts
a. Obsessions—Contamination fears, harming self or others, lack of symmetry, pathological doubt, sexual obsessions, and obsessions concerning religion or aggression.

b. Obsessions rarely carried out.

2. Types of compulsions
a. Compulsions—Five primary types: cleaning, checking, repeating, ordering/arranging, and counting.
b. Performance of act brings feeling of reduced tension and satisfaction, as well as a sense of control.

3. Consistent characteristics
a. Anxiety is the affective symptom.
b. Fear that something terrible will happen to them or to others because of them.
c. Compulsion reduces anxiety in the short term.
d. “What if” illness; this tendency to judge risks unrealistically is very common among those with OCD.
B. Prevalence, Age of Onset, and Gender Differences
1. Not as rare as once thought—2.3% lifetime prevalence.
2. More than 90% of those who present for treatment experience both obsessions and compulsions; if include mental rituals and compulsions, this jumps to 98%.
3. Divorced and unemployed people overrepresented.
4. Little or no gender difference.
5. Typically begins in late adolescence or adulthood but is not uncommon in children.
6. Early onset more common in boys and is usually associated with more severe symptoms.
7. Gradual onset and chronic once serious.

C. Comorbidity with Other Disorders
1. Depression is especially common—up to 80% may experience significant depressive symptoms.
2. Body dysmorphic disorder also rather common as a comorbid disorder.

D. Psychological Causal Factors
1. OCD as learned behavior
   a. Mowrer’s two-process theory of avoidance learning.
   b. Several classic experiments have supported this theory.
   c. Core of the most effective form of behavior therapy for OCD.
   d. Does not explain development of obsessions or abnormal assessments of risk.
2. OCD and preparedness
   a. Some fears have occurrence rates that seem nonrandom.
   b. Obsessions also adaptive in evolutionary terms.
3. Cognitive causal factors
   a. The effects of attempting to suppress obsessive thoughts
      (1) Thought suppression may lead to paradoxical increase in those thoughts later.
      (2) Normal and abnormal obsessions differ in degree to which they are resisted.
   b. Appraisals of responsibility for intrusive thoughts
      (1) Inflated sense of responsibility may lead to thought-action fusion.
   c. Cognitive biases and distortions
      (1) Problems inhibiting cognitive processing.
      (2) Predisposition to thought suppression.
      (3) Nonverbal, but not verbal, memory deficits.

E. Biological Causal Factors
1. Genetic influences
   a. Moderately high heritability.
   b. Higher rates if sub-clinical obsessive-compulsive symptoms and tic-related OCD is included.
2. OCD and the brain
   a. Abnormally active metabolic levels in the orbital frontal cortex, caudate nucleus, and cingulate cortex.
   b. Brain functions normalize after behavior or pharmacotherapy.
   c. Dysfunction of the cortico-basal-ganglionic-thalamic circuit leading to inappropriate behavioral responses that are normally inhibited.
   d. Orbital frontal cortex is responsible in the obsessions.
3. Neurotransmitter abnormalities
   a. Anafranil (clomipramine) and prozac often effective.
   b. Drugs must be taken at least 6–12 weeks before changes noted.
   c. Leads to a functional decrease in availability of serotonin.

F. Treatments
1. Behavioral and cognitive-behavioral treatments
   a. Behavioral treatment that combines exposure and response prevention is most effective.
   b. Success in 50%–70% of patients; this is superior to medication.
2. Exposure and response prevention—the treatment involves having OCD clients
develop a hierarchy of upsetting stimuli
3. Medications
   a. Serotonin-reuptake inhibitors.
   b. Relapse rates high (up to 90%) following medication discontinuance.
   c. Combining medication with behavioral treatment has not been shown to be more effective in adults; one study showed promise in children.
   d. Neurosurgery being investigated once again.
   e. Antipsychotic medications.

G. Body Dysmorphic Disorder (BDD)
1. Moved from classification as somatoform disorder in *DSM-IV-TR* to OCD and related disorders category in *DSM-5*
2. Obsessed with perceived or imagined flaw
3. Interference with social functioning, avoidance of social interactions, depression
4. Prevalence is 1%–2% of general population, 8% of people with depression, equal in men and women
5. Causal factors: A biopsychosocial approach
6. Treatment of BDD-closely related to OCD treatments, SSRIs

H. Hoarding Disorder
1. Prevalence: 10%–40% with OCD, 3%–5% of adult population
2. More disabled than people with OCD without compulsive hoarding and are at greater risk for fire and health risks.
3. Recent studies indicate that the brain scans of hoarders is different from those of persons with OCD that do not hoard.
4. Many persons with hoarding disorder do not respond to medications that work on those with OCD.

I. Trichotillomania
1. Compulsive hair pulling resulting in noticeable hair loss
2. Onset can be in childhood or later
3. Research is in early stages

VII. Cultural Perspectives
A. Cultural Differences in Sources of Worry
1. Yoruba culture in Nigeria indicates three clusters of symptoms: worry, dreams, bodily complaints.
2. Culturally related syndrome in China is called Koro.
3. Caribbean cultures and ataque de nervios.
B. Taijin Kyofusho
1. Anxiety disorder symptoms unique to Japanese cultural patterns.
2. Fear of blushing, making eye contact, emitting an offensive odor.

IX. Unresolved Issues
A. The choice of treatments: Medications or Cognitive Behavior Therapy?
1. People with anxiety or obsessive-compulsive disorders are unaware of treatment options
2. Limited specialized training for treating clinicians
3. Advantages and disadvantages of medication
4. Long-terms and cost-effectiveness of cognitive behavior treatment
Key Terms

agoraphobia
amygdala
anxiety
anxiety disorders
anxiety sensitivity
blood-injection-injury phobia
body dysmorphic disorder (BDD)
cognitive restructuring
compulsions
exposure and response prevention
exposure therapy
exteroceptive conditioning
fear
generalized anxiety disorder (GAD)

hoarding disorder
interoceptive conditioning
neurotic disorders
obsessions
obsessive-compulsive disorder (OCD)
obsessive-compulsive spectrum disorders
panic attack
panic disorder
panic provocation procedures
phobia
prepared learning
social phobia
specific phobia
trichotillomania