Adult Mental Health
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,
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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:

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:

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

<table>
<thead>
<tr>
<th>DISORDER AND LIFETIME PREVALENCE RATES</th>
<th>DEFINITION</th>
<th>MAIN DSM-IV-TR DIAGNOSTIC FEATURES</th>
<th>KEY FEATURES</th>
<th>THEORIES OF AETIOLOGY</th>
<th>MAIN FORMS OF TREATMENT</th>
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<tbody>
<tr>
<td>SPECIFIC PHOBIA (7.2%–11.3%)</td>
<td>Excessive, unreasonable, persistent fear triggered by a specific object or situation</td>
<td>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</td>
<td>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</td>
<td>Psychoanalytic accounts Classical conditioning Biological preparedness Non-associative fear acquisition Disease-avoidance model</td>
<td>Exposure therapy Systematic desensitization Flooding One-session rapid treatments</td>
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Table 5.1 (Cont’d)

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<td>SOCIAL PHOBIA (7%-13%)</td>
<td>A severe and persistent fear of social or performance situations</td>
<td>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</td>
<td>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</td>
<td>Genetic factors Role of behavioural inhibition in childhood Information processing biases (e.g. negative processing of ambiguous information) Self-focused attention</td>
<td>CBT Medication (e.g. MAOIs and SSRIs)</td>
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<tr>
<td>PANIC DISORDER (1.5%-3.5%)</td>
<td>The experience of repeated and uncontrollable panic attacks</td>
<td>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</td>
<td>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’</td>
<td>Hyperventilation model Suffocation alarm theories Noradrenergic overactivity Classical conditioning Anxiety sensitivity Catastrophic misinterpretation of bodily sensations</td>
<td>Tricyclic antidepressants and benzodiazepines Exposure-based treatments CBT</td>
</tr>
<tr>
<td>GENERALIZED ANXIETY DISORDER (GAD) (5%)</td>
<td>The experience of continual apprehension and anxiety about future events, leading to chronic and pathological worry</td>
<td>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</td>
<td>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</td>
<td>Genetic factors Information processing biases Dysfunctional beliefs about worrying The role of dispositional factors</td>
<td>Anxiolytics such as benzodiazepines Stimulus control treatment CBT (including self-monitoring, relaxation training and cognitive restructuring)</td>
</tr>
</tbody>
</table>
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).

### Table 5.1 (Cont’d)

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<td><strong>OBSESSIVE-COMPULSIVE DISORDER (OCD)</strong> (2.5%)</td>
<td>Recurrent obsessions or compulsions that are severe enough to be time-consuming or cause distress</td>
<td>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</td>
<td>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</td>
<td>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</td>
<td>Exposure and ritual prevention treatments (EPR) CBT Drug treatment (SSRIs) Cingulotomy</td>
</tr>
<tr>
<td><strong>POST-TRAUMATIC STRESS DISORDER (PTSD)</strong> (3%–8%)</td>
<td>A set of persistent, anxiety-related symptoms that occur after experiencing or witnessing an extremely traumatic event</td>
<td>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</td>
<td>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, 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</td>
<td>Theory of shattered assumptions Classical conditioning Emotional processing theory Mental defeat Dual representation theory</td>
<td>Psychological debriefing Exposure therapy Eye-movement desensitization and reprocessing (EMDR) Cognitive restructuring</td>
</tr>
</tbody>
</table>
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 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.

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
CHAPTER 5 ANXIETY-BASED PROBLEMS

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...
5.1 SPECIFIC PHOBIAS

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

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.
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. 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 this predisposition is strengthened by traumatic conditioning experiences. For example, a traumatic conditioning experience of a horse falling down could lead to the development of a horse phobia. However, this traumatic conditioning experience is not the only way in which we can learn to fear horses. Seligman suggested that we are biologically prepared to fear horses because they are large and powerful animals that can cause serious injury. This predisposition is then strengthened by traumatic conditioning experiences, such as seeing a horse fall down.

Another evolutionary theory of phobias is the one proposed by 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. 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).

The book also includes a Focus Point titled “Little Hans: The psychoanalytic interpretation of a specific phobia.” This Focus Point describes the case of Little Hans, a 5-year-old boy who developed a fear of horses after 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. 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/edopsy.html
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

- 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.
- 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.

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

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, 1996).

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 & Thornik, 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).

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 Ruijer, Rijken, Garsen, van Schak & Kraaimaat, 1988; Starcevic, Uihlenhuth, 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 & Bogujevic, 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, 1993). 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.
5.1 Specific Phobias

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).

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.

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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.

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).

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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?
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
is anxiety of these socially based situations that it has been more generally labelled as ‘social anxiety disorder’ (Liebowitz, Heimberg, Fresco, & 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.

### 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, Taijinkyofu-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.

<table>
<thead>
<tr>
<th>Table 5.5 DSM-IV-TR criteria for diagnosis of social phobia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong> 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.</td>
</tr>
<tr>
<td><strong>B</strong> Exposure to the feared social situation almost invariably provokes anxiety, which may take the form of a situationally bound or situationally predisposed panic attack.</td>
</tr>
<tr>
<td><strong>C</strong> The person recognizes that the fear is excessive or unreasonable.</td>
</tr>
<tr>
<td><strong>D</strong> The feared social or performance situations are avoided or else are endured with intense anxiety or distress.</td>
</tr>
<tr>
<td><strong>E</strong> The avoidance or anxious anticipation of the feared social or performance situation(s) interfere significantly with the person’s normal routine, occupational (academic) functioning, or social activities or relationships.</td>
</tr>
<tr>
<td><strong>F</strong> In individuals under 18 years, the duration is at least 6 months.</td>
</tr>
<tr>
<td><strong>G</strong> 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.</td>
</tr>
<tr>
<td><strong>H</strong> 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.</td>
</tr>
</tbody>
</table>

### 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,
mental factors and early experiences may precipitate the disorder other anxiety disorders, it has been argued that various develop-

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

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 disoriented. 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

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).

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’).

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 (Raee & Melville, 1997; Siqueland, Kendall & Steinberg, 1996; Bruch & Heimberg, 1994). While these 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 (Strayvynski, 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 (Fox, Franklin, Perry & Herbert, 1996; Gilboa-Schechtman, Franklin & Foxa, 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 (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, Tschuk & Tee, 1992).

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.

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.
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.


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?
- 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).

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

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 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.
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).

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 (CO₂) or by encouraging hyperventilation (Ley & Walker, 1973). Similarly, sensitivity to increases in CO₂ 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 CO₂ intake may activate an over-sensitive suffocation alarm system and give rise to the intense terror and anxiety experienced during a panic attack.

Biological challenge tests

Research in which panic attacks are induced by administering carbon dioxide-enriched air (CO₂) or by encouraging hyperventilation.

Suffocation alarm theories

Models of panic disorder in which a combination of increased CO₂ intake may activate an over-sensitive suffocation alarm system and give rise to the intense terror and anxiety experienced during a panic attack.

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 CO₂ challenge.

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-aminobutyric (GABA) neurons that inhibit
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 the nature of these anxiety-producing cues. 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.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Biological mechanism for causing panic</th>
<th>Effect on panic disorder sufferers and non-clinical controls</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium lactate</td>
<td>Possibly through alkalinization of the blood</td>
<td>Tends to induce panic attacks in panic disorder sufferers but not in normal controls</td>
<td>Bourin et al. (1998)</td>
</tr>
<tr>
<td>Carbon dioxide (CO₂)</td>
<td>Increased breathing and hyperventilation in order to remove CO₂ causes respiratory alkalosis</td>
<td>Produces an increase in subjective anxiety in panic disorder sufferers including symptoms of panic</td>
<td>Gorman et al. (1984)</td>
</tr>
<tr>
<td>Yohimbine</td>
<td>Increases central noradrenergic activity in the locus ceruleus (an area of the brain implicated in panic)</td>
<td>Tends to induce panic attacks in a majority of panic disorder sufferers, and greater levels of self-reported anxiety than controls</td>
<td>Charney et al. (1984)</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Indirectly increases norepinephrine and increases arousal</td>
<td>Tends to cause panic symptoms in panic disorder sufferers but not in normal controls</td>
<td>Charney et al. (1985)</td>
</tr>
</tbody>
</table>


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,
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).

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

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

**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 hypothesized 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 CO2 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.

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...
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

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**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.

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

---

associated with panic, the client is then asked to apply cognitive and physical techniques 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 catastrophic 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).

- 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 CO₂ 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)

**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

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

NON-WORRIER

<table>
<thead>
<tr>
<th>Topic: Getting good grades in school</th>
<th>Discomfort likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catastrophizing step</td>
<td></td>
</tr>
<tr>
<td>I might do poorly on a test.</td>
<td>3 20</td>
</tr>
<tr>
<td>I’d get a bad grade in the class.</td>
<td>3 100</td>
</tr>
<tr>
<td>That would lower my grade-point average.</td>
<td>2 100</td>
</tr>
<tr>
<td>I’d have less of a chance of getting a good job.</td>
<td>2 60</td>
</tr>
<tr>
<td>I’d end up in a bad job.</td>
<td>2 80</td>
</tr>
<tr>
<td>I’d get a low salary.</td>
<td></td>
</tr>
<tr>
<td>I’d have less money to spend on what I want.</td>
<td>2 100</td>
</tr>
<tr>
<td>I’d be unhappy.</td>
<td>2 35</td>
</tr>
<tr>
<td>It would be a strain on me.</td>
<td>2 10</td>
</tr>
<tr>
<td>I’d worry more.</td>
<td>2 5</td>
</tr>
</tbody>
</table>

5.4 GENERALIZED ANXIETY DISORDER (GAD)

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...
interpretive 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 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**

**Information processing biases**

Biases in interpreting, attending to, storing or recalling information which may give rise to dysfunctional thinking and behaving.
5.4 GENERALIZED ANXIETY DISORDER (GAD)

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
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
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).

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 (Armstrong, 2003).

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).

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 techniques, including relaxation training, cognitive restructuring, and other methods such as mindfulness and positive psychology.
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).

**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 psychological treatments of GAD attempt to bring the activity of worrying under control?
- What are the features of worry in GAD?
- What is the cardinal diagnostic feature of GAD?

**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. 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.

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 (Stekete, Frost & Kyrios, 2003), superstition 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.

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).
5.5 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 consider-able 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

<table>
<thead>
<tr>
<th>Obsessions are defined as:</th>
<th>Compulsions are defined as:</th>
</tr>
</thead>
<tbody>
<tr>
<td>(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.</td>
<td>(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.</td>
</tr>
<tr>
<td>(2) The thoughts, impulses, or images are not simply excessive worries about real-life problems.</td>
<td>(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.</td>
</tr>
<tr>
<td>(3) The person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action.</td>
<td></td>
</tr>
<tr>
<td>(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).</td>
<td></td>
</tr>
</tbody>
</table>

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.
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 (Sheffer-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 (Salkovskis, 1985; Rachman, 1998).

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.

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.

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

clients from engaging in their rituals: compulsive rituals (see Treatment in Practice Box 5.5). Preventing
practising competing behaviours, habit reversal or modification of
is ritual or response prevention, which involves strategies such as
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 dis-
tress 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:

1 allows anxiety to extinguish by habituating the links
between obsessions and their associated distress;
2 eliminates ritualistic behaviours that may negatively
reinforce anxiety (Stekete, 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 alter-
native 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
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

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

Table 2

<table>
<thead>
<tr>
<th>OCD Symptom</th>
<th>Response Prevention Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand washing or cleaning rituals</td>
<td>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)</td>
</tr>
<tr>
<td>Checking lights, switches, oven, appliances, etc.</td>
<td>Response delay; use of ritual restrictions (e.g. restrict number of checks); turning and walking away; extension strategies (whistle a happy tune)</td>
</tr>
<tr>
<td>Counting (e.g. bricks, words)</td>
<td>Refocusing techniques; singing a song; going ‘blank’; meditation</td>
</tr>
</tbody>
</table>

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 cingulotomy, 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).

**Cingulotomy** 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. cingulotomy).

**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. Such events include combat during a war, rape or other types of physical assault, child abuse, car or aeroplane crashes, or natural or humanity-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:

- 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;
**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.’

**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.

Source: www.healthboards.com/

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

<table>
<thead>
<tr>
<th>A</th>
<th>The person has been exposed to a traumatic event in which both of the following were present:</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>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.</td>
</tr>
<tr>
<td>(2)</td>
<td>The person’s response involved intense fear, helplessness, or horror.</td>
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<table>
<thead>
<tr>
<th>B</th>
<th>The traumatic event is persistently re-experienced in one (or more) of the following ways:</th>
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</thead>
<tbody>
<tr>
<td>(1)</td>
<td>Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.</td>
</tr>
<tr>
<td>(2)</td>
<td>Recurrent distressing dreams of the event</td>
</tr>
<tr>
<td>(3)</td>
<td>Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes).</td>
</tr>
<tr>
<td>(4)</td>
<td>Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.</td>
</tr>
<tr>
<td>(5)</td>
<td>Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C</th>
<th>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:</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>efforts to avoid thoughts, feelings, or conversations associated with the trauma</td>
</tr>
<tr>
<td>(2)</td>
<td>efforts to avoid activities, places, or people that arouse recollections of the trauma</td>
</tr>
<tr>
<td>(3)</td>
<td>inability to recall an important aspect of the trauma</td>
</tr>
<tr>
<td>(4)</td>
<td>markedly diminished interest or participation in significant activities</td>
</tr>
<tr>
<td>(5)</td>
<td>feeling of detachment or estrangement from others</td>
</tr>
<tr>
<td>(6)</td>
<td>restricted range of affect (e.g. unable to have loving feelings)</td>
</tr>
<tr>
<td>(7)</td>
<td>sense of foreshortened future (e.g. does not expect to have a career)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D</th>
<th>Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>difficulty falling or staying asleep</td>
</tr>
<tr>
<td>(2)</td>
<td>irritability or outbursts of anger</td>
</tr>
<tr>
<td>(3)</td>
<td>difficulty concentrating</td>
</tr>
<tr>
<td>(4)</td>
<td>hypervigilance</td>
</tr>
<tr>
<td>(5)</td>
<td>exaggerated startle response</td>
</tr>
</tbody>
</table>

| 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. |
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, Salioglu & 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 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, Schefflin & 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.)
5.6 POST-TRAUMATIC STRESS DISORDER (PTSD)

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 psycho- logical 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 (Mikhlin & 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

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 numbness 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 activate the fear network in memory). The avoidance of any contexts that might activate this fear network means that there is an inability to fully process 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.

5.6.1.5 ‘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.
that the trauma has permanently changed their life (Dunmore, Clark & Ehlers, 1999; Ehlers, Maercker & Boos, 2000).

5.6.1.6 Dual Representation Theory

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, Hellowell 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 perceptual detail, by more mentions of death, greater use of the present tense and more mention of fear, helplessness and horror. 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:

- 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, 1995)

As laudable as immediate professional help may seem in these circumstances, there is much criticism of psychological debriefing
indeed, in some cases it may evidence that debriefing reduces the incidence of PTSD – and psychological debriefing techniques suggest there is little convincing comparative studies that have attempted to evaluate the effects of psychological debriefing. 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 & Livancou, 1998).

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).

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 & Livancou, 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?

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.
Symptoms of anxiety, fear, or increased arousal

Due to the direct physiological effects of a general medical condition

NO

Due to the direct physiological effects of a substance (e.g., a medication or drug)

NO

Recurrent unexpected panic attacks plus a month of worry and concern about attacks

YES

Agoraphobia (fear of being in places where escape might be difficult)

NO

Agoraphobia (fear of being in places where escape might be difficult)

NO

Anxiety about separation from attachment figures with onset in childhood

NO

Fear of humiliation or embarrassment in social or performance situations

YES

C?

Fear cued by an object or situation

NO

Obsessions or compulsions

NO

6-month period of excessive anxiety and worry plus anxiety symptoms

NO

Anxiety in response to a severe traumatic event

NO

Re-experiencing of event, increased arousal, and avoidance of stimuli associated with event

YES

G?

ANXIETY DISORDER DUE TO A GENERAL MEDICAL CONDITION

SUBSTANCE-INDUCED ANXIETY DISORDER

AGORAPHOBIA WITHOUT HISTORY OF PANIC DISORDER

SEPARATION ANXIETY DISORDER

ANXIETY DISORDER DUE TO A GENERAL MEDICAL CONDITION

AGORAPHOBIA

ANXIETY DISORDER

SEPARATION ANXIETY DISORDER

ANXIETY DISORDER DUE TO A GENERAL MEDICAL CONDITION

AGORAPHOBIA WITHOUT HISTORY OF PANIC DISORDER

A?

B?

C?

D?

E?

F?

G?

Figure 5.9
Source: adapted from DSM-IV-TR.
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
- Anxiety disorder 118
- Anxiety sensitivity (PD) 137
- Anxiety Sensitivity Index (PD) 137
- Behavioural rehearsal (SOC, PD, OCD) 145
- Biological challenge tests (PD) 135
- Biological preparedness (SP) 125
- Catastrophic misinterpretation of bodily sensations (PD) 137
- Catastrophizing (GAD) 141
- Cingulotomy (OCD) 154
- Cognitive restructuring (SP, SOC, PD, GAD, OCD, PTSD) 145
- Comorbidity 118
- Compulsions (OCD) 146
- Disease-avoidance model (SP) 126
- Disgust (SP) 128
- Dual representation theory (PTSD) 159
- Emotional processing theory (PTSD) 158
- Exposure and ritual prevention treatments (OCD) 152
- Eye-movement desensitization and reprocessing (EMDR) (PTSD) 160
- Generalized anxiety disorder 139
- Hyperventilation (PD) 135
- Imaginal flooding (SP, PTSD) 160
- Incubation (SP) 124
- Inflated responsibility (OCD) 149
- Information processing biases in anxiety (GAD) 142
- ‘Mental defeat’ (PTSD) 158
- Mood-as-input hypothesis (OCD) 151
- Non-associative fear acquisition (SP) 125
- Obsessions (OCD) 146
- Obsessive-compulsive disorder (OCD) 146
- Panic (PD) 126
- Panic disorder 133
- Phobic beliefs (SP) 121
- Post-traumatic stress disorder 154
- Psychological debriefing (PTSD) 159
- Relaxation training (SP, GAD) 145
- Self-focused attention (SOC) 131
- Self-monitoring (GAD) 145
- Social phobia 128
- Specific phobia 121
- Stimulus control treatment (GAD) 145
- Suffocation alarm theories (PD) 135
- Theory of shattered assumptions (PTSD) 157
- Thought suppression (OCD) 151
- Thought-action fusion (OCD) 151
5.2 Social Phobia


5.3 Panic Disorder


5.4 Generalized Anxiety Disorder (GAD)


5.5 Obsessive-Compulsive Disorder (OCD)


5.6 Post-Traumatic Stress Disorder (PTSD)


**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 cingulotomy 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?