II Adult Mental Health

5 Anxiety-Based Problems

ROUTE MAP OF THE CHAPTER

This chapter describes some of the main anxiety-based problems. It discusses contemporary accounts of their causes (aetiology) and describes a range of relevant and effective treatments for each. It is divided into six main sections covering specific phobias, social phobia, panic disorder, generalized anxiety disorder, obsessive-compulsive disorder and post-traumatic stress disorder. These topics are chosen because they represent some of the most prevalent anxiety-based problems (e.g. panic disorder, generalized anxiety disorder) as well as some of the most thoroughly researched, and our understanding of their causes has become relatively well developed.

I am a baritone soloist and have performed serious music, in public, for some thirty years. The first time was for my entire high school class at graduation where I did the Coronation scene from Boris Godounov.

For the first fifteen years, it was not always a pleasant experience. I got nauseous and shook like a leaf before, during and after nearly every performance. At one point, during a Messiah performance in the middle of 'But Who May Abide', I asked myself why I was doing this. Even though scared half to death, my heart in my throat, blood pounding in my ears, I continued, almost compulsively. . . . It was always worst when I tried to achieve perfection. This was usually in front of other students at weekly recitals in graduate school, when I knew they were taking exquisite note of every mistake.

TOBIAS'S STORY

Introduction

This example of performance anxiety displays a number of features that are characteristic of the experience of anxiety, whether the everyday experiences of anxiety that we all encounter or the more debilitating and chronic experience of anxiety that is suffered by those diagnosed with clinical problems. Firstly, there are the physical symptoms of anxiety, such as muscle tension, dry mouth, perspiring, trembling and difficulty swallowing. In its more chronic form, anxiety may also be accompanied by dizziness, chronic fatigue, sleeping difficulties, rapid or irregular heartbeat, diarrhoea or a persistent need to urinate, sexual problems and nightmares. Secondly, *Tobias's Story* gives a good insight into the cognitive characteristics of anxiety. These include a feeling of apprehension or fear, usually resulting from the anticipation of a threatening event or situation. Usually accompanying anxiety are intrusive thoughts about the threat, catastrophic bouts of worrying about the possible negative outcomes associated with the threat and, in some specific types of problems, uncontrollable flashbacks about past traumas and anxiety-provoking experiences. Overly anxious people also find it hard to stop thinking negative and threatening thoughts, and this is in part due to the cognitive biases that have developed with the experience of anxiety. Interestingly, *Tobias's Story* also highlights the role that some personality (or dispositional factors) play in the development of anxiety and may act as vulnerability factors – in this case, the desire to achieve perfection.

We all experience feelings of anxiety quite naturally in many situations – such as just before an important exam, while making a presentation at college or work,

at an interview or on a first date. Most anxiety reactions are perfectly natural, and they have evolved as adaptive responses that are essential for us to perform effectively in challenging circumstances. However, anxiety can often become so intense or attached to inappropriate events or situations that it becomes maladaptive and problematic for the individual (Lepine, 2002). This is when an anxiety disorder may develop. An ***anxiety disorder*** is an excessive or aroused state characterized by feelings of apprehension, uncertainty and fear. In a sufferer of an anxiety disorder the anxiety response:

Anxiety disorder An excessive or aroused state characterized by feelings of apprehension, uncertainty and fear.

1. may be out of proportion to the threat posed by the situation or event (e.g. in specific phobias);
2. may be a state that individuals constantly find themselves in and may not be easily attributable to any specific threat (e.g. in generalized anxiety disorder or some forms of panic disorder);
3. may persist chronically and be so disabling that it causes constant emotional distress to the individual, who is unable to plan and conduct normal day-to-day living. This can result in an inability to hold down a regular job or maintain long-term relationships with friends, partners and family.

Anxiety-based problems are relatively common, and around 30–40 per cent of individuals in Western societies will develop a problem that is anxiety related at some point in their lives (Shepherd, Cooper, Brown & Kalton, 1996). As a result, pathological anxiety imposes a high individual and social burden, tends to be more chronic than many other psychological problems, and can be as disabling as physical illness. In both Europe and the USA, the cost of treating anxiety-based problems runs into many billions of pounds annually, making them more economically expensive than any other psychological problem (Rovner, 1993). These economic costs include psychiatric, psychological and emergency care, hospitalization, prescription drugs, reduced productivity, absenteeism from work and suicide (Lepine, 2002).

In this chapter we will discuss in detail six of the main anxiety disorders. The details of these disorders are summarized at the outset in Table 5.1, and readers may want to refer back to this table when they have read and digested the information on each separate disorder. The six disorders are:

- specific phobias
- social phobia
- panic disorder
- generalized anxiety disorder (GAD)
- obsessive-compulsive disorder (OCD)
- post-traumatic stress disorder (PTSD)

Anxiety disorders are diagnosed when subjectively experienced anxiety is present and recurs on such a regular and chronic basis that it disrupts normal daily living. Many of the symptoms of anxiety are common to a number of different anxiety disorders, and this can lead to ***comorbidity*** (see Chapter 1, section 1.3.2.3). Comorbidity is not unusual in the diagnosis of anxiety disorders (Rodriguez, Weisberg, Pagano, Machan et al., 2004), and may occur because a number of basic psychological processes or similar developmental experiences may be common to different diagnostic categories. Some common aspects are:

Comorbidity The co-occurrence of two or more distinct disorders.

1. Physiological symptoms of panic are found not only in panic disorder, but also in the reactions to phobic stimuli in specific phobias.

Table 5.1 *Anxiety disorders: summary*

DISORDER AND LIFETIME PREVALENCE RATES	DEFINITION	MAIN DSM-IV-TR DIAGNOSTIC FEATURES	KEY FEATURES	THEORIES OF AETIOLOGY	MAIN FORMS OF TREATMENT
SPECIFIC PHOBIA ***(7.2%–11.3%)***	Excessive, unreasonable, persistent fear triggered by a specific object or situation	Marked and specific fear trigger by a specific object or situation Exposure evokes immediate anxiety Individual recognizes the fear is excessive The phobic situation is always avoided The fear interferes significantly with daily functioning	Clinical phobias are usually restricted to a small group of objects and situations (e.g. animals, heights, water, blood and injury) Twice as many females as males develop specific phobias Phobics acquire a set of threat-relevant beliefs that maintain their phobia	Psychoanalytic accounts Classical conditioning Biological preparedness Non-associative fear acquisition Disease-avoidance model	Exposure therapy Systematic desensitization Flooding One-session rapid treatments

Table 5.1 *(Cont'd)*

DISORDER AND LIFETIME PREVALENCE RATES	DEFINITION	MAIN DSM-IV-TR DIAGNOSTIC FEATURES	KEY FEATURES	THEORIES OF AETIOLOGY	MAIN FORMS OF TREATMENT
SOCIAL PHOBIA ***(7%–13%)***	A severe and persistent fear of social or performance situations	Persistent fear of social or performance situations Exposure to social situations provokes anxiety Individual recognizes that fear is excessive Avoidance and anxiety significantly interferes with daily functioning	Anxiety of socially based situations is so pervasive it has been labelled 'social anxiety disorder' Social phobia is sometimes associated with panic attacks Social phobics appear to have developed an information processing and interpretation bias which causes them to make excessively negative predictions about future social events	Genetic factors Role of behavioural inhibition in childhood Information processing biases (e.g. negative processing of ambiguous information) Self-focused attention	CBT Medication (e.g. MAOIs and SSRIs)
PANIC DISORDER ***(1.5%–3.5%)***	The experience of repeated and uncontrollable panic attacks	Recurrent, persistent panic attacks At least 1 month of persistent concern or worry about these attacks Panic attacks cannot be accounted for by the physical effects of a substance or a general medical condition	Onset is common in adolescence or early adulthood, and normally following a period of stress Frequency of panic attacks can vary between one attack per week to frequent daily attacks Associated with fear of serious underlying medical condition or that the individual is losing control or 'going crazy'	Hyperventilation model Suffocation alarm theories Noradrenergic overactivity Classical conditioning Anxiety sensitivity Catastrophic misinterpretation of bodily sensations	Tricyclic antidepressants and benzodiazepines Exposure-based treatments CBT
GENERALIZED ANXIETY DISORDER (GAD) ***(5%)***	The experience of continual apprehension and anxiety about future events, leading to chronic and pathological worry	Excessive anxiety and worry occurring more days than not for 6 months Worry is uncontrollable Associated with 3 or more physical symptoms The anxiety or worry causes significant distress or impairment of daily functioning	Pathological worry is the cardinal diagnostic feature of GAD GAD is twice as common in women as in men 12% of those who attend anxiety clinics will present with GAD Highly comorbid with a range of other anxiety disorders and major depression	Genetic factors Information processing biases Dysfunctional beliefs about worrying The role of dispositional factors	Anxiolytics such as benzodiazepines Stimulus control treatment CBT (including self-monitoring, relaxation training and cognitive restructuring)

Table 5.1 *(Cont'd)*

DISORDER AND LIFETIME PREVALENCE RATES	DEFINITION	MAIN DSM-IV-TR DIAGNOSTIC FEATURES	KEY FEATURES	THEORIES OF AETIOLOGY	MAIN FORMS OF TREATMENT
OBSESSIVE-COMPULSIVE DISORDER (OCD) ***(2.5%)***	Recurrent obsessions or compulsions that are severe enough to be time-consuming or cause distress	Recurrent thoughts, impulses, images experienced as intrusive and inappropriate Repetitive behaviours or mental acts that the person feels driven to perform The person recognizes that these obsessions or compulsions are excessive or unreasonable The obsessions or compulsions cause marked distress	OCD onset is gradual and begins to manifest in early adolescence or adulthood – normally following a stressful life event Affects women more frequently than men The main compulsions are checking and washing behaviours – although these rarely occur together in the same individual Sometimes comorbid with other disorders such as major depression and eating disorders	Role of brain deficits in the frontal lobes and basal ganglia Memory deficits Inflated responsibility Thought–action fusion Perseveration and the role of negative mood	Exposure and ritual prevention treatments (EPR) CBT Drug treatment (SSRIs) Cingulatomy
POST-TRAUMATIC STRESS DISORDER (PTSD) ***(3%–8%)***	A set of persistent, anxiety-related symptoms that occur after experiencing or witnessing an extremely traumatic event	Experience of events involving death or threatened death Response involves intense fear, helplessness or horror The traumatic event is persistently re-experienced The individual persistently avoids stimuli associated with the trauma Physical symptoms indicating increased arousal Duration of the disturbance is more than 1 month The disturbance causes significant distress or impairment	Following a severe traumatic event, women are significantly more likely to develop PTSD than men Experiences that are likely to cause PTSD include physical assault and rape, torture, POW and combat experiences, natural disasters such as floods and earthquakes, and motor vehicle accidents Main symptoms include increased arousal, avoidance and numbing of emotions, and re-experiencing of the traumatic event	Theory of shattered assumptions Classical conditioning Emotional processing theory Mental defeat Dual representation theory	Psychological debriefing Exposure therapy Eye-movement desensitization and reprocessing (EMDR) Cognitive restructuring

2 Cognitive biases – such as information processing biases that tend anxious people to selectively attend to threatening stimuli (Mathews & McLeod, 1994) – are common to almost all anxiety disorders.

3 A number of prominent psychopathologies are characterized by the dysfunctional and uncontrollable perseveration of certain thoughts, behaviours or activities (e.g. pathological worrying in generalized anxiety disorder, perseverative compulsions in obsessive-compulsive disorder and rumination during periods of depression), and the psychological mechanism that underlies dysfunctional perseveration may be similar across all these disorders (Davey, 2006b).

4 Certain specific early experiences can be found in the aetiology of a number of different anxiety disorders (e.g. physical or sexual abuse during childhood), and experiences such as these may increase an individual's risk of developing several anxiety-based problems.

Let's look at each of the anxiety diagnostic categories in turn, starting with a closer look at specific phobias.

5.1 SPECIFIC PHOBIAS

Specific phobia An excessive, unreasonable, persistent fear triggered by a specific object or situation.

Specific phobias (SP) are defined as an excessive, unreasonable, persistent fear triggered by a specific object or situation. The DSM-IV-TR criteria for Specific Phobia are presented in Table 5.2. The phobic trigger usually elicits extreme fear and often panic, which usually means that the phobic individual develops avoidance strategies designed to minimize the possibility of contact with that phobic trigger. Phobics are normally aware that their fear of the phobic situation or event is excessive or unreasonable (in comparison either with the actual threat it represents or with the less fearful responses of other people), but they do acquire a strong set of ***phobic beliefs*** that appear to control their fear (Thorpe & Salkovskis, 1997). These beliefs normally contain information about why they think the phobia is threatening and how to react when they are in the phobic situation (e.g. avoid contact). Many contemporary psychological treatments for specific phobias are designed to challenge these dysfunctional phobic beliefs and replace them with more functional beliefs that foster approach and contact with the phobic stimulus.

Phobic beliefs Beliefs about phobic stimuli that maintain the phobic's fear and avoidance of that stimulus or situation.

Table 5.2 *DSM-IV-TR criteria for specific phobia*

A	Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation (e.g. flying, heights, animals).
B	Exposure to the phobic stimulus almost inevitably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed panic attack.
C	The person recognizes that the fear is excessive or unreasonable.
D	The phobic situation(s) is avoided or else is endured with intense anxiety or distress.
E	The avoidance, anxious anticipation or distress in the feared situation(s) interferes significantly with the person's normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.
F	In individuals under 18 years, the duration is at least 6 months.
G	The anxiety, panic attacks or phobic avoidance associated with the specific object or situation are not better accounted for by another mental disorder, such as OCD (e.g. fear of dirt in someone with obsessions about contamination), PTSD (e.g. avoidance of stimuli associated with a severe stressor), separation anxiety disorder (e.g. avoidance of school), social phobia (e.g. avoidance of social situations because of fear of embarrassment), panic disorder with agoraphobia, or agoraphobia without history of panic disorder.

5.1.1 Prevalence

Specific phobias are extraordinarily common, with surveys suggesting that a clear majority of the general population (60.2 per cent) experience 'unreasonable fears' (Chapman, 1997) – although in most cases these fears are rarely severe enough to result in impairment or distress. Around 10 per cent of people will meet DSM-IV-TR criteria for a specific phobia within their lifetime, which suggests that severe and disruptive phobic symptoms can be quite common. Table 5.4 shows the prevalence rates for some of the more common forms of specific phobia. There is also a clear gender difference in the prevalence of specific phobias, with a lifetime prevalence rate of around 7 per cent for men and 16 per cent for women (Kessler, McGonagle, Zhao, Nelson et al., 1994).

5.1.2 Common Phobias

Interestingly, common phobias tend to focus on a relatively small group of objects and situations, the main ones being animal phobias (including fear of snakes, spiders, rats, mice, creepy-crawlies such as cockroaches, invertebrates such as maggots and slugs), social phobia, dental phobia, water phobia, height phobia, claustrophobia and a cluster of blood, injury and inoculation fears (known as BII). Most other types of phobias are less common and can be thought of as quite unusual given the degree of threat they might realistically pose – such phobias include fear of cotton wool, buttons, chocolate, dolls and vegetables (McNally, 1997)! DSM-IV-TR subdivides specific phobias according to the source of the fear into four groups: (1) blood, injuries and injections (BII); (2) situational fears (e.g. aeroplanes, lifts, enclosed spaces); (3) animals; and (4) the natural

Table 5.3 *The phobic beliefs of spider phobics*

Phobics develop a set of dysfunctional beliefs about their phobic stimulus or event. These beliefs are very rarely challenged because the phobic avoids all circumstances where such beliefs might be disconfirmed. These beliefs maintain phobic fear and serve to motivate responses designed to avoid contact with the phobic stimulus. Below are some examples of phobic beliefs held by spider phobics. Such beliefs are the kinds that are challenged in both exposure therapy and cognitive therapy procedures.

Harm Beliefs

When a spider is in my vicinity I believe that the spider will:

(a) bite me
(b) crawl towards my private parts
(c) do things on purpose to tease me
(d) get on to parts of me that I cannot reach

Chaser and Prey Beliefs

When I encounter a spider it will:

(a) run towards me
(b) stare at me
(c) settle on my face
(d) not be shaken off once it is on me

Unpredictability and Speed Beliefs

When I encounter a spider:

(a) its behaviour will be very unpredictable
(b) it will be very quick
(c) it will run in an elusive way

Invasiveness Beliefs

When I encounter a spider it will:

(a) crawl into my clothes
(b) walk over me during the night
(c) will hide in places I do not want, such as my bed

Response Beliefs

When I encounter a spider I will:

(a) feel faint
(b) lose control of myself
(c) go hysterical
(d) scream

Source: adapted from Arntz, Lavy, van den Berg & van Rijsoort, 1993; Thorpe & Salkovskis, 1995.

Table 5.4 *Lifetime prevalence rates for common specific phobias*

SPECIFIC PHOBIA	*LIFETIME PREVALENCE RATES*
Social phobia	3.2%[a]
Blood-injury-injection phobia	3.5%[c]
Animal phobias generally	1.1%[a]
Dental phobia	3–5%[b]
Water phobia	3.3%[a]
Height phobia	4.7%[a]
Claustrophobia/enclosed spaces	2.4%[a]

[a] Taken from the Epidemiologic Catchment Area (ECA) study (see Chapman, 1997).
[b] Kent (1997).
[c] Bienvenu & Eaton (1998).

environment (e.g. heights, water). There is some evidence that if you suffer from a specific phobia in one of these categories, you are more likely to suffer a phobia of one or more of the others in that category (e.g. Davey, 1992b; Fredrikson, Annas, Fischer & Wik, 1996), and thus phobias within each category can have a higher incidence of comorbidity (Kendler, Myers, Prescott & Neale, 2001).

There are also important cultural differences in the kinds of stimuli and events that can become the focus of clinical phobias. For example, Taijin-kyofu-sho (TKS) is a common Japanese syndrome characterized by a fear of embarrassing or offending other people (Prince & Tchenglaroche, 1987). This is rather different from the Western syndrome of social phobia, where the fear is based on the public embarrassment experienced by the phobic individual himself or herself. Davey, McDonald, Hirisave, Prabhu et al. (1998) also found a number of important cross-cultural differences in animal fears. For example, while fear of spiders is a common phobic reaction in most Western cultures, spiders were significantly less feared in the Indian sample used in the study. This kind of cross-cultural variability suggests that 'fear-relevance' may at least in part be determined and developed by factors that are specific to individual cultures, and this should be contrasted with more biologically oriented views which argue that fear-responses have been universally pre-wired by evolutionary selection pressures (Davey, 1995; see section 5.1.3.3 below on evolutionary accounts of phobias).

5.1.3 The Aetiology of Specific Phobias

Attempts to explain specific phobias have a long history, dating back to the early days of the psychoanalytic approaches pioneered by Freud and the conditioning views developed by the behaviourist

Plate 5.1
A majority of people claim to have a phobia of some kind, although most are not severe enough to cause distress or to disrupt normal daily life. Some phobias are unusual, such as phobia of cotton wool or buttons – but they are much more common than you think.

J.B. Watson. Originally, there was a tendency to try to explain all types of phobias with just one explanatory theory (e.g. classical conditioning), but this approach has now given way to the view that different types of phobias might be acquired in quite different ways (a multifaceted approach). Over the years, an intriguing debate has taken place about whether phobias are biologically determined through evolutionary processes or whether they are responses learned during the lifetime of the individual. This debate will be an important feature of what follows.

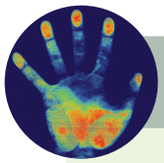
5.1.3.1 Psychoanalytic Accounts

Phobias have intrigued psychologists for more than a century. This may be because they manifest as irrational fears of things that usually pose little if any realistic threat, and their acquisition more often than not cannot be explained by recourse to simple learning experiences such as a specific traumatic event. This has led at least some approaches to psychopathology to view phobias as symbolic of other, more deep-rooted psychological difficulties. For example, psychoanalytic theory as developed by Freud saw phobias as a defence against the anxiety produced by repressed id impulses, and this fear became associated with external events or situations that had a symbolic relevance to that repressed id impulse. Focus Point 5.1 describes the classic case of Little Hans, a 5-year-old boy who developed a severe phobia of horses. Within Freud's psychoanalytic theory, the function of phobias was to avoid confrontation with the real, underlying issues (in this case, a repressed childhood conflict). However, because of the nature of psychoanalytical theorizing, there is little in the way of objective evidence to support such accounts of phobias. Nevertheless, there is often an element of insight that can be drawn from the symbolic interpretations of case histories provided by psychoanalysis, and many anxiety disorders may indeed function for the sufferer as a way of avoiding confrontation with more challenging life issues and difficulties.

5.1.3.2 Classical Conditioning and Phobias

Attempts to explain phobias in terms of classical conditioning (see Chapter 1, section 1.1.3.2) date back to the famous 'Little Albert' study reported by Watson and Rayner in 1920. Albert was an 11-month-old infant, and Watson and Rayner attempted to condition in him a fear of his pet white rat. They did this by pairing the rat – the *conditioned stimulus* (CS) – with the frightening event of a loud noise produced by striking an iron bar – the *unconditioned stimulus* (UCS), which distressed Albert (the *unconditioned response*, UCR). After several pairings of the rat with the noise, Albert began to cry (the *conditioned response*, CR) whenever the rat was introduced into the room. This type of explanation has been popular over the past 50 years, and more sophisticated contemporary conditioning models of specific phobias have been developed (Davey, 1992a, 1997). However, it is difficult to generally explain the range of features possessed by specific phobias with conditioning theories. These criticisms include:

1. Traumatic experiences are essential for conditioning accounts, yet many phobics appear unable to recall any trauma or aversive conditioning experience at the time of the onset of their phobia (Rachman, 1977; Marks, 1969; Emmelkamp, 1982). This appears to be particularly true of some animal phobics such as snake and spider phobics (Davey, 1992b; Murray & Foote, 1979), and also height and water phobics (Menzies & Clarke, 1993a,b).
2. Not all people who have pain or trauma paired with a situation develop a phobia. For example, not everyone who has a traumatic experience undergoing dental treatment acquires a dental phobia (Lautch, 1971), not everyone who experiences a violent thunderstorm acquires a thunderstorm phobia (Liddell & Lyons, 1978) and not all fliers who experience a traumatic flying accident express a subsequent anxiety of flying (Aitken, Lister & Main, 1981; Goorney, 1970). This suggests that a potential conditioning experience is itself insufficient to cause a phobia.
3. Simple conditioning models treat all stimuli as equally likely to enter into association with aversive consequences, yet fears and phobias are not evenly distributed across stimuli and experiences. People appear to develop phobias of animals (snakes, spiders), heights, water, death, thunder and fire more readily than fears of, for example, hammers, electric outlets, knives or guns, even though the latter



FOCUS POINT 5.1

Little Hans: The psychoanalytic interpretation of a specific phobia

One of the most famous cases in the history of psychoanalysis is that of 'Little Hans', a 5-year-old boy who revealed many of his perceptions, fantasies and fears to his physician father, who, in turn, reported them to Sigmund Freud. Hans began to have a fear of horses, which eventually grew to the point that he refused to leave the house. The immediate event that precipitated this phobia was seeing a big, heavy horse fall down. Freud interpreted this to mean that Hans at that moment perceived his own wish that his father would fall down. Then Hans, a little Oedipus, could take his father's place with his beautiful mother. Another part of the fear derived from the large size of horses, which Hans unconsciously identified with the great power of his father. He expressed the fear that a horse would come into his room. He also became afraid not only of horses biting him, but also of carts, furniture vans and buses. This revealed, to the psychoanalyst, still another aspect of Hans's unconscious fantasies, namely that the falling-down horse stood not only for his father, but also for his mother in childbirth, the box-like carts and vehicles representing the womb. All these complicated, repressed feelings and perception were thus incorporated in a single phobia.

It is important to note that Little Hans was basically a straightforward, cheerful child who experienced normal psychosexual development marred only by the episode of the phobia, from which he recovered rather promptly. Fourteen years later, 19-year-old Hans came to see Freud. He had continued to develop well and had survived, without unusual difficulty, the divorce and remarriage of both parents. The problems of his childhood were used by Freud to illustrate the normal process of psychosexual development – the complex, intense, erotic drama of early childhood.

Source: www.webschooling.com/edupsy.html

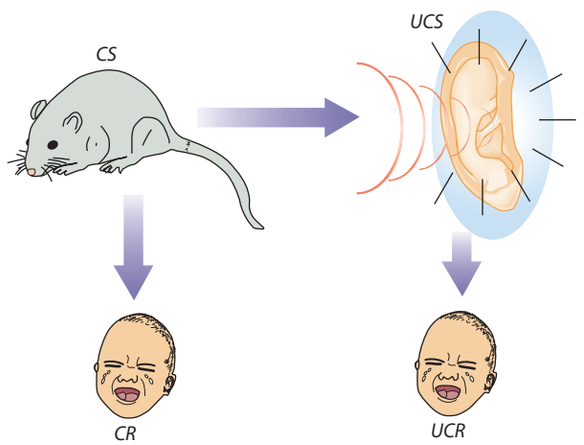


Figure 5.1
The 'Little Albert' classical conditioning study by Watson and Rayner (1920) demonstrated the acquisition of a phobia by pairing Little Albert's pet rat (the conditioned stimulus, CS) with a loud noise (unconditioned stimulus, UCS).

group of stimuli seem to have a high likelihood of being associated with pain or trauma (Seligman, 1971).

4 A simple conditioning model does not appear to account for the common clinical phenomenon of ***incubation***. Incubation is where fear increases in magnitude over successive encounters with the phobic stimulus, even though it is not followed by a traumatic consequence (Eysenck, 1979). Incubation is a phenomenon that is frequently observed clinically, but according to conditioning theory it should lead to extinction rather than enhancement of the fear response.

Incubation A common clinical phenomenon where fear increases in magnitude over successive encounters with the phobic stimulus – even though it is not followed by a traumatic consequence.

Due to these features it is problematic for a classical conditioning account to explain the acquisition of *all* phobias as resulting from traumatic conditioning episodes, but there is strong evidence that traumatic conditioning experiences are responsible for the acquisition of at least *some* phobias. These include dental phobia (Davey, 1988), choking phobia (Greenberg, Stern & Weilberg, 1988), accident phobia (Kuch, 1997), and most dog phobias (DiNardo, Guzy & Bak, 1988; Doogan & Thomas, 1992).

5.1.3.3 Evolutionary Accounts of Phobias

The fact that phobias tend to be focused on a limited set of fears that have evolutionary significance has led some researchers to suggest that we may be biologically prepared or pre-wired to acquire certain phobias. For instance, clinical phobias tend to cluster around things such as heights, water, spiders, snakes, blood and injury, all of which can be considered to have a real life-threatening significance that has been present for many thousands of years. In contrast, we rarely develop clinical phobias of life-threatening stimuli that have only appeared more recently in our phylogenetic past – such as guns and electricity. There are two predominant evolutionary theories of phobias.

First, Seligman (1971) argued that evolutionary selection pressures have evolved in us a biological predisposition to learn to associate fear with stimuli that have been hazardous for our pre-technological ancestors. That is, we tend to have a built-in predisposition to learn to fear things such as snakes, spiders, heights and water because these have been life-threatening to our ancestors, and

those of our ancestors that evolved a biological predisposition to fear these kinds of stimuli will have been more likely to survive and pass that fear predisposition on to future generations. This account is known as ***biological preparedness***, and has been supported by two lines of evidence.

Biological preparedness A theory which argues that we have a built-in predisposition to learn to fear things such as snakes, spiders, heights and water because these have been life-threatening to our ancestors.

Firstly, if participants in a classical conditioning experiment are shown pictures of 'fear-relevant' stimuli such as snakes and spiders (conditioned stimuli, CSs) paired with electric shock (unconditioned stimuli, UCSs), they develop fear of the CSs more quickly and show a greater resistance to extinction than if pictures of fear-irrelevant stimuli are used as CSs (e.g. pictures of houses) (Ohman, Erixon & Lofberg, 1975).

Also Cook and Mineka (1990) found that laboratory-reared rhesus monkeys that had never before seen a snake rapidly acquired fear reactions to snakes after being shown a demonstration of another monkey being frightened in the presence of a snake. They did not acquire fear reactions after watching a demonstration of another monkey being frightened in the presence of a stimulus such as a rabbit or a flower. Both studies suggest that humans and primates such as rhesus monkeys have an unlearned predisposition to rapidly acquire fear responses to some types of stimuli and not others (see Ohman & Mineka, 2001).

Secondly, Poulton and Menzies (2002) have argued for the existence of a limited number of innate, evolutionary-relevant fears. This ***non-associative fear acquisition*** model argues that fear of a set of biologically relevant stimuli develops naturally after very early encounters given normal maturational processes and normal background experiences, and no specific traumatic experiences with these stimuli are necessary to evoke this fear. Following repeated exposure to these stimuli, the innate fear reaction will habituate and should eventually disappear. Poulton and Menzies (2002) claim that this account explains why most children go through a discrete developmental period when they appear to be frightened of potential life-threatening stimuli such as heights and water (Graham & Gaffan, 1997), and why there is little evidence in retrospective studies for phobias such as height and water phobia being caused by specific traumatic experiences (Menzies & Clarke, 1993a,b, 1995a,b). This account then goes on to explain adult phobias as instances where these developmental phobias have failed to habituate properly.

Non-associative fear acquisition A model which argues that fear of a set of biologically relevant stimuli develops naturally after very early encounters given normal maturational processes and normal background experiences, and no specific traumatic experiences with these stimuli are necessary to evoke this fear.

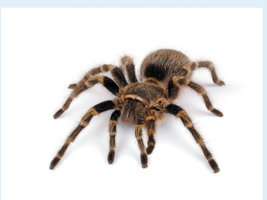
While evolutionary accounts are appealing and appear to have at least some face validity, we must be cautious about accepting them on the basis of existing evidence (Delprato, 1980). First, such accounts depend on the fact that current phobic stimuli have actually acted as important selection pressures over our evolutionary past. But this is very difficult to verify empirically. For example, do we tend to have phobic reactions to spiders because they once constituted an important life-threatening pressure on our pre-technological ancestors? There is no convincing evidence to suggest this. Secondly, evolutionary accounts can be constructed in a *post hoc* manner and are at risk of being either 'adaptive stories' (McNally, 1995) or 'imaginative reconstructions' (Lewontin, 1979) (cf. Merckelbach & de Jong, 1997). This view argues that it is possible to construct, *post hoc*, an adaptive scenario for the fear and avoidance of almost any stimulus or event (McNally, 1995) – see Activity Box 5.1. This

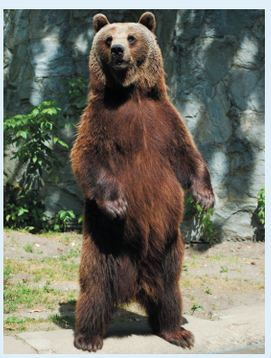
ACTIVITY BOX 5.1

Assessing evolutionary explanations of specific phobias

Some explanations of specific phobias argue that the rather limited set of fears that become the focus for clinical phobias (e.g. spiders, snakes, heights, water, blood and injury, confined spaces) are the result of evolutionary selection pressures. They argue that those of our ancestors that feared and avoided these stimuli survived and so passed their fear and avoidance tendencies on to their offspring. Evolutionary-based accounts such as these assume that those things that are the focus of phobias today did pose a real threat to the survival of our ancestors.

Have a look at what is displayed in the following pictures before taking a few minutes to write down as many reasons



as you can think of why each one might be a threat to the survival of a human being. Then read on.

You were probably able to think of a number of reasons why each of these might be a threat to the survival of a human being. Of these six stimuli, 1 and 2 are typical phobic stimuli, 3 and 4 are potentially life-threatening but are rarely the focus for phobias, and we would not normally consider 5 and 6 to be any threat to survival at all. Yet it is still not difficult to think of reasons why 5 and 6 *might* be dangerous if we are pressed to do so. This is know as the ***adaptive fallacy*** (McNally, 1995) – that is, you can usually think up reasons why any stimulus or event might be dangerous. Given that you were able to do this with all six of these stimuli:

1. Why is it that only two of them are the focus for phobias?
2. Why are two of them clearly dangerous, yet not the focus for phobias?

These are questions that evolutionary accounts of phobias need to address.

Adaptive fallacy The view that it is possible to think up a threatening or dangerous consequence for encountering any stimulus or situation.

does not mean that evolutionary accounts are wrong (see Ohman & Mineka, 2001, for a contemporary evolutionary account of phobias), merely that they are tantalizingly easy to propose but very difficult to substantiate.

5.1.3.4 Multiple Pathways to Phobias

There is no reason why the acquisition of all phobias should be explained by just a single process – and evidence is now accumulating to suggest that different types of phobias are acquired in quite different ways (Merckelbach, de Jong, Muris & van den Hout, 1996). We have already suggested that some phobias, such as dog phobia, dental phobia, choking phobia and accident phobia, are caused by traumatic conditioning experiences. In contrast, many other common phobias do not appear to be characterized by a traumatic experience at their outset – in fact, sufferers often cannot recall the exact onset of their phobia, which suggests that the onset may be gradual and precipitated by factors that are not immediately obvious to the individual. Phobias that fit this description include most animal phobias (including snake and spider phobia) (Murray & Foote, 1979; Merckelbach, Muris & Schouten, 1996), and height and water phobia (Menzies & Clarke, 1993a,b).

Recent evidence suggests that at least some phobias are closely associated with the emotion of ***disgust***. High levels of disgust sensitivity have been found to be associated with small animal phobias in general (Ware, Jain, Burgess & Davey, 1994; Davey, 1994b), spider phobia specifically (Mulkens, de Jong & Merckelbach, 1996), and has been hypothesized to play a role in mediating blood-injury-injection phobia (Page, 1994; but see de Jong & Merckelbach, 1998; Kleinknecht, Kleinknecht & Thorndike, 1997). Disgust is a food-rejection emotion whose purpose is to prevent the transmission of illness and disease through the oral incorporation of contaminated items (Davey, 1994c; Rozin & Fallon, 1987), and elevated disgust sensitivity implies increased avoidance of disgust-relevant objects (such as faeces or mucus). In the case of animal phobias, Davey (1992a) has argued that many animals that become the focus for phobic responding do so because they have disgust relevance. Specifically, they may have acquired a disgust relevance (1) by directly spreading disease and being a source of contamination (e.g. rats, cockroaches), (2) by possessing features which mimic primary disgust-relevant stimuli (by resembling, for example, faeces or mucus; e.g. slugs or animals that are perceived as slimy such as snakes, snails or lizards), or (3) by having contemporary or historical significance as stimuli that signalled disease, illness or contamination (e.g. maggots, spiders; cf. Davey, 1994a). This ***disease-avoidance model*** of animal phobias (Matchett & Davey, 1991) is supported by the findings that a high level of disgust sensitivity is a vulnerability factor for animal phobias (such as spider phobia), and can mitigate against successful therapy if it is not directly addressed in treatment (de Jong, Andrea & Muris, 1997; Mulkens, de Jong & Merckelbach, 1996).

Disgust A food-rejection emotion whose purpose is to prevent the transmission of illness and disease through the oral incorporation of contaminated items.

Disease-avoidance model The view that animal phobias are caused by attempts to avoid disease or illness that might be transmitted by these animals.

Alternatively, there is evidence that factors closely associated with ***panic*** and panic disorder (see section 5.3) are also linked to a number of specific phobias. First, there is a fairly high comorbidity rate between panic disorder and some specific phobias. Studies have identified comorbidity rates of between 40 and 65 per cent (de Ruiter, Rijken, Garssen, van Schaik & Kraaimaat, 1989; Starcevic, Uhlenhuth, Kellner, & Pathak, 1992), suggesting that panic is common in people suffering from many different types of specific phobia. Second, some categories of specific phobia – especially situational phobias – share important characteristics with panic disorder. For example, situational phobias appear to have a preponderance of spontaneous onsets typical of panic disorder (Himle, Crystal, Curtis & Fluent, 1991), have a significantly higher rate of comorbidity with panic disorder than do other types of phobias, such as animal phobias (Starcevic & Bogojevic, 1997), and frequently have uncontrollable panic attacks as one of the symptoms of phobic responding (e.g. height phobia: Antony, Brown & Barlow, 1997; flying phobia: McNally & Louro, 1992; claustrophobia: McIsaac, 1995). Similarly, both claustrophobia and height phobia have aetiological factors in common with panic disorder. For instance, subjective fear in claustrophobia is focused not just on external dangers but on anxiety expectancies and bodily sensations (Craske, Mohlman, Yi, Glover & Valeri, 1995), and spontaneous panic attacks are found significantly more often in claustrophobics than in other types of phobias (Rachman & Levitt, 1985; Craske & Sipsas, 1992). Height phobia is associated not only with heightened discrimination of bodily sensations, but also with a bias towards interpreting ambiguous bodily sensations

Panic A sudden uncontrollable fear or anxiety.

Plate 5.2
Small animal phobias are very common and include creepy-crawlies, insects, molluscs, rodents, spiders, snakes and lizards. Interestingly, if you are fearful of one of these types of animals, you are more likely to be fearful of others in this group. Fear of such animals may be related more to the emotion of disgust rather than to anxiety.

as threatening – a characteristic which is central to the aetiology of panic disorder (Davey, Menzies & Gallardo, 1997) (see section 5.3.2.2).

These examples suggest that specific phobias may have a number of different causes – depending on the nature of the phobic stimulus or event – and the aetiologies appear to involve quite different vulnerability factors and psychological processes. This being so, specific phobias are a coherent category only on the basis of their defining symptoms, and therapists may need to look more closely at the different aetiologies to construct successful treatments.

5.1.4 The Treatment of Phobias

Traditionally, successful treatment for specific phobias has tended to revolve around some form of exposure to the phobic stimulus or situation. In the past, behavioural treatments of choice for specific phobias have included systematic desensitization, flooding and counterconditioning (see Chapter 4, section 4.1.1.2). One important issue in therapy for specific phobias is to address the phobic beliefs that sufferers hold about their phobic event or situation (see Table 5.3). These beliefs are often dysfunctional in that they do not match with the reality of the threat (or lack of it) posed by the phobic stimulus, and they also maintain fear and avoidance responses. Because of their strong avoidance of any contact with their phobic situation, sufferers rarely find themselves in a situation where they encounter evidence that disconfirms their phobic beliefs (e.g. continually avoiding spiders never helps spider phobics to disconfirm their belief that, for example, 'I would come to physical harm in the presence of a spider') (Thorpe & Salkovskis, 1995, 1997). One important feature of ***exposure therapy*** is that it does put phobic individuals in situations where they can experience evidence that is contrary to their dysfunctional beliefs. More recently, specific behavioural treatments have been combined with cognitive therapy techniques to produce integrated short-term therapies that involve cognitive restructuring, intensive exposure to the phobic event or stimulus, and modelling, which can be effective in as little time as one 3-hour session (Ost, 1997) (see Treatment in Practice Box 5.1).

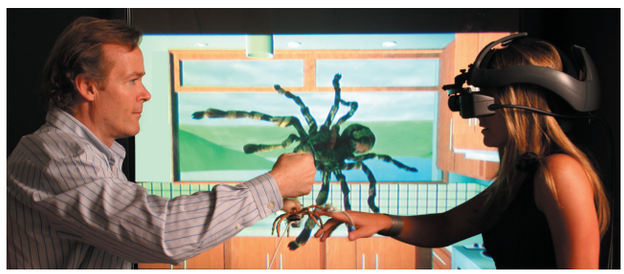


Plate 5.3
Exposure therapy is one of the most successful treatments for specific phobias. For many sufferers, however, the thought of having to encounter a real spider is severely distressing. Instead, therapists have developed virtual reality exposure treatments, in which the client can first encounter spiders in a controlled virtual environment. Photo courtesy Hunter Hoffman (Spider2007a6008in.jpg).

In conclusion, it must be remembered that many people can live with their phobias – either because the phobias are sub-clinical in intensity or people's fears are so specific that they do not interfere substantially with their daily lives. So only people with the most distressing or disabling phobias are the ones who seek treatment. In general, recently developed therapies for specific phobias have been shown to be extremely effective and successful (Ost, 1997). These therapies are usually multifaceted and combine aspects of exposure therapy with cognitive restructuring.

SELF-TEST QUESTIONS

- What are the main diagnostic criteria for specific phobias?
- What are the most common phobias, and what are the kinds of *phobic beliefs* that accompany them?
- How do classical conditioning and evolutionary theories attempt to explain the acquisition of phobias? What are their similarities and differences?
- Why is exposure such an important feature of treatment for specific phobias?

CLINICAL PERSPECTIVE: TREATMENT IN PRACTICE BOX 5.1

One-session rapid treatment of spider phobia

One-session treatments for specific phobias were developed during the 1990s and are remarkably successful as effective and long-lasting treatments for many specific phobias (Ost, 1997; Koch, Spates & Himle, 2004; Ost, Alm, Brandberg & Breitholtz, 2001). One-session treatments usually include a combination of graduated *in vivo* exposure and modelling. Below is an example of a one-session treatment procedure for spider phobia.

Step 1: Catching a small spider in a plastic bowl

The therapist first models how the client should pick up the spider by putting a bowl over it, sliding a card underneath to trap the spider, then picking up the bowl using the card as a lid. This is repeated 3–4 times. On the last occasion the client is instructed to hold the bowl in the palm of her hand. At this point a brief role-play can be carried out. The therapist plays the part of a person born blind, and the client has to describe what is happening, thus forcing the client to look at the spider in the bowl.

Step 2: Touching the spider

The therapist asks the client what she thinks will happen if she touches the spider. Most spider phobics say the spider will climb up their arm. This is a prediction that can be tested by the therapist who then touches the spider. This is repeated up to 10 times to show the client that the spider's reaction is almost always to run away. This is followed by the client touching the spider, usually with some physical guidance from the therapist.

Step 3: Holding the spider in the hand

The therapist takes the spider on her hand, letting it walk from one hand to another. The client is then encouraged to put her index finger on the therapist's hand so that the spider can walk across the finger and back to the therapist's hand. This is repeated a number of times until the spider walks across all the client's fingers. Gradually, the therapist withdraws physical support and the client allows the spider to walk from one hand to another.

These three steps are repeated with spiders of increasingly larger size. Throughout the session, the client is taught that she can acquire control over the spider by gradually being able to predict what the spider will do. The goal of the therapy is to ensure that at the end of the session, the client can handle two spiders with low or no anxiety and no longer believe her catastrophic cognitions about spiders.

Source: Ost (1997)

SECTION SUMMARY

5.1 Specific Phobias

- Specific phobias are defined as an excessive, unreasonable, persistent fear triggered by a specific object or situation.
- Around 10 per cent of people will meet DSM-IV-TR criteria for a specific phobia within their lifetime.
- **Common phobias** include small animal phobias (insects, rodents, spiders, snakes), social phobia, dental phobia, water phobia, height phobia, claustrophobia and blood-injury-inoculation (BII) phobia.
- The famous **'Little Albert'** study by Watson and Rayner (1920) is an example of how phobias can be acquired through classical conditioning.
- **Evolutionary accounts** of phobias suggest that we have an inbuilt biological predisposition to fear certain stimuli and events (e.g. heights, water, snakes), because these stimuli were life-threatening to our pre-technological ancestors. Evolutionary accounts of phobias include biological preparedness theory and the non-associative fear acquisition model.
- There is now strong evidence that different phobias may be caused by quite different processes: some involve classical conditioning, some are caused by high disgust sensitivity, while others appear to be caused by processes similar to those that cause panic disorder.
- Successful **treatment** for phobias tends to depend on some kind of exposure to the phobic stimulus or situation. Exposure therapies that are combined with cognitive behaviour therapy can be effective in as little time as one 3-hour session.

5.2 SOCIAL PHOBIA

Social phobia is distinguished by a severe and persistent fear of social or performance situations. Social phobics try to avoid any kind of social situation in which they believe they may behave in an embarrassing way or in which they believe they may be negatively evaluated. So pervasive

Social phobia A severe and persistent fear of social or performance situations.

is anxiety of these socially based situations that it has been more generally labelled as 'social anxiety disorder' (Liebowitz, Heimberg, Fresco, Travers & Stein, 2000) and is a predictor of several other debilitating problems such as depression and substance abuse (Rapee & Spence, 2004).

DSM-IV-TR describes some of the defining features of social phobia:

> individuals with social phobia experience concerns about embarrassment and are afraid that others will judge them to be anxious, weak, 'crazy', or stupid. They may fear public speaking because of concern that others will notice their trembling hands or voice. Or they may experience extreme anxiety when conversing with others because of fear they will appear inarticulate. They may avoid eating, drinking, or writing in public because of fear of being embarrassed by having others see their hands shake. Individuals with social phobia almost always experience symptoms of anxiety (e.g. palpitations, tremors, sweating, gastrointestinal discomfort, diarrhea, muscle tension, blushing, confusion) in the feared social situations, and, in severe cases, these symptoms may meet the criteria for a panic attack.
>
> (APA, 1994, p. 412)

Table 5.5 lists the DSM-IV-TR criteria for the diagnosis of social phobia.

Table 5.5 *DSM-IV-TR criteria for diagnosis of social phobia*

A	A marked persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The individual fears that he or she will act in a way that will be humiliating or embarrassing.
B	Exposure to the feared social situation almost invariably provokes anxiety, which may take the form of a situationally bound or situationally predisposed panic attack.
C	The person recognizes that the fear is excessive or unreasonable.
D	The feared social or performance situations are avoided or else are endured with intense anxiety or distress.
E	The avoidance or anxious anticipation of the feared social or performance situation(s) interferes significantly with the person's normal routine, occupational (academic) functioning, or social activities or relationships.
F	In individuals under 18 years, the duration is at least 6 months.
G	The fear or avoidance is not due to the direct physiological effects of a substance or a general medical condition, and is not better accounted for by another mental disorder.
H	If a general medical condition or another mental disorder is present, the fear in Criterion A is unrelated to it, e.g. the fear is not of stuttering, trembling in Parkinson's disease, or exhibiting abnormal eating behaviour in anorexia nervosa or bulimia nervosa.

5.2.1 Prevalence

Social phobia has a lifetime prevalence rate of between 7 and 13 per cent in Western societies and afflicts females significantly more often than males (Furmark, 2002). Age of onset is considerably earlier than for many of the other anxiety disorders, typically occurring in early to mid-teens and usually prior to 18 years of age (Rapee, 1995; Otto, Pollack, Maki, Gould et al., 2001). It is also a particularly persistent disorder and has the lowest overall remission rate of the main anxiety disorders (Massion, Dyck, Shea, Phillips et al., 2002). Cross-cultural studies have shown that prevalence rates are significantly lower in Southeast Asian countries (e.g. Korea and Taiwan) than in Western societies (Furmark, 2002), but this may be due at least in part to the fact that the expression of social anxiety differs across cultures. For example, in Japan, Taijin-kyofu-sho (TKS) is a form of social phobia in which the main fear is of offending others (see section 5.1). In Western cultures, social anxiety manifests itself primarily as fear of embarrassing oneself.

5.2.2 The Aetiology of Social Phobia

Although it is a phobia in its own right, social phobia is considered separately from simple phobias in DSM-IV-TR. There are a number of reasons for this. First, it is a highly prevalent disorder and compares with generalized anxiety disorder (GAD) as the most common of the anxiety disorders. Secondly, as we will see below, theories of social phobia suggest that factors rather specific to social anxiety are important in the aetiology of social phobia. In particular, social phobics possess a range of information processing and interpretation biases that cause them to make excessively negative predictions about future social events. We discuss these various types of bias in the following sections, after first considering genetic and developmental factors.

5.2.2.1 Genetic Factors

Evidence is accruing that there is an underlying genetic component to social phobia. For example, children with social phobia are more likely to have parents with social phobia than non-phobic children (Lieb, Wittchen, Hoefler, Fuetsch et al., 2000; Mancini, van Ameringen, Szatmari, Fugere et al., 1996), and twin studies also suggest that there is a significant but moderate genetic influence on the development of social phobia (Beatty, Heisel, Hall, Levine et al., 2002; Ollendick & Hirschfeld Becker, 2002). While indicating the importance of genetic influences, such studies do beg the question of what aspect of social phobia is inherited. However, some studies have been able to identify specific constructs related to social phobia that appear to have a genetic component. These include submissiveness, anxiousness, social avoidance and behavioural inhibition (Warren, Schmitz & Emde,

CLIENT'S PERSPECTIVE 5.1

Social phobia

'You have to be a sufferer of social phobia (SP) to understand the pure terror that a victim of this illness feels. It's the sort of blind panic dread and fear that one would feel facing a firing squad or if you fell into a lion's cage. You shake like a leaf, you blush, your mouth goes dry, you can't speak, you break out in a cold sweat, your legs feel so weak you think you're going to fall. Your thoughts become confused and disorientated. Forget butterflies in the stomach – your guts are twisted inside out with FEAR.

SP made me sink so low I ended up cleaning public toilets for a living. I never married (no, I'm not gay), I had no children, I never owned my own house. I rent a small flat in a very poor part of London all because of SEVERE SP. My parents were cold reserved people unable to show their emotions. I was never abused in any way but I look back on my childhood as a lonely unhappy time. Maybe that was the root cause of my phobia. I mention that because we can all think of something that may have been the cause. My SP started in my last year at school when I became very self-conscious and developed a fearful dread of being asked to read in front of the class. This extreme anxiety moved on with me into my working life. I was a smart looking young man so I got some good jobs, but because of SP no way could I hold them. Would you buy from a salesman who went a deep red, stammered, couldn't look you in the eye, and shook so much that even his head trembled? No – nor would the boss who in the end would say get lost, you're bad for business.

Over the years I slid down and down the social ladder with long spells out of work and, of course, no money. By the time I was thirty I could only do work where I did not have to deal with people like road sweep, night work in factories and in the end cleaning public toilets when closed at night. SP was now so bad I couldn't face going into a shop to buy something. To pass a queue of people waiting for a bus was hell. I was sure they were all staring at me. I couldn't sit facing other passengers on a train unless I had a newspaper to hide behind. If I attempted going into a restaurant or café, I'd pick a table facing the wall and if anyone sat at my table my hands would shake so much I couldn't get the food into my mouth. I became the ultimate night person, only going out late to walk the streets.'

Source: www.social-phobia.co.uk/

Clinical Commentary

This client's perspective highlights the extreme fear experienced by many social phobics in a range of social and performance situations, and the impact this can have on social functioning specifically and life planning more generally. This description highlights a number of important features of social phobia, including: (1) the biased interpretations that social phobics have of the reactions of others to them (e.g. 'To pass a queue of people waiting for a bus was hell. I was sure they were all staring at me'); (2) the belief that there are obvious physical signs of their nervousness which observers interpret judgementally (e.g. 'Would you buy from a salesman who went a deep red, stammered, couldn't look you in the eye?'); and (3) the tendency of social phobics to focus attention on themselves and their own reactions to the possible detriment of their own performance (e.g. 'My SP started in my last year at school when I became very self-conscious and developed a fearful dread of being asked to read in front of the class').

1999; Robinson, Reznick, Kagan & Corley, 1992). Other studies indicate that social phobia contains an inherited component that is shared with other anxiety disorders, which suggests that what might be inherited is a vulnerability to anxiety disorders generally rather than social phobia specifically (Kendler, Walters, Neale, Kessler et al., 1995; Nelson, Grant, Bucholz, Glowinski et al., 2000). Nevertheless, there may still be a modest inherited element that is specific to social phobia, and this has been estimated to account for as much as 13 per cent of the variance in social fears generally (Kendler, Myers, Prescott & Neale, 2001).

5.2.2.2 Developmental Factors

Because social phobia appears at a relatively early age compared to other anxiety disorders, it has been argued that various developmental factors and early experiences may precipitate the disorder (Neal & Edelmann, 2003). For example, there is considerable evidence that children who exhibit a behaviourally inhibited temperament style are at increased risk for subsequent social phobia (Neal, Edelmann & Glachan, 2002; Kagan, Reznick, Clarke, Snidman et al., 1984). However, it is also the case that a significant proportion of children who are highly behaviourally inhibited in early life do *not* subsequently develop social phobia – so childhood behavioural inhibition is not a sufficient condition for social phobia (Schwartz, Snidman & Kagan, 1999). Early parent–child interaction styles may also play a role in the development of social anxiety. Studies of parent–child interactions suggest that the parents of social phobics exert greater control over their children, show less warmth, are less sociable than the parents of individuals without social phobia and also use shame as a method of discipline (Rapee & Melville, 1997; Siqueland, Kendall & Steinberg, 1996; Bruch & Heimberg, 1994). While these factors

seem to be important predictors of subsequent social phobia, it is impossible to determine at present whether they represent actual causal factors.

5.2.2.3 Cognitive Factors

There appear to be a number of cognitive processes that are characteristic of social phobia and which may all act in some way to maintain fear of social situations (Stravynski, Bond & Amando, 2004). First, social phobics possess an information processing and interpretation bias in which they make excessively negative predictions about future social events (Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). For example, individuals with social phobia rate the probability of negative social events occurring as higher than either non-clinical controls or individuals with other anxiety disorders (Foa, Franklin, Perry & Herbert, 1996; Gilboa-Schechtman, Franklin & Foa, 2000), and this negative evaluation is likely to maintain their avoidance of social situations.

Secondly, individuals with social phobia interpret their performance in social situations significantly more critically than non-sufferers and independent assessors who have observed their behaviour (Stopa & Clark, 1993; Rapee & Lim, 1992). Social phobics also find it very difficult to process positive social feedback (Alden, Mellings & Laposa, 2004). This focus on negative aspects of the social situation, and the relative inability to take anything 'good' from a social performance, are likely to maintain the social phobic's dysfunctional beliefs that social situations are threatening and that their own performance is likely to be flawed.

Thirdly, some theories of social phobia argue that sufferers show a strong tendency to shift their attention inwards onto themselves and their own anxiety responses during social performance – especially when they fear they will be negatively evaluated (Clark & Wells, 1995; Rapee & Heimberg, 1997). This is known as ***self-focused attention*** (Spurr & Stopa, 2002; Bogels & Mansell, 2004) and has the effect of leading socially anxious individuals to believe they may look as anxious as they feel inside. This prevents objective processing of the social situation, leads them to engage in critical self-evaluation and may well adversely affect their actual performance in the social situation. Studies have shown that social phobics do indeed display higher levels of self-reported self-focused attention than non-clinical populations (Bogels & Lamers, 2002) and that they recall social memories more often from an observer perspective than from a personal perspective (suggesting that they do indeed 'observe' themselves while performing socially) (Wells, Clark & Ahmad, 1998). Self-focused attention therefore appears to have the effect of reinforcing individuals' perception of their own anxiety in the social situation, can distract individuals from focusing on the social task at hand and lead to unskilled performance, and result in avoidance of future social situations (Alden, Teschuk & Tee, 1992).

Self-focused attention A theory of social phobia arguing that sufferers show a strong tendency to shift their attention inwards onto themselves and their own anxiety responses during social performance – especially when they fear they will be negatively evaluated.

Finally, individuals with social phobia also indulge in excessive post-event processing of social events that includes critical self-appraisal of performance and assessment of symptom severity.

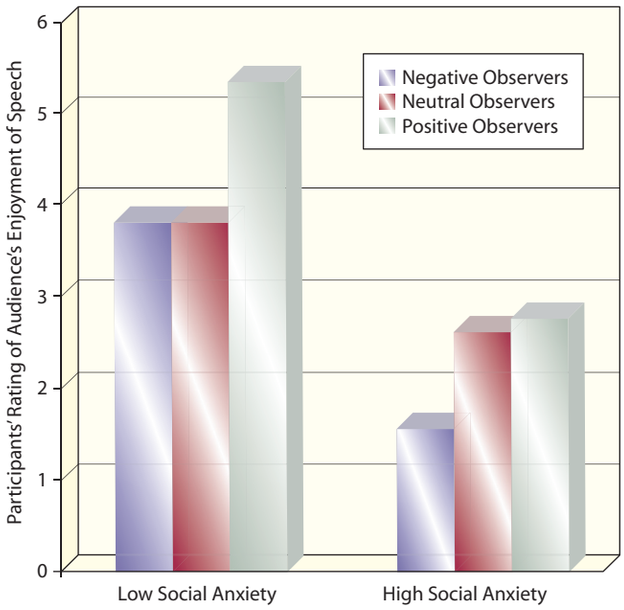


Figure 5.2
High and low socially anxious participants were asked to give a speech to a group of observers. After giving the speech, the high socially anxious participants rated observers' enjoyment of their speech significantly lower than low socially anxious participants. The high socially anxious participants do this even when the observers have been instructed to provide positive feedback, suggesting that socially phobic individuals do not attend to positive feedback cues given by an audience.

Source: Perowne & Mansell (2002).

Such post-event rumination has the effect of maintaining negative appraisals of performance over time and maintaining social anxiety (Abbott & Rapee, 2004; Rachman, Gruter-Andrew & Shafran, 2000).

5.2.3 The Treatment of Social Phobia

Both pharmacological treatments and cognitive behaviour therapies (CBT) have been shown to be effective in alleviating the symptoms of social phobia (Rodebaugh, Holaway & Heimberg, 2004; Davidson, 2003), and both are used widely to treat the disorder.

Successful CBT treatments include elements of the following:

- Exposure therapy, where the client remains in a feared social situation despite distress, either *in vivo* or through the therapist taking on the role of a stranger in a social situation (Heimberg & Becker, 2002). (See Treatment in Practice Box 5.2.)
- Social skills training, consisting of modelling, behavioural rehearsal, corrective feedback and positive reinforcement. This training addresses the social skills deficits often characteristic of social phobics.

CLINICAL PERSPECTIVE: TREATMENT IN PRACTICE BOX 5.2

Cognitive therapy for social phobia

The following is a step-by-step account of the cognitive therapy for social phobia devised by Clark and Wells (1995). The aims of this procedure are: (1) to decrease self-focused attention; (2) to reduce the level of negative interpretations of internal information (e.g. sweating as a sign of poor performance); (3) to eliminate the use of safety behaviours which maintain negative beliefs (e.g. if the phobic believes he is trembling and that this may be visible, he may grip objects tightly in order to conceal it: this response merely maintains the phobic's belief that he is anxious and trembling); and (4) to reduce negative post-event processing (see section 5.2.2.3).

Step 1: The initial phase is designed to inform clients about those factors that are maintaining their social phobia (see above), and that these are the factors that the therapy is specifically designed to target.

Step 2: The second phase attempts to manipulate safety behaviours. Here clients have to role-play a social situation and observe their own responses and identify key safety behaviours. Clients will then attempt to drop these safety behaviours during subsequent role-playing.

Step 3: Clients are trained to shift their attention externally and away from their own internal responses and cognitions.

Step 4: Video feedback of performance can be used to modify distorted self-imagery.

Step 5: Clients are provided with some behavioural experiments in which they specify their fears of particular social situations and then test out whether they occurred during role-play sessions.

Step 6: Problematic post-event processing is identified and modified using focused cognitive restructuring techniques.

Source: Stangier, Heidenreich, Peitz, Lauterbach & Clark (2003)

- Cognitive restructuring, designed to challenge and replace the negative biases in information processing and the dysfunctional negative self-evaluations of social performance, and to reduce self-focused attention (Rodebaugh & Chambless, 2004).

Each of these elements used alone does show therapeutic gains, but an integrated CBT programme appears to result in maintenance of gains over 6- to 12-month follow-up periods (Feske & Chambless, 1995).

Drugs such as monoamine oxidase inhibitors (MAOIs) and, more recently, serotonin reuptake inhibitors (SSRIs) (see Chapter 4, section 4.1.1.6) have been shown to cause improvement in measures of social anxiety (Blanco, Schneier, Schmidt, Blanco-Jerez et al., 2003; van der Linden, Stein & van Balkom, 2000). Comparative outcome studies have suggested that both pharmacological and CBT treatments are more effective than non-treatment controls (Gould, Buckminster, Pollock, Otto & Yap, 1997), but that the two types of therapy may offer complementary benefits, drug therapy offering a more immediate benefit than CBT, but CBT helping clients to maintain their therapeutic gains over time (Liebowitz, Heimberg, Schneier, Hope et al., 1999).

SELF-TEST QUESTIONS

- What are the main diagnostic criteria for social phobia and how does this disorder manifest itself?
- Can you describe the various cognitive factors that appear to play an important role in maintaining social phobia?
- How do cognitive behaviour therapies (CBT) and drug treatments complement each other in the treatment of social phobia?

SECTION SUMMARY

5.2 Social Phobia

- Social phobia is distinguished by a severe and persistent fear of social or performance situations.
- Social phobia has a lifetime prevalence rate of between 7 and 13 per cent in Western societies.
- There is evidence for a **genetic** component to social phobia, but this may be a predisposition to develop anxiety disorders generally rather than social phobia specifically.
- There are a number of **cognitive** factors that are characteristic of social phobics. These include a tendency to: (1) make excessively negative predictions about future social events; (2) over-critically evaluate their own social performance; (3) shift their attention inwards on to themselves; and (4) indulge in post-event critical appraisal of their own performance.
- Both **monoamine oxidase inhibitors** and **serotonin reuptake inhibitors** have been shown to be successful pharmacological treatments for social phobia, as well as **cognitive behaviour therapy** (CBT).

5.3 PANIC DISORDER

Panic disorder An anxiety disorder characterized by repeated panic or anxiety attacks.

As the name suggests, ***panic disorder*** is characterized by repeated panic or anxiety attacks. These attacks are associated with a variety of physical symptoms, including heart palpitations, perspiring, dizziness, hyperventilating, nausea and trembling (see Case History 5.1). In addition, the individual may experience real feelings of terror or severe apprehension and depersonalization (a feeling of not being connected to your own body or in real contact with what is happening around you). Most people will experience at least one panic attack in their lifetime, but panic disorder is diagnosed when recurrent, unexpected panic attacks keep occurring and are followed by at least one month of persistent concerns about having a further attack. For some individuals panic attacks are unpredictable, but for others they may become associated (perhaps through classical conditioning: see Chapter 1, section 1.1.3.2) to specific situations or events (e.g. riding on public transport).

DSM-IV-TR defines a panic attack as a discrete period of intense fear or discomfort in which four or more of a list of symptoms develop suddenly and reach a peak within 10 minutes (see Table 5.6). The criteria for panic disorder state that individuals must experience recurrent panic attacks, and in addition they must develop a persistent concern that future panic attacks will occur (see Table 5.7). The frequency of panic attacks in panic disorder can vary considerably between individuals from one attack per week to frequent daily attacks. Panic disorder is associated with a number of fears and apprehensions that the sufferer develops. These include fears that the attacks indicate an underlying serious medical condition (e.g. cardiac disease, seizure disorder), even though repeated medical tests indicate no life-threatening illness. Others feel they are losing control or simply 'going crazy'. Sufferers often make significant changes to their behaviour and their life as a result of the disorder. For example, they may ensure

CASE HISTORY 5.1

Panic disorder

Marilyn is a 33-year-old single woman who works at a local telephone company and lives alone in her apartment. She has panic disorder with agoraphobia and her first panic attack occurred 3 years ago when driving over a bridge on a very rainy day. She experienced dizziness, pounding heart, trembling and difficulty breathing. She was terrified her symptoms meant she was about to pass out and lose control of her car. Since that time she has experienced eight unexpected panic attacks during which she feared she was about to pass out and lose control of herself. She frequently experiences limited symptom attacks (e.g. feels dizzy and fears she may pass out). As a result of her intense fear of having another panic attack she avoids the following situations: waiting in line, drinking alcohol, elevators, movie theatres, driving over bridges, driving on the freeway, flying by plane, and heights (e.g. she will not go out on her tenth floor balcony). She is often late for work because of taking a route that doesn't require her to take the freeway. She also finds herself avoiding more and more activities. She frequently feels tearful and on guard. Sometimes she gets very angry at herself as she does not understand why she has become so fearful and avoidant.

Sharon is a 38-year-old single mother of two teenage daughters who works as a fitness instructor at a local gym. She experienced her first panic attack during her teens when watching a horror movie with friends at a local movie theatre. Since that time she has experienced one to two full panic attacks per year that come out of the blue in a variety of situations (e.g. while waiting in line at the bank, at a shopping mall, walking alone at the park). The panic attacks recurred out of the blue when she was 29 while eating a hot and spicy meal at a local restaurant. Her panic attacks always include dizziness, feeling of choking, dry mouth, unreality, feeling detached from her body and feeling as if she may lose bowel control. Her main fear is that she is dying due to a stroke, although medical problems have been ruled out. Sharon does not avoid anything to prevent the panic attacks and there has not been a huge negative impact of the panic attacks upon her work, family or social functioning.

Source: www.anxietybc.com/disorders/PANIC.html

Clinical Commentary

Both Marilyn and Sharon exhibit a number of physical symptoms typical of panic attacks, although these examples show that not everyone experiences similar symptoms. Panic attacks often come 'out of the blue' and are unpredictable, which adds to their frightening nature. In both examples the individual believes that the symptoms are signs of impending physical illness or loss of control (catastrophic misinterpretation). The pervasive fear of further attacks means that Marilyn has developed avoidance responses in an attempt to minimize future attacks. These avoidance responses interfere with her normal daily life (causing further stress), and inadvertently help to maintain dysfunctional catastrophic beliefs.

Table 5.6 *DSM-IV-TR criteria for a panic attack*

A discrete period of intense fear or discomfort, in which four (or more) of the following symptoms develop abruptly and reach 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 (feelings of unreality) or depersonalization (being detached from oneself)
(10) Fear of losing control or going crazy
(11) Fear of dying
(12) Paresthesias (numbness or tingling sensations)
(13) Chills or hot flushes

Table 5.7 *DSM-IV-TR criteria for diagnosing panic disorder*

A Both (1) and (2):
  (1) recurrent unexpected panic attacks
  (2) at least one of the attacks has been followed by 1 month (or more) of one (or more) of the following:
    (a) persistent concern about having additional attacks
    (b) worry about the implications of the attacks or their consequences (e.g. losing control, having a heart attack)
    (c) a significant change in behaviour related to the attacks

B The panic attacks are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition.

C The panic attacks are not better accounted for by another mental disorder, such as social phobia, specific phobia, OCD, PTSD, or separation anxiety disorder.

NB Panic disorder will normally be diagnosed 'with' or 'without' agoraphobia, depending on whether the individual exhibits anxiety about being in places or situations from which escape might be difficult.

that there is always a 'safe' place available in case they have an attack, and this may cause them to avoid social situations and even quit their job. Concerns about future attacks often result in the development of avoidant behaviour; sufferers may find it difficult to leave the 'safety' of their own home, in which case panic disorder with agoraphobia is a common diagnosis.

5.3.1 Prevalence

The lifetime prevalence of panic disorder is between 1.5 and 3.5 per cent (DSM-IV-TR) and is experienced more by women than men (relative lifetime prevalence rates of 5 per cent and 2 per cent respectively; Kessler et al., 1994). Onset is common in adolescence or early adulthood and can often be associated with a period of stress in the individual's life (Pollard, Pollard & Corn, 1989). There is some evidence for culturally determined variance in both the prevalence of panic disorder and in the way that panic disorder may manifest itself. For example, in some Asian societies, prevalence is particularly low, possibly because of the stigma related to admitting and reporting psychological disorders (e.g. in Taiwan; Weissman, Bland, Canino, Faravelli et al., 1997). However, in other cultures, panic disorder may be expressed in the form of quite different symptoms. For example, *Ataque de Nervios* is an anxiety-based disorder found almost exclusively in Latinos in the Caribbean. This appears to be a form of panic disorder brought on by stressful life events (such as economic or marital difficulties) but whose expression is determined by the social and cultural norms within that cultural group (see Chapter 1, Focus Point 1.3). In particular, Latino cultures place less emphasis on self-control and emotional restraint than other Western cultures, and so the distress of panic disorder in Latinos tends to be externalized in the form of screaming, uncontrolled behaviour and aggression (Salman et al., 1998). In contrast, in Western cultures the distress of panic disorder is usually coped with by adopting avoidance and withdrawal strategies – hence the common diagnosis of panic disorder with agoraphobia.

It is important to remember that panic attacks may be a feature of the symptoms in a number of the anxiety disorders (e.g. specific phobias and social phobia). However, panic disorder itself is characterized by frequent uncontrollable panic attacks, and an important aspect of this anxiety-based problem is the individual's intense fear of experiencing panic attacks. As we shall see, it is this latter feature of panic disorder that plays a central role in theories of the disorder.

5.3.2 The Aetiology of Panic Disorder

Because of the intense nature of the physical symptoms of panic disorder, many researchers have looked towards biological causes. However, it has become clear that there are also important psychological and cognitive factors that contribute to the aetiology and maintenance of panic disorder.

5.3.2.1 Biological Theories of Panic Disorder

The Role of Hyperventilation ***Hyperventilation*** is a common feature of panic attacks, and Ley (1987) has suggested that dysfunctional breathing patterns may trigger a series of autonomic reactions that precipitate a full-blown panic attack. Hyperventilation is defined as a 'minute ventilation that exceeds metabolic demand' and has an end result of raising blood pH level. Oxygen is then delivered less efficiently to body cells, which can lead to cardiovascular changes that try to help compensate for the lack of oxygen in the cells, and in turn can produce the symptoms of panic attacks that are recognized as anxiety (Zvolensky & Eifert, 2001).

Hyperventilation A rapid form of breathing that results in ventilation exceeding metabolic demand and has an end result of raising blood pH level. A common feature of panic attacks.

This type of explanation has been partially supported by evidence from what are called ***biological challenge tests*** (see Figure 5.3), where panic attacks have been induced by administering carbon dioxide-enriched air (CO_2) or by encouraging hyperventilation (Ley & Walker, 1973). Similarly, sensitivity to increases in CO_2 have been suggested as a risk factor for panic disorder (Papp, Klein & Gorman, 1993), and have given rise to what are known as ***suffocation alarm theories*** of panic disorder, where a combination of increased CO_2 intake may activate an over-sensitive suffocation alarm system and give rise to the intense terror and anxiety experience during a panic attack (Klein, 1993). In support of this account, panic disorder patients do report significantly more symptoms of shortness of breath when anxious, and more frequent frightening suffocation experiences than other anxiety patients. However, when panic disorder patients are asked to participate in periods of breath-holding, they do not report any greater levels of anxiety than non-anxious controls, suggesting that they do not necessarily possess a more sensitive suffocation alarm system (Roth, Wilhelm & Trabert, 1998).

Biological challenge tests Research in which panic attacks are induced by administering carbon dioxide-enriched air (CO_2) or by encouraging hyperventilation.

Suffocation alarm theories Models of panic disorder in which a combination of increased CO_2 intake may activate an oversensitive suffocation alarm system and give rise to the intense terror and anxiety experienced during a panic attack.

One further intriguing feature of the hyperventilation account is that, while biological challenge tests produce physiological changes that often provoke full-blown panic attacks, they only tend to do so in individuals with a history of panic attacks or panic disorder (Margraf, Ehlers & Roth, 1986). This is the case even though the physiological changes caused by biological challenge tests are the same in people diagnosed with panic disorder and those who have no anxiety disorder (Gorman, Kent, Martinez et al., 2001). This evidence quite strongly suggests that an important causal factor in panic disorder is the way that individuals *interpret* the physiological changes caused by the biological challenge, and this gives rise to the psychological accounts of panic disorder that have been developed over the past 25 years.

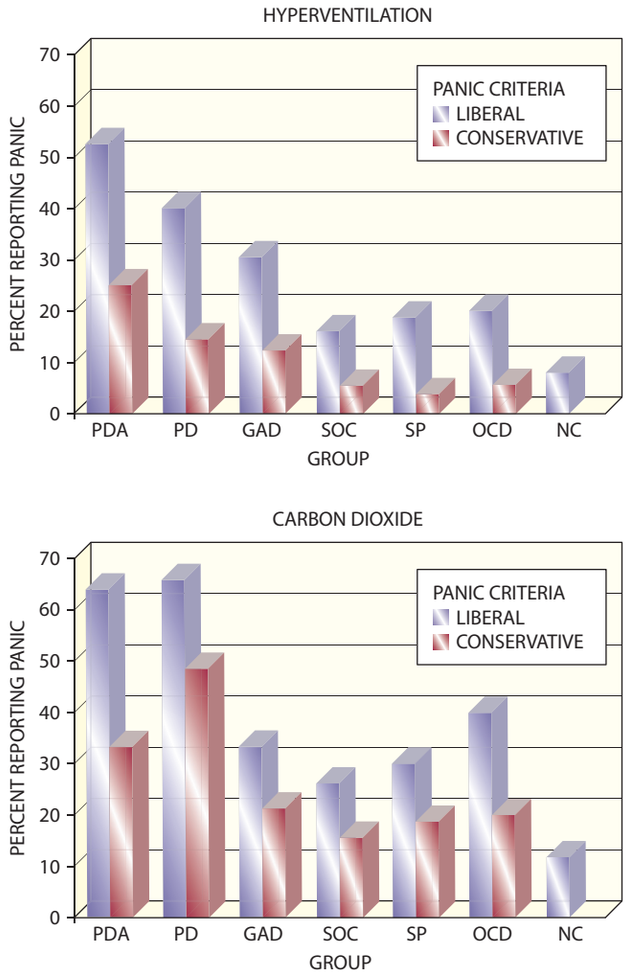


Figure 5.3
Proportion of participants with a range of anxiety disorders who report a panic attack after either (a) being asked to hyperventilate or (b) receiving a CO_2 challenge. (PDA = panic disorder with agoraphobia; PD = panic disorder; GAD = generalized anxiety disorder; SOC = social phobia; SP = simple phobia; OCD = obsessive-compulsive disorder; NC = non-anxious controls).

Source: Rapee, Brown, Antony & Barlow (1992).

Noradrenergic Overactivity A further account of panic disorder that alludes to biological differences between those who suffer panic disorder and those who do not is that the disorder may be caused by overactivity in the noradrenergic neurotransmitter system (Redmond, 1977). There is certainly evidence that the noradrenaline network may mediate the effects of biological challenges in producing panic attacks (Bailey, Argyropoulos, Lightman & Nutt, 2003), and that norepinephrine is implicated in the symptomatology of panic disorder (Sand, Mori, Godau, Stober et al., 2002). One particular view related to putative overactivity in the noradrenergic system is that patients with panic disorder are deficient in the gamma-aminobutryic (GABA) neurons that inhibit

RESEARCH METHODS IN CLINICAL PSYCHOLOGY BOX 5.1

Biological challenge procedures and panic disorder

A number of provocative agents can be used to induce panic attacks. In research on panic disorder, they have been used to investigate whether individuals with panic disorder have a greater sensitivity to such agents. Indeed, all of the agents listed below induce panic attacks more readily in panic disorder sufferers than in normal controls. This suggests that individuals with panic disorder may be biologically or psychologically sensitive to the effects of these agents.

Agent	*Biological mechanism for causing panic*	*Effect on panic disorder sufferers and non-clinical controls*	*References*
Sodium lactate	Possibly through alkalinization of the blood	Tends to induce panic attacks in panic disorder sufferers but not in normal controls	Bourin et al. (1998) Krystal et al. (1996)
Carbon dioxide (CO_2)	Increased breathing and hyperventilation in order to remove CO_2 causes respiratory alkalosis	Produces an increase in subjective anxiety in panic disorder sufferers including symptoms of panic	Gorman et al. (1984) Perna et al. (1995)
Yohimbine	Increases central noradrenergic activity in the locus ceruleus (an area of the brain implicated in panic)	Tends to induce panic attacks in a majority of panic disorder sufferers, and greater levels of self-reported anxiety than controls	Charney et al. (1984) Charney et al. (1987)
Caffeine	Indirectly increases norepinephrine and increases arousal	Tends to cause panic symptoms in panic disorder sufferers but not in normal controls	Charney et al. (1985) Beck & Berisford (1992)

noradrenergic activity, and PET scan studies have tended to support this view (Malizia, Cunningham, Bell, Liddle et al., 1998). Nevertheless, it is still unclear whether the role of the noradrenergic system is to mediate the symptoms of panic attacks when they occur, or whether noradrenergic overactivity represents a vulnerability factor in the aetiology of panic disorder.

5.3.2.2 Psychological Theories of Panic Disorder

Classical Conditioning Goldstein and Chambless (1978) were the first to suggest that an important feature of panic disorder was the sufferer's 'fear of fear'. That is, when they detected what they thought were any internal signs of a panic attack (e.g. mild dizziness), sufferers would immediately become fearful of the possible consequences. This would then precipitate a full-blown attack. Goldstein and Chambless (1978) interpreted this as a form of interoceptive classical conditioning, in which the internal cue (such as dizziness) had become established as an internal conditioned stimulus (CS) predicting a panic attack (the unconditioned stimulus, UCS). However, while this account has intuitive appeal, it is not clear in conditioning terms what is the CS and what is the UCS. For example, is a skipped heartbeat a CS that precipitates a panic attack, or is it a symptom of the panic attack itself (the UCS) (McNally, 1990)? Bouton, Mineka and Barlow (2001) have attempted to address these conceptual difficulties by suggesting that anxiety and panic are separable aspects of panic disorder. They suggest that anxiety is anticipatory and prepares the system for a trauma, whereas panic deals with a trauma that is already in progress. In this conditioning account, anxiety is the learned reaction, called conditioned response (CR), to the detection of cues, the conditioned stimulus (CS), that might predict a panic attack, and once conditioned anxiety develops it will exacerbate subsequent panic attacks and lead to the development of panic disorder. As predicted by this model, studies confirm that panic attacks are regularly preceded by anxiety in individuals with panic disorder (Barlow, 1988; Kenardy & Taylor, 1999).

Anxiety Sensitivity What is clear about the phenomenology of panic disorder is that sufferers become extremely anxious when they detect any cues (internal or external) that may be indicative of a panic attack. So any theory of panic disorder needs to explain

why sufferers are made anxious by the detection of these cues, and how this subsequently leads to a full-blown panic attack. Individuals who do *not* suffer panic disorder report a number of interoceptive and affective responses in biological challenge tests, but they are only rarely made anxious by these symptoms and hardly ever panic (Bass & Gardner, 1985; Starkman, Zelnik, Nesse & Cameron, 1985). So, what determines whether someone will panic in response to unusual bodily sensations? Reiss and McNally (1985) proposed that some individuals have pre-existing beliefs that bodily sensations may predict harmful consequences. They developed the construct of ***anxiety sensitivity***, which refers to fears of anxiety symptoms that are based on beliefs that such symptoms have harmful consequences (e.g. that a rapid heartbeat predicts an impending heart attack). In order to measure this construct, Reiss, Peterson, Gursky and McNally (1986) developed the ***Anxiety Sensitivity Index*** (ASI) (see also the Revised Anxiety Sensitivity Index, ASI-R: Taylor & Cox, 1998), which contains items such as 'Unusual body sensations scare me' and 'It scares me when I feel faint' (see Table 5.8). Studies have shown that individuals with panic disorder score significantly higher on the ASI than either non-clinical controls or individuals diagnosed with other anxiety disorders (Taylor & Cox, 1998; Rapee, Ancis & Barlow, 1988). Furthermore, in a prospective study, high ASI scores predicted the occurrence of subsequent panic attacks in army recruits undergoing a stressful period of training (Schmidt, Lerew & Jackson, 1997), which suggests that elevated anxiety sensitivity may be a risk factor for panic and perhaps panic disorder (McNally, 2002).

Anxiety sensitivity Fears of anxiety symptoms based on beliefs that such symptoms have harmful consequences (e.g. that a rapid heart beat predicts an impending heart attack).

Anxiety Sensitivity Index A measure, developed by Reiss, Peterson, Gursky & McNally (1986), to measure anxiety sensitivity.

Table 5.8 *Example items from the Anxiety Sensitivity Index (ASI-R) (Taylor & Cox, 1998)*

The ASI-R measures anxiety sensitivity, which is a measure of an individual's fear of anxiety. Anxiety sensitivity is one of the best predictors of future panic attacks and may be a risk factor for panic disorder.

1. When I feel like I'm not getting enough air I get scared that I might suffocate.
2. When my chest feels tight, I get scared I won't be able to breathe properly.
3. It scares me when I feel faint.
4. When my throat feels tight, I worry that I could choke to death.
5. It scares me when my heart beats rapidly.
6. It scares me when I feel shaky (trembly).
7. When I have trouble swallowing, I worry that I could choke.
8. It scares me when my body feels strange or different in some way.
9. I think it would be horrible for me to faint in public.
10. When I tremble in the presence of others I fear what people might think of me.
11. When I feel a strong pain in my stomach, I worry it could be cancer.
12. When my heart is beating rapidly, I worry that I might be having a stroke.
13. When I feel dizzy, I worry there is something wrong with my brain.
14. When my stomach is upset, I worry that I might be seriously ill.
15. It scares me when I feel tingling or prickling sensations in my hands.
16. When I feel 'spacey' or spaced out I worry that I may be mentally ill.

Catastrophic Misinterpretation of Bodily Sensations

Based on the fact that panic disorder sufferers are clearly anxious about the possible consequences of bodily symptoms, Clark (1986, 1988) developed an influential model of panic disorder in which he hyopthesized that panic attacks are precipitated by individuals catastrophically misinterpreting their bodily sensations as threatening. Many body sensations are ambiguous: for example, the heart skipping a beat could mean either an imminent heart attack (negative interpretation) or that someone you like has just walked into the room (positive interpretation). However, individuals who tend to develop panic disorder appear to ***catastrophically misinterpret bodily sensations***, that is, they have a cognitive bias towards accepting the more threatening interpretation of their sensations (Clark, Salkovskis, Ost, Breitholz et al., 1997; see Austin & Richards, 2001, for a review). Clark argues that this leads to a vicious cycle where any apprehension is interpreted threateningly and increases the perceived threat, which leads to an escalation of anxiety symptoms that then precipitate a panic attack. This is represented schematically in Figure 5.4. There is a good deal of evidence to support this psychological account. Individuals with panic disorder have been shown to attend to and discriminate their bodily sensations more closely than individuals without panic disorder (Ehlers & Breuer, 1992), and panic disorder sufferers report that thoughts of imminent danger typically accompany their attacks (Hibbert, 1984; Ottaviani & Beck, 1987). In addition, individuals with panic disorder will experience a panic attack when they have been told they will receive a CO_2 challenge, but in fact are given only *compressed air* (Sanderson, Rapee & Barlow, 1989), suggesting that just the *expectancy* of an attack is enough to trigger one.

Catastrophic misinterpretation of bodily sensations A feature of panic disorders where there is a cognitive bias towards accepting the more threatening interpretation of an individual's own sensations.

All of these accounts suggest there is likely to be an important psychological component to the development of panic disorder that involves a negatively valenced bias in how individuals interpret and react to their own bodily sensations. This interpretation

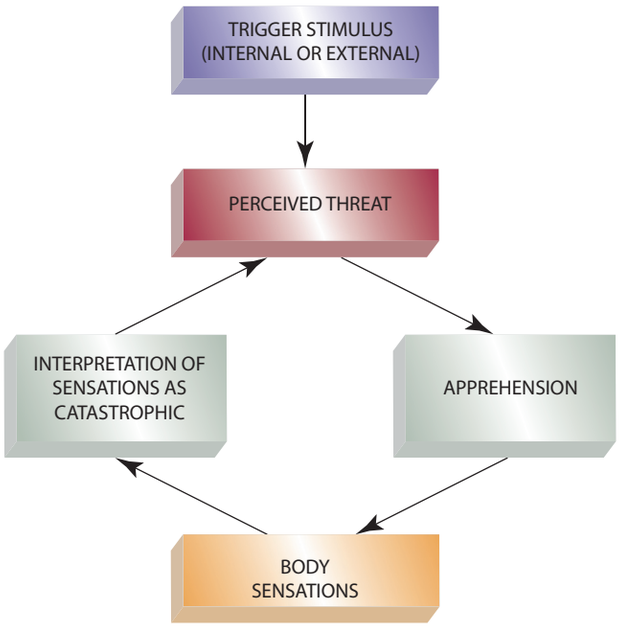


Figure 5.4 *Clark's (1986) model of panic disorder*
Perception of a threat triggers apprehension and then bodily sensations associated with that apprehension are interpreted catastrophically. This causes further anxiety, which feeds into a vicious cycle that triggers a full-blown panic attack.

bias appears to trigger anxiety, which in turn triggers a panic attack. The issues that remain to be resolved in these accounts are (1) exactly how the anxiety elicited by catastrophic misinterpretation of bodily sensations leads to panic, and (2) why some individuals have acquired high levels of anxiety sensitivity and catastrophic beliefs in the first place.

5.3.3 The Treatment of Panic Disorder

Because of the distressing physical symptoms experienced in panic disorder, psychoactive medication is usually the first line of treatment provided for sufferers, and both tricyclic antidepressants and benzodiazepines may be effective in controlling symptoms (Roy-Byrne & Cowley, 1998) (see Chapter 4, section 4.1.1.6). However, there is good evidence that structured exposure therapy or cognitive behaviour therapy (CBT) is as effective, if not superior, to drug treatments over the longer term (e.g. Craske, Brown & Barlow, 1991).

In exposure-based treatments, the client is persuaded to experience the conditions that precipitate a panic attack in the controlled environment of the therapy situation (Craske & Barlow, 2001). For example, someone whose attacks are preceded by bouts of dizziness may be asked to spin around in a chair, or if hyperventilation is a trigger, the individual will be asked to breathe rapidly for a period of time. At the first bodily signs of the symptoms

CLINICAL PERSPECTIVE: TREATMENT IN PRACTICE BOX 5.3

Cognitive therapy for panic disorder

The following transcript gives an example of how a cognitive therapist (T) would try to challenge the catastrophic beliefs of a panic disorder sufferer (P) who believes that signs of an impending panic attack are signals for an imminent heart attack.

P: When I'm panicking, it's terrible, I can feel my heart pounding; it's so bad I think it could burst through my chest.

T: What thoughts go through your mind when your heart is pounding like that?

P: Well, I'll tell you what I think; it's so bad that I think I'm going to have a heart attack. It can't be good for your heart beating like that.

T: So you're concerned that anxiety can damage your heart or cause a heart attack.

P: Yes, it must do you some damage. You hear of people dropping down dead from heart attacks caused by stress.

T: Do you think more people have stress in their lives than die of heart attacks?

P: Yes, I suppose so.

T: How can that be if stress causes heart attacks?

P: Well, I suppose it doesn't always cause problems. Maybe it does only in some people.

T: Yes, that's right; stress can cause some problems in some people. It tends to be people who have something wrong with their hearts in the first place. But stress is not necessarily the same as sudden anxiety or panic. When you panic your body releases adrenalin which causes the heart to speed up and your body to work faster. It's a way of preparing you to deal better with danger. If adrenalin damaged the heart or body, how would people have evolved from dangerous primitive times? Wouldn't we all have been wiped out?

P: Yes, I suppose so.

T: So maybe panic itself doesn't cause heart attacks, there has to be something physically wrong for that to happen. When people have had heart attacks they are often given an injection of adrenalin directly into the heart in order to help start it again. Do you think they would do that if it damaged the heart even more?

P: No, I'm sure they wouldn't.

T: So, how much do you now believe that anxiety and panic will damage your heart?

Source: Wells (1997), pp. 123–124

associated with panic, the client is then asked to apply cognitive and physical techniques designed to manage the attack (such as applying relaxation techniques). This enables the client to manage the attack under relatively 'safe' conditions, and to learn to exercise control over the cues that would normally predict panic (Craske, Maidenberg & Bystritsky, 1995).

Clearly, an important distinguishing feature of individuals with panic disorder is their fear of bodily sensations, their catastrophic misinterpretation of these sensations, and the effect these cognitions have in triggering a panic attack. The development of CBT for panic disorder has therefore focused specifically on providing clients with challenges to these beliefs in the form of both corrective information and experiences designed to eliminate faulty emotional responding (e.g. Clark, Salkovskis, Hackmann, Middleton et al., 1994; Telch, Lucas, Schmidt, Hanna et al., 1993; Luermans, De Cort, Scruers & Griez, 2004). A typical treatment programme would include:

1. Education about the nature and physiology of panic attacks.
2. Breathing training designed to control hyperventilation.
3. Cognitive restructuring therapy to identify and challenge faulty threat perceptions.
4. Interoceptive exposure to reduce fear of harmless bodily sensations.
5. Prevention of 'safety' behaviours that may maintain attacks and avoid disconfirmation of maladaptive threat beliefs.

Such programmes have been shown to produce a durable reduction in symptoms and a significant increase in quality of life for panic disorder sufferers (Barlow, Gorman, Shear & Woods, 2000; Telch, Schmidt, Jaimez, Jacquin & Harrington, 1995). More recent studies have also suggested that such CBT programmes may be effective specifically because they significantly reduce the tendency to react fearfully to benign bodily sensations (Smits, Powers, Cho & Telch, 2004).

SELF-TEST QUESTIONS

- Can you describe the main symptoms of a panic attack, and the diagnostic criteria for panic disorder?
- How does hyperventilation cause a panic attack?
- What role does the catastrophic misinterpretation of bodily sensations play in the acquisition and maintenance of panic disorder?
- Can psychological explanations of panic disorder explain more of the facts of panic disorder than biological explanations?
- What are the important features of cognitive behaviour therapy for panic disorder?

SECTION SUMMARY

5.3 Panic Disorder

- Panic disorder is characterized by repeated panic or anxiety attacks associated with a variety of physical symptoms, including heart palpitations, dizziness, perspiring, hyperventilation, nausea, trembling and depersonalization.
- The lifetime prevalence rate for panic disorder is between 1.5 and 3.5 per cent, although prevalence rates do differ between different cultures.
- **Hyperventilation** is a common feature of panic disorder, and some theorists have argued that the effect of hyperventilation on body CO_2 levels is a causal factor in the development of a panic attack.
- Individuals with panic disorder have high levels of **anxiety sensitivity**, which is a fear of anxiety symptoms.
- Individuals who develop panic disorder tend to **catastrophically misinterpret** bodily sensations and interpret them as signs of an imminent physical threat (e.g. an imminent heart attack signalled by a missed heartbeat). This cognitive bias leads to a vicious cycle which increases the anxiety symptoms that precipitate a panic attack.
- **Tricyclic antidepressants** and **benzodiazepines** are an effective first line treatment for panic disorder, but structured **exposure therapy** or **cognitive behaviour therapy** (CBT) is as effective, if not superior, to drug treatments over the longer term.

5.4 GENERALIZED ANXIETY DISORDER (GAD)

Generalised anxiety disorder (GAD)
A pervasive condition in which the sufferer experiences continual apprehension and anxiety about future events, which leads to chronic and pathological worrying about those events.

Generalized anxiety disorder (GAD) is a pervasive condition in which the sufferer experiences continual apprehension and anxiety about future events, which leads to chronic and pathological worrying about those events. We all worry about things to some degree – and, indeed, many people find it beneficial to think about how they might deal with challenging future events. However, worrying for the individual with GAD has a number of features that make it disabling and a source of extreme emotional discomfort. For example:

1. Worrying is a chronic and pathological activity that is directed not only to major life issues (e.g. health, finances, relationships, work-related matters), but also to many minor day-to-day issues and hassles that others would not perceive as threatening (Craske, Rapee, Jackel & Barlow, 1989; Tallis, Davey & Capuzzo, 1994).
2. Worrying is perceived as uncontrollable – individuals with GAD feel they cannot control either the onset or termination of a worry bout.

Table 5.9 *DSM-IV-TR criteria for diagnosing generalized anxiety disorder (GAD)*

A Excessive anxiety and worry (apprehensive expectation) occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

B The person finds it difficult to control the worry.

C The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more days than not for the past 6 months):

(1) restlessness or feeling keyed up or on edge
(2) being easily fatigued
(3) difficulty concentrating or mind going blank
(4) irritability
(5) muscle tension
(6) sleep disturbance

D The focus of the anxiety or worry is not confined to features of an Axis I disorder, e.g. the anxiety or worry is not about symptoms of other mental or physical disorders (e.g. having a panic attack in panic disorder).

E The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F The disturbance is not due to the direct physiological effects of a substance or a general medical condition, and does not occur exclusively during a mood disorder, a psychotic disorder, or a pervasive developmental disorder.

Table 5.10 *Catastrophizing in worriers and non-worriers*

These catastrophizing sequences generated by a chronic worrier (top) and a non-worrier (bottom) were produced using the catastrophic interview procedure. The individual is first asked, 'What is your main worry at the moment?' In this case both participants replied, 'Getting good grades in school'. The interviewer then passes this response back to the participant by saying, 'What is it that worries you about getting good grades in school?' Each time the participant responds, the interviewer passes the response back by asking what it is about the response that worries the participant. The interview continues until the participant can no longer think of any reasons.

By looking at the catastrophizing sequences below, we can deduce a number of things about chronic worriers: (1) they produce significantly more catastrophizing steps than non-worriers; (2) they experience increasing emotional distress as catastrophizing continues, as evidenced by their 'discomfort' scores; and (3) the content of their catastrophizing steps becomes more and more threatening and catastrophic, as evidenced by their increasing 'likelihood' scores as catastrophizing progresses.

Table 5.10 *(Cont'd)*

CHRONIC WORRIER

Topic: Getting good grades in school

	Discomfort	likelihood
Catastrophizing step		
I won't live up to my expectations.	50	30
I'd be disappointed in myself.	60	100
I'd lose my self-confidence.	70	50
My loss of self-confidence would spread to other areas of my life.	70	50
I wouldn't have as much control as I'd like.	75	80
I'd be afraid of facing the unknown.	75	100
I'd become very anxious.	75	100
Anxiety would lead to further loss of self-confidence.	75	80
I wouldn't get my confidence back.	75	50
I'd feel like I wouldn't have any control over my life.	75	80
I'd be susceptible to things that normally wouldn't bother me.	75	80
I'd become more and more anxious.	80	80
I'd have no control at all and I'd become mentally ill.	85	30
I'd become dependent on drugs and therapy.	50	30
I'd always remain dependent on drugs.	85	50
They'd deteriorate my body.	85	100
I'd be in pain.	85	100
I'd die.	90	80
I'd end up in hell.	95	80

NON-WORRIER

Topic: Getting good grades in school

Catastrophizing step		
I might do poorly on a test.	3	20
I'd get a bad grade in the class.	3	100
That would lower my grade-point average.	2	100
I'd have less of a chance of getting a good job.	2	60
I'd end up in a bad job.	2	80
I'd get a low salary.		
I'd have less money to spend on what I want.	2	100
I'd be unhappy.	2	100
It would be a strain on me.	2	35
I'd worry more.	2	10
	2	5

Source: Vasey & Borkovec (1992).

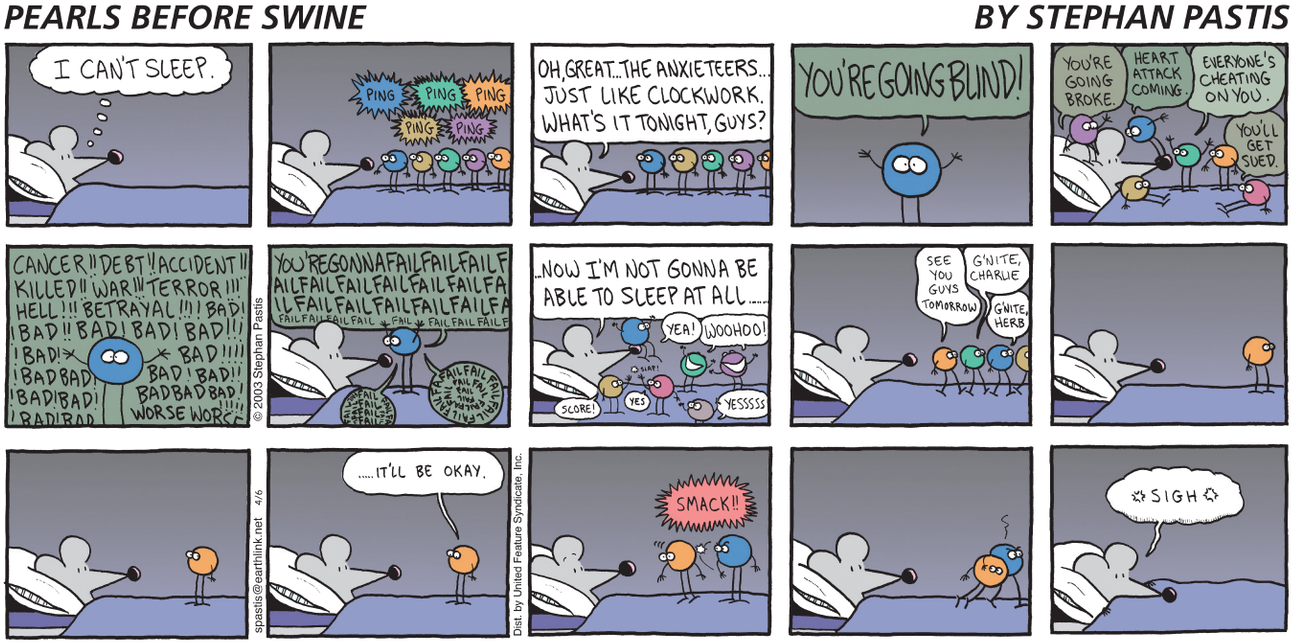


Plate 5.4

Catastrophizing An example of magnification, in which the individual takes a single fact to its extreme (e.g. a scratch on a new car means the car is wrecked and needs replacing).

3 Worrying is closely associated with the ***catastrophizing*** of worries – that is, worry bouts persist for longer in GAD, they are associated with increasing levels of anxiety and distress as the bout continues, and worrying seems to make the problem *worse* rather than better (see Table 5.10). While pathological and chronic worrying is the cardinal diagnostic feature of GAD, it may also be accompanied by physical symptoms such as fatigue, trembling, muscle tension, headache and nausea.

GAD is diagnosed if:

- excessive anxiety and worry occur more days than not for a period of at least 6 months;
- the individual reports difficulty in controlling the worry;
- worry is accompanied by at least three additional symptoms from a list including irritability, muscle tension, being easily fatigued, difficulty concentrating, restlessness and disturbed sleep;
- finally, a consequence of these symptoms will be clinically significant distress or impairment in social, occupational or other important areas of functioning. (See Table 5.9.)

GAD is twice as common in women as in men, and can often persist from adolescence to old age (Barlow, Blanchard, Vermilyea, Vermilyea & DiNardo, 1986). Over 5 per cent of the population will be diagnosed with GAD at some point in their lifetime (Wittchen & Hoyer, 2001), and over 12 per cent of those who attend anxiety disorder clinics will present with symptoms typical of GAD (Kessler, Keller & Wittchen, 2001). GAD is also highly comorbid with a range of other anxiety disorders and with depression (Brown, O'Leary & Barlow, 2001).

5.4.1 The Aetiology of Generalized Anxiety Disorder (GAD)

The challenge in explaining GAD is to understand why some individuals worry chronically and pathologically, while many other individuals – often with more stressful lifestyles – worry significantly less. Theories will therefore have to explain why GAD sufferers persist with their worrying even when it causes them significant distress.

5.4.1.1 Biological Theories

There is some evidence for a genetic component in both anxiety generally and GAD specifically (Noyes, Woodman, Garvey, Cook et al., 1992), which suggests that GAD has an inherited component. However, although there is a familial component to GAD, the evidence of a specific genetic component is modest (Hettema, Neale & Kendler, 2001), and it is doubtful that the claim of GAD sufferers that they are 'born worriers' is defensible. Given this, the main theories of GAD have focused on psychological and cognitive features of the pathological worrier.

Information Processing Biases in GAD A good deal of research has now indicated that anxious individuals, and especially

Information processing biases Biases in interpreting, attending to, storing or recalling information which may give rise to dysfunctional thinking and behaving.

those suffering GAD, have a series of ***information processing biases*** which appear to maintain their hypervigilance for threat, create further sources for worry and maintain anxiety. For example, experimental evidence has demonstrated that individuals with GAD preferentially allocate attention to threatening stimuli and threatening information (Mogg & Bradley, 1998; Mathews & MacLeod, 1994). These types of studies have indicated that:

- Preferential allocation of attention to threatening stimuli occurs pre-attentively (i.e. prior to the anxious individual becoming consciously aware of the threat) (Mogg, Bradley, Williams & Mathews, 1993; Mogg, Bradley & Halliwell, 1994). (See Figure 5.5.)
- Preferential allocation occurs to both verbal stimuli and to pictures of threatening emotional faces (Bradley, Mogg, Falla & Hamilton, 1998; Bradley, Mogg, Millar, Bonham-Carter et al., 1997).
- The bias towards attending to threatening stimuli in anxious individuals is mirrored by the tendency of non-anxious individuals to attentionally *avoid* threat, i.e. to shift attention *away from* threatening stimuli (Bradley, Mogg, White et al., 1999; Mogg & Bradley, 1998).

There is accumulating evidence that attentional biases to threat may actually *cause* anxiety (rather than being simply an outcome or consequence of being anxious). For example, studies that have attempted to experimentally induce information processing biases in non-clinical populations have suggested that attentional and interpretative biases for threat may indeed have a causal effect on experienced anxiety and the processing of future information (Wilson, MacLeod, Mathews & Rutherford, 2006). These studies use a computer-based task to train participants to choose the threatening interpretations of ambiguous statements (see Activity Box 5.2). They have shown that experimentally induced processing biases for threat will not only cause corresponding changes in state anxiety, but also tend the individual to interpret new information in a threatening way (Mathews & MacLeod, 2002; Hertel, Mathews, Peterson & Kintner, 2003; Mathews & Mackintosh, 2000). These attentional biases in GAD appear to be part of a wider set of cognitive biases in anxious individuals, all of which appear to

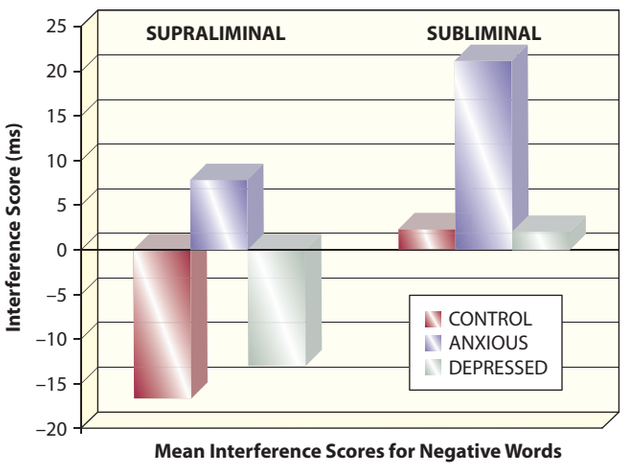


Figure 5.5
Anxious participants show the longest reaction times to name the colour of a threat word (the Emotional Stroop procedure – see Research Methods Box 5.2), suggesting that they have a tendency to prioritize the meaning of threat words. This is also the case when threat words are presented subliminally (right-hand figure), suggesting that this attentional bias occurs before the anxious individual is consciously aware of the meaning of the word.

Source: Mogg, Bradley, Williams & Mathews (1993).

ACTIVITY BOX 5.2

Interpretation biases and anxiety: The homophone spelling task

Homophones are words that sound the same but have different meanings. These types of words have been used to detect interpretation biases for threat in anxious individuals. For example, the homophones *Die/Dye* have different meanings, one of which is potentially negative or threatening. If anxious individuals are given auditory presentations of threat/neutral homophones and asked to spell the words they hear, they are more likely to write down the threatening rather than the neutral interpretation (Blanchette & Richards, 2003). This is evidence of a bias towards interpreting ambiguous information as threatening that is related to anxiety, and this bias can be found in most of the anxiety disorders.

Read out the following list of homophones to a fellow student (at the rate of about one every 2 seconds), and get him or her to spell the words as he or she hears them. How many does your participant spell in the threatening way? If your participant is anxious – perhaps because of an imminent exam – he or she is likely to respond with mainly threatening spellings.

Die/Dye
Pain/Pane
Patients/Patience
Mourn/Morn
Weak/Week
Bury/Berry
Groan/Grown
Flu/Flew
Slay/Sleigh
Tense/Tents
Tied/Tide
Ail/Ale
Wail/Whale
War/Wore
Flee/Flea

RESEARCH METHODS IN CLINICAL PSYCHOLOGY BOX 5.2

The Emotional Stroop Procedure

During the 1980s and 1990s, clinical psychology borrowed a number of very useful experimental techniques from cognitive psychology that allowed researchers to investigate some of the important cognitive processes involved in anxiety and depression.

The Emotional Stroop is one such example. This procedure allows researchers to determine whether individuals with anxiety or depression have a bias towards attending to and processing anxiety-relevant or depression-relevant information (e.g. Mogg, Bradley, Williams & Mathews, 1993) – a factor that may maintain their anxious state.

In this procedure, participants are presented with individual words in coloured ink. Some of the words are anxiety- (or depression-) relevant words, and others are emotionally neutral words. For example:

DEATH (an anxiety-relevant word)
or
CARPET (a neutral word)

Participants have to name the *colour of the ink* as quickly as possible, and their reaction time is recorded.

The implication of this procedure is that if individuals automatically attend to the meaning of threatening words (such as death), then this will delay their processing the colour and responding to it. So, if there is an attentional bias to anxiety-relevant words, then reaction times to name the *colour* of the word will be slower with anxiety-related words than with neutral, control words.

Most Emotional Stroop studies and their associated procedures do indicate that individuals suffering anxiety have an attentional bias towards anxiety-relevant words and stimuli (including anxiety-relevant faces; Mogg, Millar & Bradley, 2000), and that this attentional bias occurs pre-attentively (i.e. before the individual becomes consciously aware of the meaning of the word).

facilitate threatening interpretations of on-going and future events, focus attention on threat-related events and make this information processing bias resistant to change (Davey, 2006c).

Cognitions, Beliefs and the Function of Worrying We mentioned earlier that individuals with GAD persist chronically with their worrying even though it causes them considerable distress and generates symptoms that disrupt normal day-to-day living. This suggests that worrying may serve a particular *function* for such individuals, and this functionality may outweigh the negative effects of their worrying. Some theories of GAD emphasize this functional aspect of worrying. First, both pathological worriers and individuals diagnosed with GAD hold strong beliefs that worrying is a necessary process that must be undertaken fully and properly in order to avoid future catastrophes (Davey, Tallis & Capuzzo, 1996; Wells, 2006b; Borkovec, Hazlett-Stevens & Diaz, 1999). These dysfunctional beliefs about the utility of worrying appear to motivate worriers to persist with their worrying (see Table 5.11). Secondly, there is growing evidence that worrying may indeed be reinforced because it distracts the worrier from experiencing other negative emotions and processing even more stressful phobic images. That is, worry is an internal narrative process that prevents the individual from processing other – often more stressful – information (Borkovec, 1994; Borkovec & Lyonfields, 1993). Evidence to support this view comes from the fact that worry produces very little physiological or emotional arousal (Hoehn-Saric & McLeod, 1988) and appears to block the processing of emotional images (Borkovec, Lyonfields, Wiser & Diehl, 1993).

Dispositional Characteristics of Worrying While there is still some way to go in understanding the psychological and developmental processes that lead to individuals becoming pathological worriers, there is a good deal of knowledge available about what kinds of psychological features they possess. For example, worriers are intolerant of uncertainty (Ladouceur, Talbot & Dugas, 1997), are high on perfectionism (Pratt, Tallis & Eysenck, 1997) and have feelings of responsibility for negative outcomes (Startup & Davey, 2003; Wells & Papageorgiou, 1998). All of this suggests that they possess characteristics that will drive them to attempt to think about resolving problematic issues. However, worriers also have poor problem-solving confidence (Davey, 1994c) and couch their worries in ways that reflect personal inadequacies and insecurities (Davey & Levy, 1998). This contrasting combination of characteristics appears to drive individuals to try to resolve problems, but the process is thwarted by their personal doubt about their own ability to solve them successfully (Davey, 1994d).

5.4.2 The Treatment of Generalized Anxiety Disorder (GAD)

As with most of the anxiety disorders, GAD can be treated either with drugs or with structured psychological therapy such as CBT, or with a combination of both. However, deciding what type of treatment to use is often the most important decision for service

Table 5.11 *Dysfunctional beliefs in pathological worriers*

A number of studies indicate that pathological worriers and individuals with GAD possess a very strong and stable set of beliefs about worry as an important activity to indulge in – largely because they believe that if they do *not* worry, then bad things will happen to them.

The following are some of the positive beliefs that individuals diagnosed with GAD hold about worry as a necessary thing to do:

- 'Worry helps to motivate me to get things done that I need to get done.' (Motivation)
- 'Worrying is an effective way to problem solve.' (Problem solving)
- 'If I worry about something, when something bad does happen, I'll be better prepared for it.' (Preparation)
- 'If I worry about something, I am more likely to actually figure out how to avoid or prevent something bad from happening.' (Avoidance/Prevention)
- 'Worrying about most of the things I worry about is a way to distract myself from worrying about even more emotional things, things that I don't want to think about.' (Distraction from more emotional topics)
- 'Although it may not actually be true, it feels like if I worry about something, the worrying makes it less likely that something bad will happen.' (Superstition)

Individuals with GAD hold these beliefs more strongly than non-anxious individuals, and they drive the individual with GAD to worry chronically (Borkovec, Hazlett-Stevens & Diaz, 1999).

Source: Borkovec & Roemer (1995).

providers and therapists – especially for a disorder that is as prevalent as GAD. The UK National Institute for Clinical Excellence (NICE) has made some recommendations about how decisions should be made for treating GAD in primary care. These are outlined in Figure 5.6. Medication is recommended if immediate management of the problem is important (if the patient is experiencing extreme distress or has suicidal ideation). Otherwise, longer-term structured psychological therapy (such as CBT) or self-help programmes should be offered. This decision tree shows that comparisons of different types of therapy (e.g. drug vs. psychological therapy) are not just about their relative effectiveness over the long term, but also concern their value at various points in treatment, taking into account the patient's preferences and the severity of the symptoms.

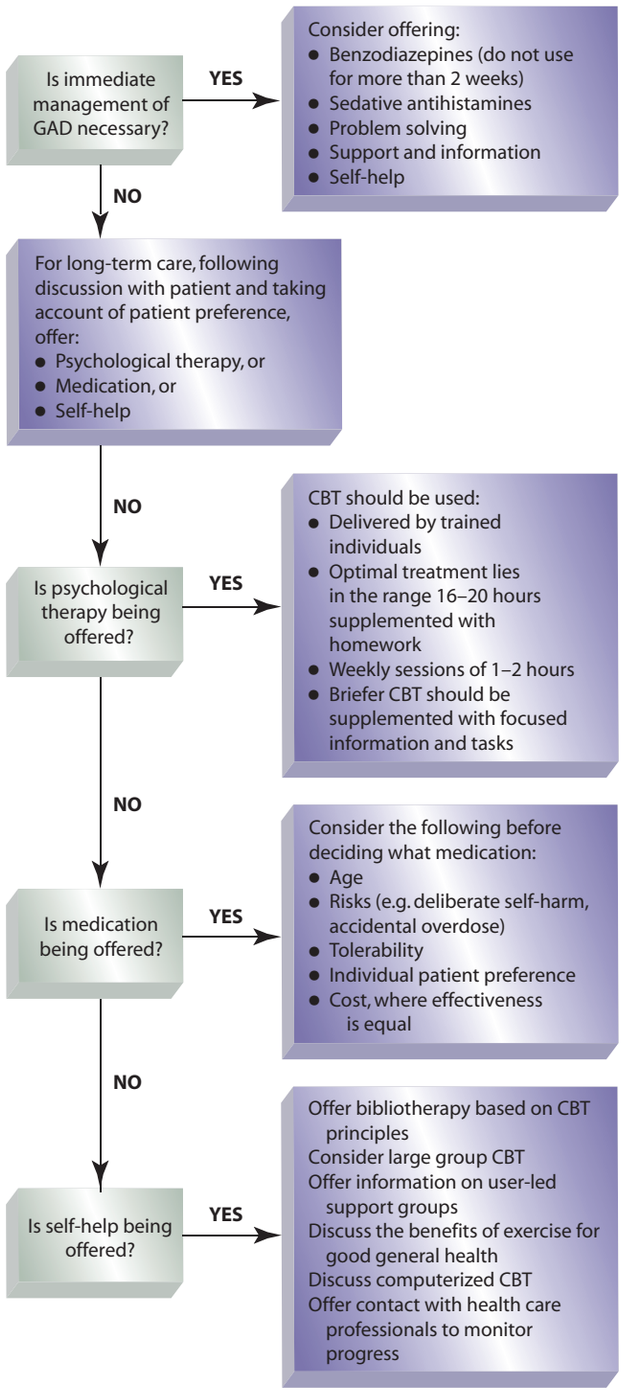


Figure 5.6
Primary care management of individuals with GAD.

5.4.2.1 Pharmacological Treatment

Because GAD involves chronic daily anxiety and emotional discomfort, anxiolytics – such as the benzodiazepines – are usually the first line of treatment for sufferers. Studies show that they are a more effective treatment for sufferers than placebo controls (Apter & Allen, 1999).

5.4.2.2 Stimulus Control Treatment

One of the earliest behavioural interventions for worry in GAD adopted the principle of stimulus control. This is based on the conditioning principle that the environments in which behaviours are enacted come to control the future occurrence of those

CLINICAL PERSPECTIVE: TREATMENT IN PRACTICE BOX 5.4

Stimulus control treatment for GAD

Stimulus control treatment for GAD is an effective treatment for reducing the frequency of worry by controlling the range of contexts in which the activity occurs (see section 5.4.2.2).

There are four basic instructions underpinning this procedure:

1 Learn to identify worrisome thoughts and other thoughts that are unnecessary or unpleasant. Distinguish these from necessary or pleasant thoughts related to the present moment.

2 Establish a half-hour worry period to take place at the same time and in the same location each day.

3 When you catch yourself worrying, postpone the worry to the worry period and replace it with attending to present-moment experience.

4 Make use of the half-hour worry period to worry about your concerns and to engage in problem solving to eliminate those concerns.

Source: Borkovec (2005); Borkovec, Wilkinson, Folensbee & Lerman (1983)

behaviours, and can act to elicit them (the principle of stimulus control). Because worrying can occur almost anywhere, and so come under the control of a vast range of contexts, the first aim of ***stimulus control treatment*** is to limit the contexts in which worrying occurs. This is achieved by telling clients that they can worry – but only for a specific period in a particular location each day (Borkovec, Wilkinson, Folensbee & Lerman, 1983). For example, they are instructed to worry at a specific time (e.g. between waking and the end of breakfast) or in a particular location (the living room).

Stimulus control treatment An early behavioural intervention for worry in GAD which adopted the principle of stimulus control. This is based on the conditioning principle that the environments in which behaviours are enacted come to control their future occurrence and can act to elicit those behaviours (the principle of stimulus control).

5.4.2.3 Cognitive Behaviour Therapy

In the previous section we reviewed a number of psychological theories of GAD which suggest that cognitive biases and dysfunctional beliefs about the function of worrying may be central to the development and maintenance of the disorder. This being so, integrated cognitive behavioural therapy seems a suitable method to tackle GAD. CBT for GAD normally consists of a number of elements, the main ones being:

- self-monitoring
- relaxation training
- cognitive restructuring
- behavioural rehearsal

Self-monitoring involves making clients aware of their fixed patterns of behaviour and the triggers that may precipitate worry. These triggers are often thoughts about future events that have very low probabilities of happening (e.g. the accidental death of a loved one while driving to work), and the client's attention is drawn to the fact that these are cognitively constructed rather than real events. ***Relaxation training*** is an obvious way of dealing with the chronic stress experienced by GAD sufferers. The specific technique of progressive muscular relaxation is often used (Bernstein, Borkovec & Hazlett-Stevens, 2000), and relaxation is found to be as effective as some forms of cognitive therapy (Arntz, 2003).

Self-monitoring A form of clinical observation which involves asking clients to observe and record their own behaviour, to note when certain behaviours or thoughts occur, and in what contexts they occur.

Relaxation training A method of dealing with the chronic stress experienced by psychopathology sufferers. A specific technique of progressive muscular relaxation is often used, and relaxation is found to be as effective as some forms of cognitive therapy.

Cognitive restructuring methods are used to challenge the biases that GAD sufferers hold about how frequently bad events might happen (Beck, Emery & Greenberg, 1985) and to generate thoughts that are more accurate (Borkovec, 2005). One way of doing this is by using an outcome diary in which clients write down on a daily basis their worries and how likely they think the focus of their worries will actually happen. Clients can then compare their own inflated estimate of the likelihood of the event with subsequent reality (Borkovec, Hazlett-Stevens & Diaz, 1999). Other types of cognitive restructuring involve the challenging and replacement of dysfunctional beliefs about the advantages of worrying (Wells, 1999) or the belief held by pathological worriers that uncertainty has to be resolved by thinking through every possible scenario (Dugas, Ladouceur, Leger, Freeston et al., 2003).

Cognitive restructuring Methods used to challenge the biases that a client might hold about how frequently bad events might happen and to generate thoughts that are more accurate.

Finally, ***behavioural rehearsal*** involves either the actual or imagined rehearsal of adaptive coping responses that need to be deployed when a worry trigger is encountered. These coping strategies may involve the deployment of relaxation

Behavioural rehearsal A coping strategy that involves either the actual or imagined rehearsal of adaptive coping responses that need to be deployed when a worry trigger is encountered.

SELF-TEST QUESTIONS

- What is the cardinal diagnostic feature of GAD?
- What are the features of worry in GAD that make it a distressing experience for the sufferer?
- How do information processing biases and cognitive factors contribute to the acquisition, maintenance and experience of anxiety in GAD?
- How do psychological treatments of GAD attempt to bring the activity of worrying under control?

exercises or pleasant distracting activities designed to avoid worry (Butler, Fennell, Robson & Gelder, 1991). CBT for GAD has been shown to be effective with or without the use of pharmacological treatments (Lang, 2004) and has long-term effectiveness for a significant proportion of clients (Durham, Chambers, MacDonald, Power & Major, 2003).

SECTION SUMMARY

5.4 Generalized Anxiety Disorder (GAD)

- The cardinal diagnostic characteristic of GAD is chronic uncontrollable worrying, which is accompanied by physical symptoms such as irritability, muscle tension, fatigue, poor concentration, restlessness and disturbed sleep.
- Over 5 per cent of the population will be diagnosed with GAD in their lifetime, and 12 per cent of those who attend anxiety disorder clinics will present with GAD.
- Individuals with GAD possess an **information processing bias** which appears to maintain their hypervigilance for threat and create the opportunity to catastrophically worry about events. There is evidence that these information processing biases may actually cause anxiety generally.
- Worrying in GAD appears to be maintained by **dysfunctional beliefs** about the utility of worrying which appear to motivate individuals with GAD to persist with their worrying.
- **Anxiolytics** are useful for dealing with the anxiety symptoms exhibited by individuals with GAD, but treatments based on controlling the process of worrying and challenging dysfunctional beliefs about worrying appear to have more longer-term benefit.

5.5 OBSESSIVE-COMPULSIVE DISORDER (OCD)

We have all occasionally gone back to check whether we locked a door or have experienced a sudden, intrusive thought that we find disturbing and out of place (e.g. harming our own child). However, for the person with ***obsessive-compulsive disorder (OCD)***, such thoughts and actions are repeated often and result in a distressing and disabling way of life (see Client's Perspective Box 5.2). OCD has two important and sometimes independent characteristics, obsessions and compulsions.

Obsessive-compulsive disorder (OCD) A disorder characterized either by obsessions (intrusive and recurring thoughts that the individual finds disturbing and uncontrollable) or by compulsions (ritualized behaviour patterns that the individual feels driven to perform in order to prevent some negative outcome happening).

Obsessions are intrusive and recurring thoughts that the individual finds disturbing and uncontrollable. These obsessive thoughts frequently take the form of causing some harm or distress to oneself or to some important other person (such as a partner or offspring). Common obsessions take the form of fear of contamination (i.e. contaminating oneself or important others), fear of directly or indirectly causing physical harm to others, and fears of expressing some immoral, sexual or aggressive impulse. Obsessive thoughts can also take the form of pathological doubting and indecision, and this may lead to sufferers developing repetitive behaviour patterns such as compulsive checking or washing.

Obsessions Intrusive and recurring thoughts that an individual finds disturbing and uncontrollable.

Compulsions represent repetitive or ritualized behaviour patterns that the individual feels driven to perform in order to prevent some negative outcome happening. This can take the form of ritualized and persistent checking of doors and windows (to ensure that the house is safe), or ritualized washing activities designed to prevent infection and contamination. Ritualized compulsions such as these also act to reduce the stress and anxiety caused by the sufferer's obsessive fears. Whilst the main compulsions are usually related to checking or washing, OCD can also manifest itself less regularly as compulsive hoarding (Steketee, Frost & Kyrios, 2003), superstitious ritualized movements or the systematic arranging of objects (Radomsky & Rachman, 2004). In most cases compulsions are clearly excessive, and are usually recognized as such by the sufferer. Rituals can become rigid, stereotyped sequences of behaviours which the individual is driven to perform as a result of cognitive triggers such as intrusive thoughts related to the individual's specific fears. For example, individuals distressed by unwanted immoral or blasphemous thoughts can attempt to suppress the thought and reduce anxiety by indulging in compulsive acts such as counting backwards from a number until the thought has gone.

Compulsions Repetitive or ritualized behaviour patterns that an individual feels driven to perform in order to prevent some negative outcome happening.

Table 5.12 shows the main DSM-IV-TR diagnostic criteria for OCD. Diagnosis is dependent on the obsessions or compulsions causing marked distress, being time consuming or significantly interfering with the person's normal daily living. This latter diagnostic criterion delineates OCD compulsions from other urges, such as the uncontrollable desire to eat, drink or gamble, because the latter are often engaged in with pleasure (see Chapters 8 and 9 for discussions of these alternative types of compulsions).

CLIENT'S PERSPECTIVE 5.2

Obsessive-compulsive disorder (OCD)

The following accounts describe the experiences of two individuals who have suffered different forms of obsessive-compulsive disorder. The first exhibits washing compulsions and superstitious avoidance rituals, while the second experiences persistent obsessive and intrusive thoughts that cause considerable distress.

'I first remember hand washing when I was about 4. My mother had died of cancer the year before – also my cousin and playmate, and my dog. I think that gave me some sense of having to DO something to ward off death. Then I had nose twitching for a while at about 11.

Then in school a guy I despised sneezed on me at 44 minutes after the hour and I thought I changed identity with him. Ever since I've had a superstitious avoidance of 44. It has popped up and frozen me in fear. Then I started to say my best friend's name like a mantra to offset anything bad. In the navy I didn't want to stand on the centerline of the ship. Thus began my avoidance of symmetry. It happens all day. I look like I've seen a ghost – the fear is so great.'

'Basically what I'm dealing with right now are these weird thoughts. I'll be sitting with someone, like my mum for instance, who I am very close to, and just think "I wish she would die, or I wish you would die" when I don't think I mean it. But then what happens is I start wondering if I really do mean it. Another thing, someone will say something horrible, like "I just found out my mum has cancer" and I'll immediately think "I wish my mum had cancer" when of course I don't wish that. Recently I had a huge breakdown. I started believing I actually wanted to harm my mother. I thought I was going to stab her with a knife and actually thought I might do it. At one point I even said to myself in my head, you should just do it. I got so scared I went straight to the doctor that day. This thought made me so sick I lost all this weight, couldn't sleep. All because I couldn't figure out how you know if you really want to kill someone. It sounds so crazy but I actually thought, how do I know if I really love her or if I don't? How do I know I won't just pick up a knife and stab her? I thought I had become a sociopath or something.'

Source: www.stuckinadoorway.co.uk/

Table 5.12 *The main DSM-IV-TR diagnostic criteria for obsessive-compulsive disorder*

Obsessions are defined as:

(1) Recurrent and persistent thoughts, impulses, or images that are experienced, at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress.

(2) The thoughts, impulses, or images are not simply excessive worries about real-life problems.

(3) The person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action.

(4) The person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion).

Compulsions are defined as:

(1) Repetitive behaviours (e.g. hand washing, ordering, checking) or mental acts (e.g. praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly.

(2) The behaviours or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviours or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive.

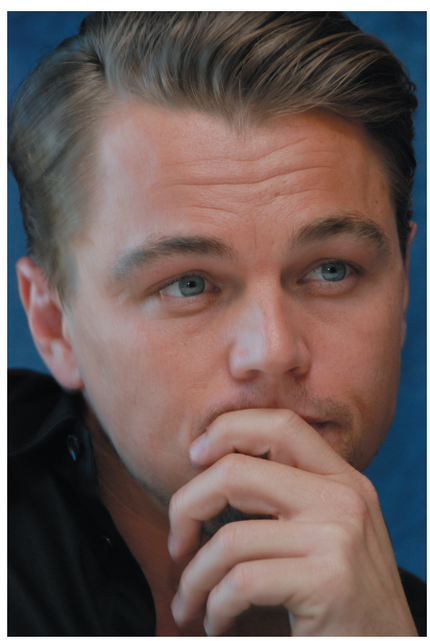


Plate 5.5
Film star Leonardo DiCaprio has revealed he suffers from obsessive-compulsive disorder. The *Titanic* star says he has to force himself not to step on every chewing gum stain when walking along and fight urges to walk through a doorway several times, because he doesn't want his condition taking over his life.

CASE HISTORY 5.2

Obsessive-compulsive disorder

Catherine was 28 years old. It was a week before her wedding. The alarm went off at 7.00 a.m. as usual. She turned to switch it off, and was immediately overwhelmed by the feeling of dread that had been her constant companion for the past 6 months.

She went to the bathroom and washed her hands. She stepped inside the shower and started to wash herself, washing her hands repeatedly between washing different parts of her body. Three-quarters of an hour later she emerged, gathered up her nightwear and towel and dumped them in the washing machine. Her hands felt dirty again so she returned to the bathroom to clean them. By now her hands were washed red raw so she rubbed in some moisturizer. They didn't feel as clean and she felt a strong urge to wash them once more. She looked at her reflection in the mirror and told herself not to be so stupid, that she would be late for work again if she didn't get a move on. But she couldn't resist the urge. She quickly washed her hands and hurried into the kitchen, kicking the door open to avoid contact with the germ-ridden doorknob.

After breakfast – and several more hand washes – she systematically went from room to room locking and checking all the doors and windows. Finally she stepped outside the house. By now she was already late for work but thought she'd better go back inside and check everything one more time, just to be sure. When this was done, she got into her car and drove down the street. Before she reached the end of the road she wondered if she had locked the front door properly. Back she went and checked the handle five more times.

She had almost reached her workplace when she was suddenly plagued by the idea that she might have accidentally hit a cyclist. She mentally retraced her steps. She remembered driving past a few cyclists but certainly didn't notice that she had hit any of them at the time. However, just to be on the safe side, she decided to drive round the block again. It came as no surprise when she didn't see any injured cyclists lying on the road and she cursed herself for giving in to such an irrational idea.

When she finally pulled up outside her place of work she looked at her watch and saw that, yet again, she was unacceptably late. Work started at 8.30 a.m. not 10.30 a.m.

Catherine was diagnosed as having obsessive-compulsive disorder in 1994. Her diagnosis came as a relief. At least now she knew she wasn't going crazy and that the behaviour that had inexplicably taken over her life had a name and was treatable. While her family was generally supportive she said the majority of people didn't understand. 'A lot of people around me were shocked but felt I should just get over it, they couldn't see any logical reason for the behaviour or the distress.'

Source: www.ocd.org.nz/personal_story.htm

Clinical Commentary

This example shows how obsessions and compulsions in OCD are often compelling and difficult for the sufferer to resist – even when the individual is aware that these thoughts and actions are 'stupid' or irrational. Catherine's compulsions are fuelled by the 'feelings of dread' that she experiences most mornings when she wakes up, and this provides the highly anxious state under which compulsions (such as compulsive washing) are performed. 'Doubting' is also a common feature of OCD, and Catherine experiences this more than once on her way to work (in the form of doubting whether the door is locked or whether she may have hit a cyclist on her way to work). The high levels of inflated responsibility usually possessed by OCD sufferers such as Catherine means that they are driven to continually check that their doubts are unfounded.

5.5.1 Prevalence

OCD onset is usually gradual and frequently begins to manifest itself in early adolescence or early adulthood following a stressful event such as pregnancy, childbirth, relationship or work problems (Kringlen, 1970). Lifetime prevalence is around 2.5 per cent, with a 1-year prevalence rate of 1.5–2.1 per cent, and women are marginally more frequently affected than men (Stein, Forde, Anderson et al., 1997). Few studies have investigated the effect of cultural factors on the prevalence and manifestation of OCD symptoms. However, a cross-cultural study reported by Fontenelle, Mendlowicz, Marques and Versiani (2004) concluded that universal characteristics of sufferers regardless of cultural background included a predominance of females, a relatively early age of onset and a preponderance of mixed obsessions and compulsions. The exception to this was the apparent content of obsessions, with Brazilian and Middle Eastern samples exhibiting a predominance of aggressive or religious obsessions (compared with North America, Europe and Africa).

5.5.2 The Aetiology of Obsessive-Compulsive Disorder

When considering the aetiology of OCD, readers should be aware that it represents a psychological problem that possesses a number

of quite different, and often independent, features. For example, obsessions do not always occur with compulsions, and the two main types of compulsions (washing and checking) rarely occur together in the same individual. This means that many theories of the aetiology of OCD have been developed to address only some of its features (e.g. thought suppression accounts are relevant to explaining only obsessive thoughts) and are not meant to be universal explanations of OCD. Bear this in mind when reading through the following sections.

5.5.2.1 Biological Factors

Onset of OCD can be associated with traumatic brain injury or encephalitis (Jenike, 1986), which suggests that there may be a neurophysiological deficit in some forms of OCD. This neurophysiological deficit may give rise to the 'doubting' that things have been done properly that is a central feature of many forms of OCD. Areas of the brain that have been identified as important in this respect include the frontal lobes and the basal ganglia. When sufferers are shown stimuli representative of their obsession or compulsion (e.g. an unlocked door), blood flow increases in both the frontal lobes and the basal ganglia, suggesting that these areas may have at least some role in OCD (Rauch, Jenike, Alpert, Baer et al., 1994).

In other neuropsychological studies OCD sufferers appear to demonstrate a variety of basic information processing and executive functioning deficits (the latter refers to processes that are involved in planning and attentional control), including deficits in spatial working memory, spatial recognition, visual attention, visual memory and motor response initiation (Greisberg & McKay, 2003). However, while such executive deficits may contribute to 'doubting' that something has been done properly, they do not necessarily indicate that neurophysiological deficits play a causal role in OCD.

Traditional neuropsychological studies have tended to rely on intensive study of individuals with known brain lesions and investigated the effect of these lesions on executive functions related to OCD. However, because such lesions may result in symptoms similar to those found in OCD, it does not mean that all OCD sufferers possess such lesions. An alternative view of brain dysfunction and OCD is based on findings from neuroimaging studies. For example, Rapoport (1989) has argued that obsessions and compulsions are genetically stored and learned behaviours that are involuntarily triggered by the brain. Baxter, Ackerman, Swerdlow, Brody et al. (2000) have developed this approach by suggesting that uncontrollable compulsions in OCD result from the brain being unable to inhibit these genetically stored behaviours. In particular, they use evidence from neuroimaging studies to argue that OCD compulsions result from the failure of inhibitory pathways projecting via the basal ganglia to inhibit innate behaviour patterns (Saxena, Brody, Schwartz et al., 1998). While this hypothesis can account for the rather restricted set of behaviours that manifest as compulsions (i.e. it is argued that only certain behaviours have been genetically stored), it still represents an oversimplification of how brain structures such as the basal ganglia might be involved in OCD (Frampton, 2003).

5.5.2.2 Psychological Factors

Memory Deficits 'Doubting' is a central feature of OCD, and especially the compulsions associated with the disorder. As a result, it has been suggested that OCD may be characterized by memory deficits that give rise to the doubting that, for example, doors have been locked or hands have been washed properly. Memory deficit models take a number of different forms. It has been suggested that OCD sufferers may have:

- a general memory deficit (Sher, Mann & Frost, 1984);
- less confidence in the validity of their memories (Sheffler-Rubenstein, Peynircioglu, Chambless & Pigott, 1993); or
- a deficit in the ability to distinguish between the memory of real and imagined actions (Brown, Kosslyn, Breiter, Baer & Jenike, 1994).

However, while OCD sufferers do claim to have doubts about their recollection of having either checked or washed properly, recent evidence suggests that lack of confidence in recall may be a *consequence* of compulsive checking or washing rather than a cause of it (van den Hout & Kindt, 2003; Tolin et al., 2001). In effect, the more someone checks, the less confident that person will be about what he or she has checked.

Inflated Responsibility Everyone experiences uncontrollable intrusive thoughts on almost a daily basis (Rachman & DeSilva, 1978). However, what differentiates these normal intrusive thoughts from the distressing and obsessive thoughts experienced in OCD is the meaning attached to them by OCD sufferers. Individuals diagnosed with OCD appear to have developed a series of dysfunctional beliefs about their obsessional thoughts. For example:

1. Because they had the thought, they feel responsible for its content – so, if a sufferer thinks of murdering his child, he believes he may be going crazy and *will* murder his child (Salkovskis, 1985).
2. Sufferers appraise obsessional thoughts as having potentially harmful consequences. This causes intense anxiety and triggers compulsive actions designed to eradicate the thought or to make sure that the perceived harm does not occur (e.g. compulsive thought suppression strategies such as counting backwards or checking and rechecking locks and windows to ensure that the home is safe).
3. Individuals with OCD tend to have inflated conceptions of their own responsibility for preventing harm. This ***inflated responsibility*** appears to be an important vulnerability factor in developing OCD (Salkovkis, 1985; Rachman, 1998).

Inflated responsibility The belief that one has power to bring about or prevent subjectively crucial negative outcomes. These outcomes are perceived as essential to prevent. They may be actual, that is, having consequences in the real world, and/or at a moral level.

Salkovskis, Rachman, Ladouceur, Freeston et al. (1996) have defined inflated responsibility as 'the belief that one has power which is pivotal to bring about or prevent subjectively crucial

Table 5.13 *Inflated responsibility and the Responsibility Attitude Scale*

Inflated responsibility is a central characteristic of individuals diagnosed with OCD and appears to be an important precipitating factor in the disorder. Below are some items from the Responsibility Attitude Scale (RAS) (Salkovskis et al., 2000) designed to measure responsibility-related beliefs as they apply to OCD. This will give you some idea of the beliefs that make up the construct of inflated responsibility.

1. I often feel responsible for things that go wrong.
2. If I don't act when I can foresee danger, then I am to blame for any consequences if it happens.
3. If I think bad things, this is as bad as DOING bad things.
4. I worry a great deal about the effects of things that I do or don't do.
5. To me, not acting to prevent disaster is as bad as making disasters happen.
6. I must always think through the consequences of even the smallest actions.
7. I often take responsibility for things that other people do not think are my fault.
8. Everything I do can cause serious problems.
9. I must protect others from harm.
10. If I can have even a slight influence on things going wrong, then I must act to prevent it.
11. For me, even slight carelessness is inexcusable when it might affect others.
12. Even if harm is a very unlikely possibility, I should always try to prevent it at any cost.
13. I have to make sure other people are protected from all of the consequences of things I do.
14. If I take sufficient care, then I can prevent any harmful accidents.
15. I often think that bad things will happen if I am not careful enough.

negative outcomes. These outcomes are perceived as essential to prevent. They may be actual, that is having consequences in the real world, and/or at a moral level.' (See Table 5.13.) There is considerable evidence that inflated responsibility is a characteristic that is a central causal feature of obsessive-compulsive disorder generally (Salkovskis, Shafran, Rachman & Freeston, 1999; Salkovskis, Wroe, Gledhill, Morrison et al., 2000) and compulsive checking specifically (Rachman, 2002; Bouchard, Rheaume & Ladouceur, 1999). Experimental studies that have manipulated inflated responsibility have shown that it *causes* increases in perseverative activities such as compulsive checking (Lopatka & Rachman, 1995; Bouchard, Rheaume & Ladouceur, 1999).

Figure 5.7 shows a schematic representation of a cognitive behavioural model of OCD which incorporates both the misinterpretation of the significance of intrusions by sufferers and their

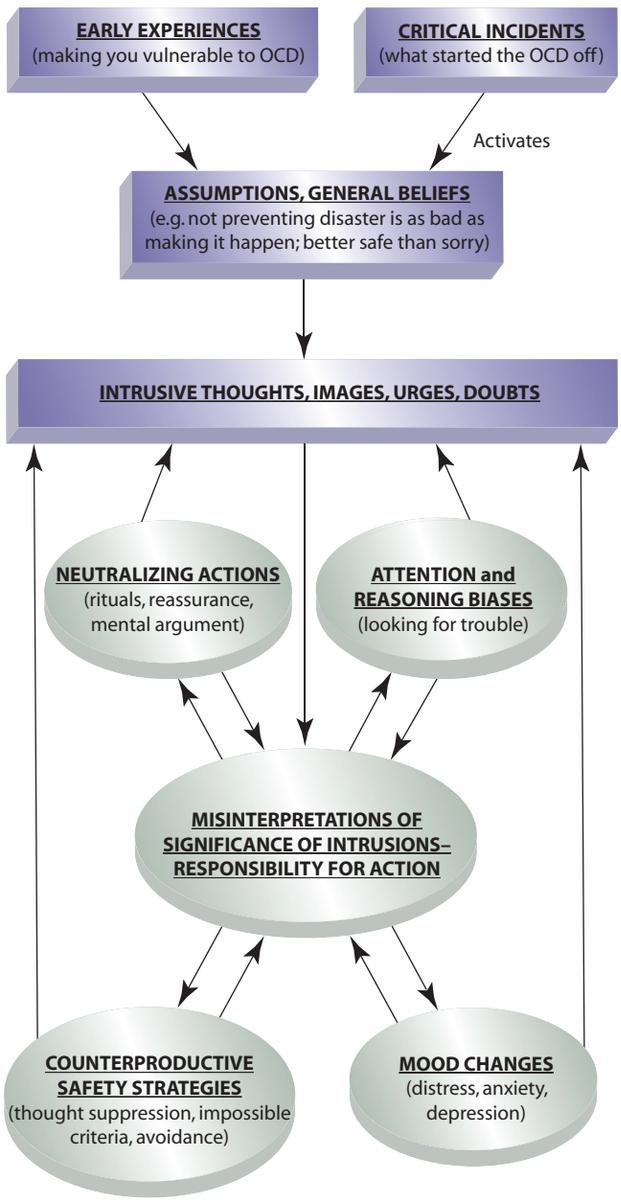


Figure 5.7 *A cognitive model of OCD proposed by Salkovskis, Wroe, Gledhill, Morrison et al. (2000).*
This model highlights the importance of a number of factors in maintaining obsessive-compulsive symptoms. These include (1) misinterpreting the importance and meaning of intrusive thoughts, (2) inflated responsibility for negative outcomes and (3) the role of rituals and neutralizing actions in maintaining obsessive thoughts and compulsive actions.

perceived responsibility for any negative or harmful outcomes (Salkovskis, Shafran, Rachman & Freeston, 1999). This model assumes that the dysfunctional beliefs that characterize OCD patients are learned over long periods from childhood onwards, and may be formed as a result of extreme events or circumstances. The dysfunctional assumptions held by OCD sufferers include the following:

Thought-action fusion A dysfunctional assumption held by OCD sufferers that having a thought about an action is like performing it.

1 having a thought about an action is like performing it (this is known as ***thought–action fusion***; Shafran & Rachman, 2004);

2 failing to prevent harm to oneself or others is the same as having caused the harm in the first place;

3 responsibility is not reduced by other factors such as something being improbable;

4 not trying to neutralize or suppress an intrusive thought is equivalent to wanting the harm involved in the thought to happen; and

5 one should always try to exercise control over one's thoughts (Salkovskis & McGuire, 2003).

The fact that such dysfunctional beliefs are held by OCD sufferers and that they appear to contribute significantly to the symptoms of the disorder makes them a promising target for cognitive behavioural treatment interventions (see below).

Thought Suppression Because individuals with obsessive thoughts find these intrusions aversive and distressing, they may try to actively suppress them (using either ***thought suppression*** or distraction techniques). However, there is good evidence that actively suppressing an unwanted thought will actually cause it to occur more frequently once the period of suppression or inhibition is over (known as a 'rebound' effect; see Figure 5.8). This may account to some degree for the fact that OCD sufferers experience significantly more intrusions than non-clinical populations (Wenzlaff & Wegner, 2000). Wenzlaff, Klein and Wegner (1991) have also argued that suppressing an unpleasant thought induces a strong negative emotional state that results in the suppressed thought becoming associated with that negative mood state. Whenever that negative mood state occurs in the future, it is therefore more likely to elicit the unwanted and aversive thought, and this may also contribute to the OCD sufferer's experiencing regular, uncontrollable intrusions.

Thought suppression A defence mechanism used by individuals with obsessive thoughts to actively suppress them (using either thought suppression or distraction techniques).

Perseveration and the Role of Mood OCD is one example of a number of perseverative psychopathologies, each of which is characterized by the dysfunctional perseveration of certain thoughts, behaviours or activities (others include pathological worrying and chronic rumination in depression). In almost all examples of these psychopathologies, the perseveration (e.g. compulsive checking, washing) is viewed as excessive, out of proportion to the functional purpose that it serves, and a source of emotional discomfort for the individual concerned. In this context, some theories have attempted to explain why OCD sufferers *persevere* at an activity for significantly longer than non-sufferers. One such account is the ***mood-as-input hypothesis*** (Martin & Davies, 1998; Davey, 2006b; MacDonald & Davey, 2005a). This model states that OCD sufferers persevere with their compulsive activities because (1) they use an implicit 'stop rule' for the compulsive activity which says they must only stop when they are sure they have completed the task fully and properly (known as an 'as many as can' stop rule); and (2) they undertake the task in a strong negative mood (usually an anxious mood). The mood-as-input account claims that OCD sufferers use their concurrent mood as 'information' to assess whether they have met their strict stop rule criteria. However, their endemic negative mood is interpreted as providing information that they have *not* completed the task properly – so they persevere (i.e. a negative mood implies all is not well and the criteria have not been met). This model is supported by the fact that the inflated responsibility that OCD sufferers possess is likely to give rise to deployed 'as many as can' stop rules (to ensure that, for example, checking or washing is done properly so that no harm will ensue) (Startup & Davey, 2003). Interestingly, and consistent with the mood-as-input account, inflated responsibility is not a *sufficient* condition for an individual to persevere at a compulsive activity – it has to be accompanied by negative mood (MacDonald & Davey, 2005b). This is because a negative mood is continually being interpreted as providing feedback that the important goals of the compulsive activity have not been met, so the activity needs to be continued.

Mood-as-input hypothesis A hypothesis claiming that people use their concurrent mood as information about whether they have successfully completed a task or not.

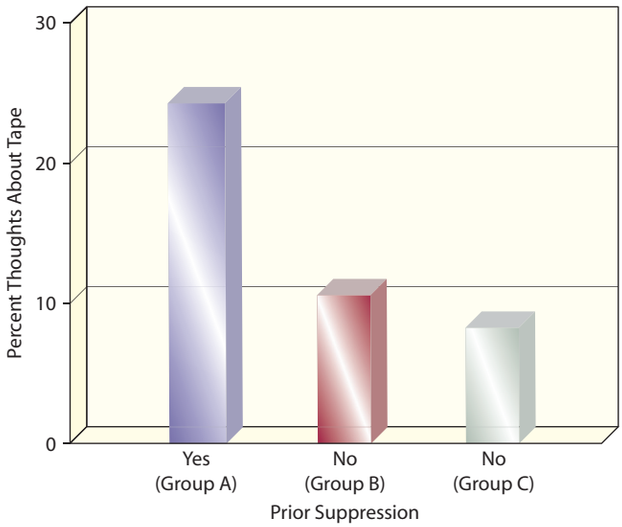


Figure 5.8 *The 'rebound' effect.*
After listening to a tape of a recorded story, participants were asked to verbalize their stream of conscious. However, participants in one group (Group A) were asked to suppress their thoughts of the tape during this period, while other participants were not (Groups B and C). In the final period, all participants were asked to think about anything. The figure shows that participants in the suppression condition subsequently reported more thoughts about the tape than participants in the other groups (a 'rebound' effect).
Source: Clark, Ball & Pape (1991).

SUMMARY

As we mentioned at the outset of this section on aetiology, many of these theories are designed to address only specific features of OCD rather than represent universal explanations of the disorder. For example, some theories try to explain why 'doubting' is a central feature of OCD (these include both neurophysiological and memory deficit models), others address why intrusive thoughts become so aversive and uncontrollable (e.g. inflated responsibility and thought suppression accounts), and yet others try to explain why individuals with OCD show dysfunctional perseveration at activities such as checking or washing (e.g. the mood-as-input model). Undoubtedly, a full account of OCD will contain at least some, if not all, of these different elements of explanation.

5.5.3 The Treatment of Obsessive-Compulsive Disorder

5.5.3.1 Exposure and Ritual Prevention Treatments (EPR)

The most common, and arguably the most successful, treatment for OCD is ***exposure and ritual prevention*** (ERP, also known as exposure and response prevention) (Meyer, 1966; Kyrios, 2003). This therapy consists of two components. The first is graded exposure to the situations and thoughts that trigger distress – for example, for someone with compulsive washing, this may involve touching a dirty dish or imagining touching a dirty dish (the latter is called imaginal exposure). Clients will encounter their triggers in a graded and planned way until distress levels have significantly decreased. The second component is ritual or response prevention, which involves strategies such as practising competing behaviours, habit reversal or modification of compulsive rituals (see Treatment in Practice Box 5.5). Preventing clients from engaging in their rituals:

Exposure and ritual prevention treatments A means of treatment for obsessive-compulsive disorder (OCD) which involves graded exposure to the thoughts that trigger distress, followed by the development of behaviours designed to prevent the individual's compulsive rituals.

1. allows anxiety to extinguish by habituating the links between obsessions and their associated distress;
2. eliminates ritualistic behaviours that may negatively reinforce anxiety (Steketee, 1993);
3. contributes to the disconfirmation of dysfunctional beliefs (e.g. 'I will catch an infectious disease if I touch a dirty cup') by forcing clients to encounter feared situations and experiencing the reality of the outcomes associated with that action.

ERP is a highly flexible therapy that can be adapted to group, self-help, inpatient, outpatient, family therapy and computer-guided interventions (Fischer, Himle & Hanna, 1998; Grayson, 1999; Wetzel, Bents & Florin, 1999; Hand, 1998; Nakagawa, Marks, Park, Bachofen et al., 2000). Controlled outcome studies suggest that ERP is a long-term effective treatment for around 75 per cent of clients treated with ERP (Franklin & Foa, 1998; Kyrios, 2003).

5.5.3.2 Cognitive Behaviour Therapy (CBT)

Although ERP has been the treatment of choice for OCD for over 20 years, it is often a difficult treatment for many sufferers to enter. This is because sufferers may feel unable to expose themselves to their fear triggers and find it impossible to prevent themselves acting out their rituals. As many as 30 per cent of clients drop out of ERP before completing treatment (Wilhelm, 2000). An alternative form of therapy for such individuals is cognitive behaviour therapy (CBT), based on targeting and modifying the dysfunctional beliefs that OCD sufferers hold about their fears, thoughts and the significance of their rituals (Abramowitz, Brigidi & Roche, 2001; Salkovskis, 1999; Wilhelm, 2000; Marks, 2003; see section 5.5.2.2, inflated responsibility). Dysfunctional beliefs that are usually challenged in cognitive therapy for OCD include:

- *responsibility appraisals*, where sufferers believe they are solely responsible for preventing any harmful outcomes;
- the *over-importance of thoughts*, where sufferers believe that having a thought about an action is like performing the action (thought–action fusion) (see Figure 5.7);
- *exaggerated perception of threat*, where sufferers have highly inflated estimates of the likelihood of harmful outcomes (van Oppen & Arntz, 1994).

An integrated cognitive therapy for OCD would thus consist of:

- educating clients that intrusive thoughts are quite normal, and that having a thought about an action is not the same as performing it (Salkovskis, 1999);
- focusing on changing clients' abnormal risk assessment – perhaps by working through the probabilities associated with feared outcomes (van Oppen & Arntz, 1994);
- providing clients with behavioural exercises that will disconfirm their dysfunctional beliefs (e.g. a client who fears shouting out blasphemous thoughts in church would be asked to go to church and see if this happens) (Salkovskis, 1999).

5.5.3.3 Pharmacological and Neurosurgical Treatments

Pharmacological treatments have proved to be a short-term effective and cheap way of treating OCD, although relapse tends to be common on discontinuation of the drug treatment (McDonough, 2003; Pato, Zohar-Kadouch, Zohar & Murphy, 1988). Serotonin and serotonin reuptake inhibitors (SSRIs) are the most commonly prescribed drugs and have the effect of increasing

CLINICAL PERSPECTIVE: TREATMENT IN PRACTICE BOX 5.5

Exposure hierarchies and response prevention in ERP treatments of OCD

Arguably the most effective therapies for OCD are exposure and ritual prevention treatments (ERP) (see section 5.5.3.1). Table 1 gives examples of a graded exposure regime for fear of contamination from germs and distressing thoughts about sexual abuse. Table 2 provides some examples of response prevention techniques.

Table 1

Example 1 *Fear of contamination* (distress level/100)	*Example 2* *Teacher's distressing intrusive thoughts about sexually abusing students* (distress level/100)
1 Touch rim of own unwashed coffee cup. (30)	1 Watch video or listen to audio tape of expert discussing sexual abuse of children. (40)
2 Touch rim of partner's unwashed coffee cup. (40)	2 Listen to tape of expert while looking at class photo. (50)
3 Eat snack from dish in cupboard after touching partner's unwashed coffee cup. (45)	3 Listen to loop tape of own distressing thoughts about sexually abusing students in general. (60)
4 Drink water from partner's glass. (55)	4 Listen to loop tape about students in general looking at class photo. (65)
5 Eat snack straight from unwashed table top. (65)	5 Listen to loop tape of distressing thought about sexually abusing specific student. (70)
6 Have coffee at a café. (70)	6 Listen to loop tape about specific student looking at class photo. (75)
7 Have meal at a restaurant. (80)	7 Listen to loop tape holding specific student's homework. (80)
8 Touch toilet seat at home without washing hands for 15 mins. (85)	8 Stand in front of class repeating statement on loop tape to self. (90)
9 Touch toilet seat at home without washing hands for 30 mins. (90)	9 Stand close to specific student repeating statement on loop tape to self. (95)
10 Use public toilet. (100)	10 Stand next to specific student repeating statement on loop tape to self. (100)

Table 2

OCD Symptom	*Response Prevention Strategy*
Hand washing or cleaning rituals	Response delay (i.e. extending period between 'contamination' and cleaning or washing); use of ritual restrictions (e.g. decreasing cleaning or washing time); clenching fists; extension strategies to undermine avoidance (e.g. touch self, clothes)
Checking lights, switches, oven, appliances, etc.	Response delay; use of ritual restrictions (e.g. restrict number of checks); turning and walking away; extension strategies (whistle a happy tune)
Counting (e.g. bricks, words)	Refocusing techniques; singing a song; going 'blank'; meditation

Source: Kyrios (2003)

brain serotonin levels (see Chapter 4, section 4.1.1.6). However, there is still no consensus view on a model of serotonin dysfunction in OCD (Delgado & Moreno, 1998), and it has been suggested that the beneficial effects of SSRIs may be restricted simply to their non-specific ameliorative effect on dysfunctional brain circuits. Tricyclic antidepressants can have beneficial effects across some specific symptoms of OCD (such as reducing the persistence and frequency of compulsive rituals), but seem to have their effect only when OCD is comorbid with depression (Hohagen, Winkelmann, Rasche-Rauchle, Hand et al., 1998). Comparative studies have suggested that both SSRIs and tricyclic drugs are less effective than standard psychological therapies such as exposure and ritual prevention (ERP) (Greist, 1998). In general, ERP is equally as effective as drug treatments in the short term, free from physical and psychological side effects, and associated with greater long-term gains (Greist, 1998; Marks, 1997). When pharmacological and psychological treatments have failed, neurosurgery has become an intervention of last resort in OCD. The most common procedure is ***cingulatomy***, which involves destroying cells in the cingulum, close to the corpus callosum. These treatments do report some improvement in OCD symptoms (Dougherty, Baer, Cosgrove, Cassem et al., 2002), but there is a lack of evidence on the longer-term gains of neurosurgical treatments and their possible side effects (McDonough, 2003).

Cingulatomy A neurosurgical treatment involving destroying cells in the cingulum, close to the corpus callosum.

SELF-TEST QUESTIONS

- Can you describe what *obsessions* and *compulsions* are, and provide some examples of each?
- How have biological theories attempted to explain the obsessions and compulsions found in OCD?
- How does the construct of 'inflated responsibility' help to explain how OCD is acquired and maintained?
- What are the similarities and differences between exposure and ritual prevention treatment (EPR) and cognitive behaviour therapy (CBT) for OCD?

SECTION SUMMARY

5.5 Obsessive-Compulsive Disorder (OCD)

- OCD is characterized by either **obsessions** – which are intrusive and recurring thoughts that the individual finds disturbing and uncontrollable – or **compulsions** – which are ritualized behaviour patterns that the individual feels driven to perform in order to prevent some negative outcome happening.
- Common compulsions include washing, checking and, to a lesser degree, hoarding and the systematic arrangement of objects.
- OCD onset is usually gradual and has a lifetime prevalence rate of around 2.5 per cent.
- **Biological theories** of OCD argue that there may be neurological deficits underlying OCD which either give rise to the typical 'doubting' behaviour common in the disorder, or result in an inability to inhibit certain behaviour patterns (such as checking).
- OCD tends to be associated with a number of **dysfunctional beliefs**. The most prominent is sufferers' inflated conception of their own responsibility for preventing harm, and this **inflated responsibility** appears to be an important vulnerability factor in developing OCD.
- **Exposure and ritual prevention** (EPR) treatments are the most common, and arguably the most successful, means of treatment for OCD. These involve graded exposure to the thoughts that trigger distress, followed by the development of behaviours designed to prevent the individual's compulsive ritual.
- **Pharmacological treatments** for OCD can also be effective (e.g. serotonin and serotonin reuptake inhibitors), and psychosurgery is sometimes a treatment of last resort (e.g. **cingulatomy**).

5.6 POST-TRAUMATIC STRESS DISORDER (PTSD)

Post-traumatic stress disorder (PTSD) was not recognized as a specific category of psychopathology until 1980, when it was included for the first time in DSM-III. PTSD is somewhat different from the other anxiety-based disorders because its definition and diagnosis include identifying exposure to a specific fear-evoking event as a cause of the symptoms (see Table 5.14). PTSD is a set of persistent symptoms that occur after experiencing or witnessing an extremely fear-evoking traumatic event (see Client's Perspective 5.3). Such events include combat during a war, rape or other types of physical assault, child abuse, car or aeroplane crashes, or natural or human-caused disasters. The symptoms of PTSD are also somewhat different from those experienced in other anxiety disorders, and can be grouped into three main categories:

Post-traumatic stress disorder (PTSD) A set of persistent anxiety-based symptoms that occur after experiencing or witnessing an extremely fear-evoking traumatic event.

- *increased arousal*, which includes an exaggerated startle response (Shalev, Peri, Brandes, Freedman et al., 2000), difficulty sleeping, hypervigilance and difficulty concentrating;
- *avoidance and numbing of emotions*: the individual will attempt to avoid all situations or events that might trigger memories of the traumatic event, and there is a sense of detachment and lack of feelings of positive emotion;

Table 5.14 *DSM-IV-TR criteria for the diagnosis of PTSD*

A The person has been exposed to a traumatic event in which both of the following were present:
- **(1)** The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
- **(2)** The person's response involved intense fear, helplessness, or horror.

B The traumatic event is persistently re-experienced in one (or more) of the following ways:
- **(1)** Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
- **(2)** Recurrent distressing dreams of the event
- **(3)** Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes).
- **(4)** Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
- **(5)** Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
- **(1)** efforts to avoid thoughts, feelings, or conversations associated with the trauma
- **(2)** efforts to avoid activities, places, or people that arouse recollections of the trauma
- **(3)** inability to recall an important aspect of the trauma
- **(4)** markedly diminished interest or participation in significant activities
- **(5)** feeling of detachment or estrangement from others
- **(6)** restricted range of affect (e.g. unable to have loving feelings)
- **(7)** sense of foreshortened future (e.g. does not expect to have a career)

D Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
- **(1)** difficulty falling or staying asleep
- **(2)** irritability or outbursts of anger
- **(3)** difficulty concentrating
- **(4)** hypervigilance
- **(5)** exaggerated startle response

E Duration of the disturbance is more than 1 month.

F The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

CLIENT'S PERSPECTIVE 5.3

Post-traumatic stress disorder

'It's been 8 months since my experience and I still deal with these feelings. I'm doing a lot better but throughout the week, I can feel myself getting worse and worse until I break down. The worst part is the irritability and rage I have inside of me. I don't know why I'm so mad at life but the littlest things will set me off. I don't have the "flashbacks" anymore . . . just the feelings I had when I was going through the ordeal. It's a very dark and depressing place and it's getting harder to come out of it each time it happens. I almost feel blinded and out of control when I get these attacks. It scares me to think of what I'm capable of doing. The worst part about this is that I don't know what triggers these feelings. I can be fine all day and then my mood will change for the worse. I generally feel very depressed and it's hard to deal with at times. Just when I think I don't have to worry about it anymore, it hits ten times harder. I've tried just about every remedy there is. I've seen 4 therapists and have been on 3 SSRIs . . . all of which made me worse. I feel very discouraged with life. I don't know if this even has to do with PTSD because I thought I was over it.'

Source: www.healthboards.com/

Clinical Commentary

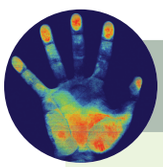
This description is typical of many PTSD sufferers and highlights feelings of depression, lack of control and anger. Some theories of PTSD (such as 'mental defeat') emphasize that those who develop PTSD after a severe trauma tend to view themselves as victims, process all information about the trauma negatively and view themselves as unable to act effectively. Such individuals believe they are unable to influence their own fate and do not have the necessary skills to protect themselves from future trauma. Ehlers and Clark (2000) suggest that such individuals only partially process their memory of the trauma because of their perceived lack of control over it, and so they do not integrate that event fully into their own autobiographical knowledge.

- *re-experiencing*: the individual regularly recalls very vivid flashbacks of events experienced during the trauma, and these images often occur in recurrent nightmares. Associated problems and symptoms include depression, guilt, shame, anger, marital problems, physical illness, sexual dysfunction, substance abuse, suicidal thoughts and stress-related violence (Jacobsen, Southwick & Kosten, 2001; Zatzick, Marmar, Weiss, Browner et al., 1997; Hobfoll, Spielberger, Breznitz, Figley et al., 1991).

The kinds of traumatic events that precipitate PTSD are often life-threatening in their severity. Studies suggest that PTSD symptoms are developed by up to 90 per cent of rape victims (Rothbaum, Foa, Riggs, Murdock et al., 1992), between 70 and 90 per cent of torture victims (Moisander & Edston, 2003), over 50 per cent of prisoners of war (POWS) (Engdahl, Dikel, Eberly & Blank, 1997), between 20 and 25 per cent of earthquake and flood survivors (Basoglu, Kilic, Salcioglu & Livanou 2004; North, Kawasaki, Spitznagel & Hong, 2004) and around 15 per cent of motor vehicle accident victims (Bryant & Harvey, 1998). More recently, *severe stress* has been included in DSM-IV-TR as a possible causal factor in PTSD, and this has led to the inclusion in this category of cases where the stressor has not been life-threatening to the sufferer (e.g. suffering PTSD after the loss of a loved one; Breslau, Davis, Andreski & Peterson, 1991) or has involved simply viewing stressful images of life-threatening traumas (e.g. watching images of the 9/11 terrorist attacks on TV; Piotrowski & Brannen, 2002). This extension of the diagnostic criteria for PTSD has generated controversy, either because it makes the symptoms of PTSD easier to fake in those who might benefit financially from a diagnosis (Rosen, 2004), or because it confuses PTSD with merely experiencing stress (McNally, 2003a). Some of the controversial issues are listed in Focus Point 5.2.

The DSM-IV-TR diagnostic criteria for PTSD are:

1 The person has been exposed to a traumatic event in which the person experienced or witnessed actual or threatening death to the self or others, or his or her response involved intense fear, helplessness or horror.



FOCUS POINT 5.2

Controversies in the study of PTSD

In many ways, post-traumatic stress disorder is a controversial topic. Controversy has arisen because of the way it is diagnosed; the potential that individuals have to fake the disorder (especially when they are involved in lawsuits to secure financial compensation for involvement in a disaster or accident); and the issue of whether repressed and then recovered memories of sexual abuse play a role in PTSD. Harvard psychologist Richard McNally (2003a) has reviewed some of these controversial issues in an article in the *Annual Review of Psychology*. They are considered below.

'Conceptual bracket creep' in the definition of trauma

Recent changes in the criteria for diagnosis of post-traumatic stress disorder mean that PTSD can be diagnosed if 'the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or *a threat to the integrity of self or others*'. McNally points out that PTSD could now be attributed to someone who merely learns about someone else being threatened with harm, or to a range of non-life-threatening stressors, such as exposure to sexual jokes in the workplace (Avina & O'Donohue, 2002).

Erroneously equating PTSD with merely experiencing stress

After the terrorist attacks on the World Trade Centre on 11 September 2001, surveys suggested that a majority of Americans were suffering substantial levels of stress as a result of the attack. Technically this would allow almost anyone to claim to have developed PTSD according to the broadened diagnostic criteria, yet this clearly fails to distinguish between normal stress and a psychological disorder.

Faking symptoms of PTSD

It is notoriously easy to fake most of the symptoms of PTSD. Because it is a diagnosis linked to explicit traumatic experiences, there are many cases of individuals faking the disorder in order to obtain financial compensation or damages against those who might have been involved in causing the trauma, or to obtain disability payments (McGrath & Frueh, 2002). Indeed, it is estimated that around 75 per cent of Vietnam war veterans who are currently claiming PTSD compensation are either faking symptoms or never actually saw combat (Burkett & Whitley, 1998).

Recovered memories of trauma

A highly controversial debate has waged for many years now about whether disorders such as PTSD may be caused by traumatic experiences (such as childhood sexual abuse) that are repressed in memory, but then subsequently recovered using contentious techniques like hypnosis (Brown, Scheflin & Hammond, 1998). The jury is still out on this issue, but as McNally points out, there is accumulating evidence that those individuals who claim to have recovered memories of previous trauma are prone to exhibit false memory effects (i.e. in laboratory tests of memory, they claim to recall and recognize items that they have not previously been shown). Clancy, McNally, Schacter, Lenzenweger et al. (2002) found that individuals who had reported being abducted by space aliens also exhibited these false memory effects in the laboratory! (See also Chapter 13, Focus Point 13.2.)

2 The traumatic event is re-experienced in intrusive thoughts and images, nightmares, the vivid reliving of events or dissociative flashbacks.

3 The persistent avoidance of stimuli associated with the trauma (e.g. avoiding thoughts and feelings associated with the trauma, or avoiding activities, places or people associated with the trauma).

4 Persistent symptoms of increased arousal, including difficulty sleeping, anger outbursts, difficulty concentrating, hypervigilance and an exaggerated startle response. For diagnosis, the duration of the symptoms should be more than 1 month and should cause significant distress or impairment in social or occupational functioning. (See Table 5.14.)

The lifetime prevalence rate for PTSD is between 1 and 3 per cent (Helzer, Robins & McEvoy, 1987). However, around 50 per cent of adults experience at least one event in their lifetime that might qualify as a PTSD-causing trauma (Ozer & Weiss, 2004). Following such events, women are significantly more likely than men to develop PTSD (by a factor of 2.4:1), and this is not explained simply by differences in the perceived threat to life from the experience (Holbrook, Hoyt, Stein & Sieber, 2002). Apart from gender differences in prevalence rates, there is also some emerging evidence on the role that cultural variables play in PTSD. Ethnic groups can differ quite significantly in the prevalence of PTSD – Caucasian disaster victims show lower prevalence rates than Latinos or African Americans – and these differences cannot be entirely explained simply by differences in the frequency of exposure to traumatic experiences (Perilla, Norris & Lavizzo, 2002; Norris, Perilla, Ibanez & Murphy, 2001).

5.6.1 The Aetiology of Post-Traumatic Stress Disorder

The diagnostic criteria for PTSD specify either a life-threatening trauma or severe stress as a causal factor in the disorder. However, not everyone who has these kinds of experiences develops PTSD. This is the main challenge for any theory of the aetiology of PTSD – why do some people develop PTSD symptoms after these experiences, but not others? The answer must lie either in psychological or biological vulnerability factors, or in the psychological strategies that individuals have developed to deal with events like trauma and stress (e.g. differences in learned coping strategies). Also, because PTSD has many different symptom features, some theories address specific features of the symptomatology (e.g. the flashbacks), and others address the time course of the disorder and how it is emotionally experienced (Brewin & Holmes, 2003). We will explore these various possibilities below when we look at the five main theories of the aetiology of PTSD:

- theory of shattered assumptions
- conditioning theory
- emotional processing theory
- 'mental defeat'
- dual representation theory

5.6.1.1 Vulnerability Factors

As not everyone who experiences a life-threatening trauma develops PTSD, there must be factors that make some people more vulnerable than others. A number of factors have been identified that characterize those individuals likely to develop PTSD after trauma. These include:

- a tendency to take personal responsibility for the traumatic event and the misfortunes of others involved in the event (Mikhliner & Solomon, 1988);
- developmental factors such as early separation from parents or an unstable family life during early childhood (King, King, Foy & Gudanowski, 1996);
- a family history of PTSD (Foy, Resnick, Sipprelle & Carroll, 1987);
- existing high levels of anxiety or a pre-existing psychological disorder (Breslau, Davis, Andreski, Peterson et al., 1997).

Interestingly, low intelligence is also a vulnerability factor (VasterlingDuke, Brailey, Constans et al., 2002), and high IQ is the best predictor of resistance to the development of PTSD (Silva, Alpert, Munoz et al., 2000). This may be because there is a link between IQ level and the development of coping strategies to deal with experienced trauma or stress. Other important predictors of PTSD development are the experiences reported by trauma victims at the time of the trauma. These include the reporting of dissociative symptoms at the time of the trauma (e.g. feelings of depersonalization, out-of-body experiences, time-slowing and amnesia) (Ehlers, Mayou & Bryant, 1998; Candel & Merckelbach, 2004) and a belief that one is about to die (McNally, 2003b). These types of experiences may be important in that they may relate to how the individual processes and stores information about the trauma at the time, and this is significant in some specific theories of PTSD symptoms.

5.6.1.2 Theory of Shattered Assumptions

Theory of shattered assumptions
A theory of PTSD that argues that a severe traumatic experience will shatter a person's belief in the world as a safe and benign place, resulting in the symptoms typical of PTSD.

Many people develop schemas of the world that portray it as a benevolent, safe place, the people who live in it as good, moral and well-meaning, and they view themselves as being worthy people (Janoff-Bulman, 1992). Therefore, when a traumatic event occurs which severely challenges these beliefs (e.g. being assaulted by a stranger or involved in a serious traffic accident while oneself obeying all the driving laws), the individual is left in a state of disbelief, shock and conflict. This challenges the individual's core beliefs, leaves him or her in a state of 'unreality', and – because the trauma survivor has had to update his or her assumptions about the world in a negative way – may adversely affect long-term adjustment to the trauma (Bolton & Hill, 1996; Janoff-Bulman, 1992). However, while this may sound like a reasonable explanation of why some individuals exhibit shock and numbing following trauma, the facts do not entirely support it. Paradoxically, it is those who have already experienced the world as an unsafe place (i.e. have experienced previous trauma) who are most likely to develop PTSD, *not* those who have a core belief that the world is safe and benevolent (Resick, 2001).

5.6.1.3 Conditioning Theory

Because there is always an identifiable traumatic experience in the history of PTSD, it is quite reasonable to suppose that many of the symptoms of PTSD may be due to classical conditioning (see Chapter 1, section 1.1.3.2). That is, trauma (the unconditioned stimulus, UCS) becomes associated at the time of the trauma with situational cues associated with the place and time of the trauma (the conditioned stimulus, CS) (Keane, Zimering & Caddell, 1985). When these cues (or similar cues) are encountered in the future, they elicit the arousal and fear that was experienced during the trauma. For example, seeing a pile of bricks on the ground may elicit strong arousal, fear and startle responses for an earthquake survivor, because such cues had become associated with the fear experienced during the traumatic earthquake experience. The conditioning model would further argue that such conditioned fear responses do not extinguish because sufferers develop both cognitive and physical avoidance responses which distract them from fully processing such cues and therefore do not allow the associations between cues and trauma to extinguish. The reduction in fear resulting from these avoidance responses reinforces those responses and maintains PTSD symptoms. There is probably an element of classical conditioning in the development of PTSD, largely because formally neutral cues do come to elicit PTSD symptoms. There is also evidence that individuals suffering PTSD will more readily develop conditioned responses in laboratory-based experiments than non-sufferers (Orr, Metzger, Lasko, Macklin et al., 2000). However, classical conditioning does not provide a full explanation of PTSD. It does not explain why some individuals who experience trauma develop PTSD and others do not, and it cannot easily explain the range of symptoms that are peculiar to PTSD and rarely found in other anxiety disorders, such as re-experiencing symptoms, dissociative experiences, and so on.

5.6.1.4 Emotional Processing Theory

Foa, Steketee and Rothbaum (1989) have suggested that the intense nature of the trauma in PTSD creates a representation of the trauma in memory that becomes strongly associated with other contextual details of the event (e.g. if a person has been badly injured in a serious traffic accident, cues to do with roads, cars, hospitals and even travelling generally will come to selectively activate the fear network in memory). The avoidance of any contexts that might activate this fear network means that there is little opportunity for the PTSD sufferer to weaken these associations between fear and the everyday cues that will activate that fear. This account has elements in common with classical conditioning models of PTSD, but it differs in some significant ways. First, ***emotional processing theory*** claims that severe traumatic experiences are of such major significance to the individual that they lead to the formation of representations and associations in memory that are quite different from those formed as a result of everyday experience. For example, if severe trauma has become associated with certain cues (e.g. after being assaulted in an alleyway), this experience will now override any other positive associations formed as a result of previous experience with that cue (the alleyway). Secondly, severe trauma not only results in cues eliciting very strong fear responses, it also changes the individual's previous assumptions about how safe the world is, so many more cues than previously will come to elicit responses related to fear, startle and hypervigilance (Foa & Rothbaum, 1998).

Emotional processing theory
Theory that claims that severe traumatic experiences are of such major significance to an individual that they lead to the formation of representations and associations in memory that are quite different to those formed as a result of everyday experience.

The emotional processing theory is an example of those theories of PTSD that have attempted to include explanations about how fear responses are learned, stored and triggered, and about how the traumatic event changes individuals' assumptions and beliefs about themselves and the world. These types of theories have come to be known as information processing models because they specify how fear memories are laid down and activated in fear networks. They have also given rise to some successful therapeutic procedures for PTSD which address how the fear network resulting from traumatic experience can be modified (Foa & Rothbaum, 1998). In addition, this account of PTSD has been elaborated more recently to take account of the fact that individuals who prior to the trauma have relatively fixed views about themselves and the world are actually more vulnerable to PTSD (Foa & Riggs, 1993).

5.6.1.5 'Mental Defeat'

Ehlers and Clark (2000) have suggested that there is a specific psychological factor that is important in making an individual vulnerable to PTSD. This is a specific frame of mind called ***'mental defeat'***, in which individuals see themselves as victims: they process all information about the trauma negatively, and view themselves as unable to act effectively. This negative approach to the traumatic event and its consequences simply adds to the distress, influences the way the individual recalls the trauma, and may give rise to maladaptive behavioural and cognitive strategies that maintain the disorder. In effect, these individuals believe they are unable to influence their own fate and do not have the necessary skills to protect themselves from future trauma. Ehlers and Clark suggest that such individuals only partially process their memory of the trauma because of their perceived lack of control over it, and so they do not integrate that event fully into their own autobiographical knowledge. This leads to symptoms such as re-experiencing the trauma in the present (outside of a temporal context), difficulty in recalling events from the trauma, and dissociation between the experience of fear responses and their meaning. The 'mental defeat' model is supported by evidence suggesting that PTSD sufferers do indeed have negative views of the self and the world, including negative interpretations of the trauma (Dunmore, Clark & Ehlers, 1999), negative interpretations of PTSD symptoms (Clohessy & Ehlers, 1999; Mayou, Bryant & Ehlers, 2001), negative interpretations of the responses of others (Dunmore, Clark & Ehlers, 1999) and a belief

'Mental defeat' A theoretical view of PTSD in which individuals see themselves as victims, process information about the trauma negatively, and view themselves as unable to act effectively.

that the trauma has permanently changed their life (Dunmore, Clark & Ehlers, 1999; Ehlers, Maercker & Boos, 2000).

5.6.1.6 Dual Representation Theory

Dual representation theory An approach to explaining post-traumatic stress disorder (PTSD) suggesting that it may be a hybrid disorder involving two separate memory systems.

A rather different approach to explaining PTSD, called ***dual representation theory***, is that it may be a hybrid disorder consisting of the involvement of two separate memory systems (Brewin, 2001; Brewin, Dalgleish & Joseph, 1996). The *verbally accessible memory* (VAM) system registers memories of the trauma that are consciously processed at the time. These memories are narrative in nature and contain information about the event, its context and personal evaluations of the experience. They are integrated with other autobiographical memories and can be readily retrieved. The *situationally accessible memory* (SAM) system, however, records information from the trauma that may have been too brief to apprehend or take in consciously, and this includes information about sights and sounds, and extreme bodily reactions to trauma. The SAM system is thus responsible for the vivid, uncontrollable flashbacks experienced by PTSD sufferers which are difficult to communicate to others (because these memories are not stored in a narrative form).

There is good neuropsychological evidence for the existence of these two separate memory systems and their links with the brain centre associated with fear (the amygdala) (Brewin, 2001). There is also evidence that is consistent with predictions from the dual representation theory. For example, Hellawell and Brewin (2004) found that, when describing their memories, PTSD sufferers characterized flashback periods with greater use of detail, particularly perceptual detail, by more mentions of death, greater use of the present tense and more mention of fear, helplessness and horror. In contrast, ordinary memories were characterized by greater mentions of secondary emotions such as guilt and anger. These findings are consistent with the view that flashbacks are the result of sensory and response information stored in the SAM system.

SUMMARY

Once again, it is clear that PTSD has a number of different features, each of which requires explanation. Some theories have tried to explain some of the specific features of PTSD (such as dual representation theory's attempt to explain specific features such as flashbacks), while others have tried to identify the dispositional features that make some individuals vulnerable to developing PTSD while others do not (e.g. theory of shattered assumptions and 'mental defeat'). Others attempt to describe why severe trauma causes the symptoms that it does, and why these anxiety-based symptoms persist for such long periods (e.g. conditioning theory and emotional processing theory).

5.6.2 The Treatment of Post-Traumatic Stress Disorder

The treatment of PTSD has two main aims. The first is to try to prevent the development of PTSD after an individual has experienced a severe trauma. The second is to treat the symptoms of PTSD once these symptoms have developed. Rapid psychological debriefing has usually been the accepted way of intervening immediately after trauma in order to try to prevent the development of PTSD, although there is now some doubt about whether this kind of rapid intervention provides any therapeutic gains. Once symptoms have developed, most psychological therapies rely on some form of exposure therapy (usually involving clients imagining events during their traumatic experience) in an attempt to extinguish fear symptoms. Therapies that possess this exposure element include imaginal flooding, eye-movement desensitization and reprocessing (EMDR) and cognitive restructuring.

5.6.2.1 Psychological Debriefing

Over the past 20 years or so there has been a growing belief amongst mental health professionals that PTSD can be prevented by immediate and rapid ***debriefing*** of trauma victims within 24–72 hours after the traumatic event (Caplan, 1964; Bisson, 2003). The exact form of the intervention can vary, with the most widely used techniques referred to as crisis intervention or critical incident stress management (CISM) (Everly, Flannery & Mitchell, 2000). The purpose of these interventions is to reassure the participants that they are normal people who have experienced an abnormal event, to encourage them to review what has happened to them, to express their feelings about the event, and to discuss and review support and coping strategies in the immediate post-trauma period. Psychological debriefing has been used with survivors, victims, relatives, emergency care workers and providers of mental health care (Bisson, 2003). The scale of this type of intervention can be gauged by reactions to the 9/11 terrorist attacks on the World Trade Centre, when more than 9,000 counsellors went to New York to offer immediate aid to victims and families of the attack (McNally, Bryant & Ehlers, 2003). Critical incident stress debriefing comprises a number of components, including:

Psychological debriefing A structured way of trying to intervene immediately after trauma in order to try to prevent the development of PTSD.

- explanation of the purpose of the intervention;
- asking participants to describe their experiences;
- discussion of the participants' feelings about the event;
- discussion of any trauma-related symptoms participants may be experiencing;
- encouraging participants to view their symptoms as normal reactions to trauma;
- discussing the participants' needs for the future (Mitchell & Everly, 1993)

As laudable as immediate professional help may seem in these circumstances, there is much criticism of psychological debriefing

Plate 5.6
The psychological impact of the devastating Asian tsunami of December 2004 is difficult to calculate. Over the past 20 years it was felt that immediate counselling of victims was the best way to prevent the development of PTSD. However, more recent research has suggested that such immediate interventions may not be helpful, and in many cases may impede natural recovery.

and its value as an intervention for PTSD. First, it is not clear whether victims will gain any benefit from being counselled by strangers and possibly 'coerced' into revealing thoughts and memories that in the immediate wake of the trauma may be difficult to express. Secondly, many of the survivors of severe trauma do not display symptoms of psychological disorders, nor will they develop PTSD. Psychological debriefing techniques make little attempt to differentiate these survivors from those who may genuinely need longer-term guidance and treatment. Thirdly, controlled comparative studies that have attempted to evaluate the effects of psychological debriefing techniques suggest there is little convincing evidence that debriefing reduces the incidence of PTSD – and indeed, in some cases it may *impede* natural recovery following trauma (Bisson, 2003; McNally, Bryant & Ehlers, 2003).

5.6.2.2 Exposure Therapies

Arguably the most effective form of treatment for PTSD is exposure therapy, in which sufferers are helped by the therapist to confront and experience events and stimuli relevant to their trauma and their symptoms. The rationale behind exposure therapy is that (1) it will help to extinguish associations between trauma cues and fear responses (Foa & Rothbaum, 1998), and (2) it will help individuals to disconfirm any symptom-maintaining dysfunctional beliefs that have developed as a result of the trauma (e.g. 'I can't handle any stress') (Foa & Rauch, 2004).

For individuals suffering PTSD, exposure to their fear triggers is often a difficult step to take, and may even make symptoms worse in the early stages of treatment (Keane, Gerardi, Quinn & Litz, 1992). This being the case, exposure can be tackled in a number of different forms – especially in various imaginal forms. This can be achieved (1) by asking clients to provide a detailed written narrative of their traumatic experiences (Resick & Schnicke, 1992); (2) with the assistance of virtual reality technology using computer-generated imagery (Rothbaum, Hodges, Ready, Graap & Alarcon, 2001); or (3) by simply asking clients to visualize feared, trauma-related scenes for extended periods of time (known as ***imaginal flooding***) (Keane, Fairbank, Caddell & Zimering, 1989). Such imaginal treatments are usually then supplemented with subsequent *in vivo* exposure that would require graded exposure to real trauma-related cues. Comparative studies generally indicate that exposure-based therapies provide therapeutic gains that are superior to medication and social support (Foa & Meadows, 1997; Marks, Lovell, Noshirvani & Livanou, 1998).

Imaginal flooding A technique whereby a client is asked to visualize feared, trauma-related scenes for extended periods of time.

A recently developed and controversial form of exposure therapy for PTSD is known as ***eye-movement desensitization and reprocessing*** (EMDR) (Shapiro, 1989, 1995). In this form of treatment, clients are required to focus their attention on a traumatic image or memory while simultaneously visually following the therapist's finger moving backwards and forwards in front of their eyes. This continues until clients report a significant decrease in anxiety to the image or memory. The therapist then encourages clients to restructure the memory positively, by thinking positive thoughts in relation to that image (e.g. 'I can deal with this'). The rationale for this procedure is that combining eye movements with attention to fearful images encourages rapid deconditioning and restructuring of the feared image (Shapiro, 1995, 1999).

Eye-movement desensitization and reprocessing A form of exposure therapy for PTSD in which clients are required to focus their attention on a traumatic image or memory while simultaneously visually following the therapist's finger moving backwards and forwards before their eyes.

There is evidence that EMDR is more effective than no treatment, supportive listening and relaxation (McNally, 1999), but some studies have shown that it has a higher relapse rate than cognitive behaviour therapy (Devilly & Spence, 1999). Critics of EMDR argue that, although it does have some success in treating the symptoms of PTSD, it is little more than just another form of exposure therapy. Indeed, there is growing evidence that the eye movement component of EMDR is not necessary for improvement (McNally, 1999; Cahill, Carrigan & Frueh, 1999), and this has led McNally (1999, p. 2) to conclude that 'what is effective in EMDR (imaginal exposure) is not new, and what is new (eye movements) is not effective'.

5.6.2.3 Cognitive Restructuring

There are various forms of cognitive restructuring therapy for PTSD, but most attempt to help clients do two things: evaluate and replace intrusive or negative automatic thoughts; and evaluate and change dysfunctional beliefs about the world, themselves and their future that have developed as a result of the trauma (Marks, Lovell, Noshirvani & Livanou, 1998; Foa & Rothbaum, 1998). For example, Foa and Rothbaum (1998) suggested that two basic dysfunctional beliefs mediate the development and maintenance of PTSD. These are (1) 'the world is a dangerous place' and (2) 'I am totally incompetent'. Foa and Cahill (2001) argued that immediately after a severe trauma, all victims develop a negative view of the world and themselves, but for most individuals these beliefs become disconfirmed through daily experience. However, those who avoid trauma-related thoughts will also

avoid disconfirming these extreme views, and this will foster the development of chronic PTSD. While exposure therapy alone may encourage experiences that disconfirm these dysfunctional beliefs, cognitive therapists have proposed that procedures that directly attempt to alter PTSD-related cognitions should also be included in the treatment (Resick & Schnicke, 1992; Steil & Ehlers, 2000). However, studies that have analysed treatments that contain both exposure and cognitive restructuring components suggest that cognitive restructuring does not significantly augment exposure therapy in producing changes in dysfunctional cognitions (Foa & Rauch, 2004).

SELF-TEST QUESTIONS

- Can you describe the main symptoms of PTSD and how they may differ from the symptoms found in other anxiety disorders?
- Can you list some of the important risk factors for PTSD, and describe how they might contribute to the development of PTSD?
- We discussed five main theories of the aetiology of PTSD (theory of shattered assumptions, conditioning theory, emotional processing theory, 'mental defeat', and dual representation theory), can you describe the main features of at least two of these and discuss their similarities and differences?
- What are the main treatments for PTSD, and how have these been derived from theories of the aetiology of PTSD?

SECTION SUMMARY

5.6 Post-Traumatic Stress Disorder (PTSD)

- The diagnosis of PTSD is based on identifying exposure to a specific fear-evoking, and usually life-threatening, event (e.g. being involved in a natural disaster or serious physical assault).
- The main symptoms of PTSD include increased arousal, numbing of emotions, flashbacks and the re-experiencing of the trauma.
- The lifetime prevalence rate for PTSD is between 1 and 3 per cent (even though around 50 per cent of adults experience at least one event in their lifetime that might qualify as a PTSD-causing event).
- **Vulnerability factors** for PTSD include a tendency to take personal responsibility for the event, developmental factors such as an unstable early family life, a family history of PTSD and existing high levels of anxiety or a pre-existing psychological disorder.
- There is no consensus on a specific psychological model of PTSD, and current explanations include: (1) the **theory of shattered assumptions**, (2) **classical conditioning**, (3) **emotional processing theory**, (4) **'mental defeat'** and (5) **dual representation theory**.
- Attempting to prevent the development of PTSD through the rapid and immediate debriefing of trauma victims (**critical incident stress debriefing**) is now generally acknowledged to be ineffective and even counterproductive.
- The most effective forms of treatment for PTSD are **exposure therapies**, where sufferers are helped by the therapist to confront and experience events and stimuli relevant to their trauma. These may include **imaginal flooding** or **eye-movement desensitization and reprocessing** (EMDR). Graduated exposure treatment can be supplemented with **cognitive restructuring** designed to evaluate and change dysfunctional beliefs about the world.

5.7 ANXIETY-BASED PROBLEMS REVIEWED

In this chapter we have reviewed six of the main anxiety-based problems – specific phobia, social phobia, panic disorder, generalized anxiety disorder (GAD), obsessive-compulsive disorder (OCD) and post-traumatic stress disorder (PTSD). Common to all of these disorders is the intense experience of anxiety that the individual finds distressing and which causes significant impairment in social, occupational or other important areas of functioning. At this point it is worth referring back to Table 5.1, where we began by summarizing some of the important features of these problems. This table shows that anxiety manifests itself in many different ways in these different disorders – as pathological worrying in GAD, as compulsive ritualized thoughts and actions in OCD, as physical panic attacks in panic disorder and as the re-experiencing of trauma in PTSD. Many of these anxiety problems are precipitated by periods of stress in a person's life (e.g. panic disorder, GAD, OCD), yet we do not yet know why an individual who has experienced a period of life stress will develop one particular disorder (e.g. OCD) rather than another (e.g. panic disorder). This will be an important issue for future clinical research.

Just as the symptoms of these anxiety-based problems are often quite different, so are the theories that try to explain them, and there is certainly no single, unified theory that can convincingly account for the development of anxiety-based problems generally. However, there are some features that are common to these different problems and this may provide some insight into how different anxiety-based problems develop. These features include the information processing and interpretational biases that accompany most anxiety disorders (see section 5.4.1.1), and also the dysfunctional beliefs that anxiety sufferers seem to form which maintain their symptoms (e.g. the spider phobic's beliefs that spiders are threatening and harmful, and the GAD sufferer's belief that worrying is an important and necessary activity to engage in). These phenomena may eventually form the basis of a unified theory of anxiety-based problems.

Finally, now that you have reached the end of this chapter, you can test out your knowledge of the symptoms of anxiety-based disorders by completing the diagnostic tree shown in Figure 5.9.

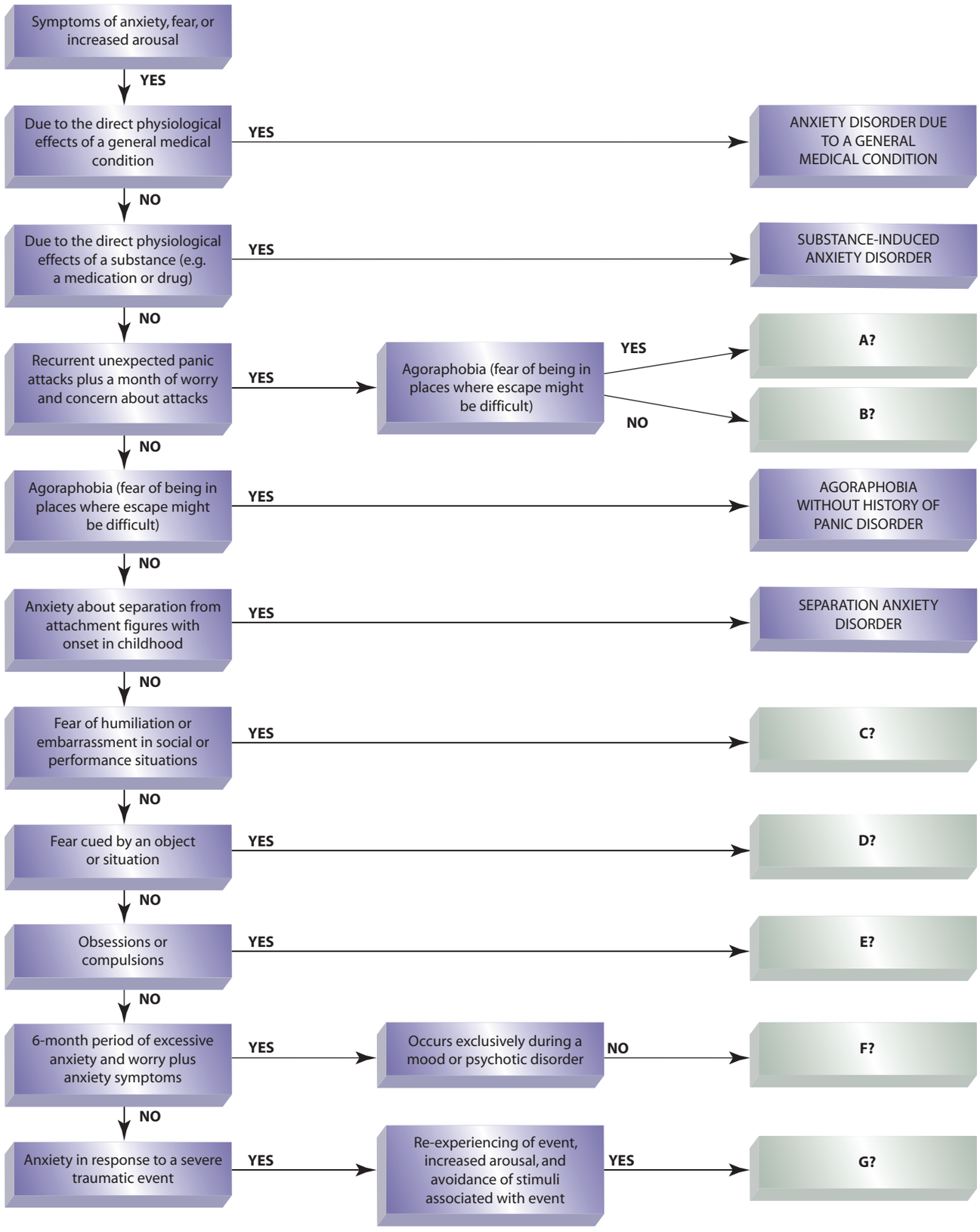


Figure 5.9
Source: adapted from DSM-IV-TR.

LEARNING OUTCOMES

When you have completed this chapter, you should be able to:

1. Describe the kinds of presenting symptoms that are associated with individual anxiety-based problems.
2. Describe the characteristics and diagnostic criteria of six of the important anxiety disorders.
3. Describe, compare and contrast at least two contemporary theories of the aetiology of each disorder.
4. Distinguish between biological and psychological explanations of anxiety-based problems.
5. Describe the relevance of research methodologies that have contributed to the understanding of the acquisition of anxiety.
6. Describe, compare and contrast at least two therapeutic procedures used for each individual anxiety disorder.

KEY TERMS

Adaptive fallacy (SP) *126*
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Anxiety Sensitivity Index (PD) *137*
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RESEARCH QUESTIONS

- Classical conditioning accounts of phobias would suggest that anyone who has a traumatic experience with a stimulus or situation should develop a phobia of it. However, this is certainly not the case. Why is it that some people acquire phobias after traumatic experiences and others do not?

- Why is it that clinical phobias usually cluster around only a small sub-set of stimuli and situations?
- Are the inherited components found in social phobia specific to this disorder, or is the genetic component a more general one that is shared with other disorders (e.g. a vulnerability to anxiety disorders generally rather than social phobia specifically)?
- Noradrenergic overactivity appears to be a characteristic of panic disorder, but is it a cause of the disorder or just a factor that mediates the symptoms of panic when an attack is triggered?
- People who suffer from panic disorder have a tendency to catastrophically misinterpret bodily sensations as threatening, but how do they acquire this interpretation bias?
- Why do individuals with GAD worry chronically and pathologically when many others – often with more stressful lifestyles – worry significantly less?
- People who are chronic worriers have a tendency to claim that they are 'born worriers' – but is there any evidence for this?
- Neuropsychological studies suggest individuals with OCD possess a number of executive functioning deficits – but do these deficits contribute to the symptoms of OCD or to the sufferer's 'doubting' that things have been done properly?
- A number of anxiety disorders are characterized by the dysfunctional perseveration of certain thoughts, behaviours or activities (e.g. pathological worrying in GAD, compulsive checking in OCD). Is pathological perseveration caused by a single process that is common to these different disorders?
- Around 50 per cent of adults will experience a severe traumatic experience during their lives, but why do only a proportion of those people develop symptoms of PTSD?
- IQ is one of the best predictors of resistance to the development of PTSD. What role does this factor play in preventing PTSD?

CLINICAL ISSUES

- Dysfunctional beliefs about the threats posed by a phobic stimulus or situation are a central feature of specific phobias. These beliefs will probably need to be challenged and changed to ensure successful treatment.
- High levels of disgust sensitivity are a feature of a number of phobias. Successful treatment may rely not only on reducing fear in these cases, but also on reducing levels of disgust sensitivity.
- Fear of social situations is so pervasive that it has been more generally labelled as 'social anxiety disorder'.
- Cognitive behaviour therapy and pharmacological treatments can often be used together with complementary benefits. In the case of social phobia, drug therapy offers an immediate benefit and CBT helps to maintain therapeutic gains over time.
- Panic disorder appears to manifest itself in different symptoms across different cultures. What implications might this have for the diagnosis and assessment of panic disorder?
- Information processing biases appear to have a causal effect on the experience of anxiety. If so, it may be possible to develop training procedures to rectify these biases and so alleviate the experience of anxiety.
- OCD sufferers often feel unable to expose themselves to their fear triggers and prevent themselves acting out their rituals. How might these problems be overcome when considering treatment for OCD?
- Neurosurgery is often a treatment of last resort for psychological disorders. Is there any justification for performing cingulatomy to treat OCD?
- 'Severe stress' has been included in DSM-IV-TR as a possible causal factor in PTSD. Does this make the symptoms of PTSD easier to fake in those who might benefit financially from a diagnosis?
- The immediate and rapid debriefing of trauma victims does not seem to reduce the subsequent incidence of PTSD. Should any form of intervention or support be offered to victims immediately following large-scale disasters?
- It is claimed that eye-movement desensitization and reprocessing (EMDR) is not a treatment for PTSD that offers anything new beyond existing therapies (even though it appears to have some therapeutic benefits). If so, should it still be used to treat PTSD sufferers?